

ARTICLE

Evolution Mechanisms of Thoracic Blunt Load Transfer and Cardiopulmonary Responses in the Protected Thorax under Rifle-Bullet Impact

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ABSTRACT: Body armor prevents projectile penetration, but thoracic visceral injury may still result from pressure-wave transmission and back-face deformation. However, thoracic responses to single and multiple impacts remain insufficiently understood, numerical human thoracic models need further validation, and load transfer between the rib cage and cardiopulmonary system remains unclear. Therefore, this study combined live-fire blunt-impact tests on a biomimetic thoracic target with a human thoracic finite element model with filled thoracic cavity gaps. Load transfer and cardiopulmonary responses under non-penetrating rifle-bullet impact were investigated using a SiC/UHMWPE composite ballistic insert. The results showed that, first, under a single impact, the right-lung pressure fluctuated rapidly in the initial stage, then entered an oscillatory process and gradually tended to stabilize; under multiple impacts, the peak right-lung pressure exhibited nonlinear fluctuation characteristics. Second, after the thoracic visceral organs were encapsulated with soft-tissue materials, the model was validated, and the continuity of internal load transfer was significantly enhanced. Third, during the initial establishment stage of back-plate deformation, the displacement responses of the sternum, costal cartilage, and cardiopulmonary tissues could be described by a linear-exponential function; after significant compression of the back plate occurred, the stress responses of the rib cage, right lung, and heart conformed to the characteristics of a power-exponential composite function. Finally, the pressure peak on the posterior side of the sternum was only 39.3% of that on the impact-facing surface; relative to the rib cage, the stress peaks in the right lung and heart were attenuated by 65.3% and 82.2%, respectively, indicating that the protection of cardiopulmonary visceral organs by the rib cage was mainly achieved through the synergistic effect of rigid shielding by the sternum and flexible energy dissipation by the costal cartilage. The research results can provide references for the back-plate stiffness design of ballistic inserts, optimization of buffer-layer structures, and assessment of thoracic blunt injury risk.

KEYWORDS: Multiple blunt impact injury; pulmonary band-like high-response pattern; thoracic finite element model; power-exponential right-lung pressure response; four-stage impact response

1 Introduction

With the advancement of individual protective technologies, composite body armor can effectively prevent projectile penetration. However, transient backface deformation and impact loading may still transfer substantial impact energy to the human thorax [1], thereby causing behind-armor blunt trauma (BABT) [2,3]. In this context, as the primary load-bearing structure of the thorax, the rib cage directly influences the transmission of impact loads to heart and lungs through its injury patterns and impact response characteristics [4]. Accordingly, previous studies have shown that rib fractures [5,6], costal cartilage injuries [7], and stress concentration in the sternum [8] are among the most common skeletal injury types in BABT. Such injuries can alter the overall stiffness distribution of the rib cage and modify load transmission pathways. Moreover, Yoganandan et al. [9] pointed out that the development of BABT involves protective-structure deformation, pressure-wave transmission, and the coupled response between rib cage and internal organs, whereas no stable correlation has been established between conventional backface deformation metrics and the actual severity of visceral injury. Therefore, it is important to clarify the transmission characteristics of impact loads under BABT. It is also necessary to understand the impact response of the rib cage and cardiopulmonary tissues from the perspective of human thoracic response. These findings can provide a basis for optimizing the structure of composite body armor and assessing the risk of thoracic injury.

In real combat environments, the human thorax is often exposed to complex threats arising from repeated gunfire and different projectile types. Zhang and Yuan [10] reported that projectile type significantly affects the mode of impact energy dissipation and the response pattern of protective structures. Jull et al. [11] proposed multi-hit ballistic resistance requirements. In recent years, some studies have begun to focus on the issue of multiple impacts. However, related work has mainly concentrated on the multi-hit ballistic resistance and repeated-impact performance of ceramic/metal composite armor, ceramic/UHMWPE composite armor, or cushioning materials. Shen et al. [12] investigated the effects of adhesive-layer type, thickness, and interfacial properties on the multi-hit ballistic performance of ceramic/metal composite armor. Fejdyš et al. [13] analyzed the ballistic performance of SiC, Al₂O₃ ceramics, and multilayer UHMWPE composite armor under multi-hit projectile impacts. Koohbor et al. [14] conducted a study on the dynamic response and energy-buffering capacity of elastomeric foam materials under multiple-impact conditions. However, the above studies mainly focused on the penetration resistance of protective materials [15] themselves or the repeated-impact performance of cushioning materials, rather than the internal responses of thoracic organs under BABT conditions. Research on differences in intrathoracic pressure responses under multiple impacts remains limited, with a particular lack of systematic understanding of the evolution patterns of thoracic organ responses.

Current research on BABT mainly relies on three approaches: ballistic gelatin models, physical rib-cage surrogate models, and finite element human models. Among these, ballistic gelatin models can be used to measure backface deformation and pressure responses [16]. However, because they lack skeletal structures and organ constraints, they are unable to capture load distribution characteristics at the bone-soft tissue interface. To address this limitation, Chaufer et al. [17] developed a physical rib-cage model incorporating surrogate materials for both the ribs and internal organs to obtain visceral response data. Nevertheless, differences still remain between such models and actual human tissues in terms of strain-rate effects and interfacial mechanical properties. Compared with these experimental surrogates, finite element human models provide a more powerful tool for investigating BABT because they can simulate protective-structure failure [18], rib-cage impact responses [19], nonlinear soft-tissue deformation [20], and the temporal transfer of impact energy [21], thereby enabling quantitative prediction of rib stress and organ pressure. Even so, such models are typically established based on idealized assumptions, and their physical validity and predictive reliability still need to be verified through live-fire experiments. For instance,

Wang et al. [22] developed a finite element model of the human torso based on the anthropomorphic structure of the Total Human Model for Safety (THUMS) and analyzed the transmission of pressure waves along the muscle-spine-visceral organs pathway after a rifle projectile penetrated a ballistic insert. However, because the thoracic cavity was not filled with continuous soft-tissue materials, the model was unable to adequately capture the reflection and attenuation of high-frequency pressure waves within the cavity, as well as their medium-coupled transmission with cardiac and pulmonary tissues. Fan et al. [23] further improved this modeling strategy by incorporating thoracic muscle soft tissues and internal organ filling, and investigated the dynamic response characteristics of the rib cage-organ system under the combined effects of shock waves and fragments. However, that study mainly focused on blast scenarios rather than BABT, and therefore did not address the internal transmission of pressure waves under multiple BABT-related loading conditions.

Existing studies have shown that lung injuries under thoracic impact loading often exhibit a non-uniform spatial distribution pattern. Jaffin et al. [24] further observed, both macroscopically and microscopically, hemorrhagic lesions associated with rib imprints in the right lung of rats subjected to blast injury. Marro et al. [25] also reported that lung injuries in automobile collisions commonly manifested as patchy hemorrhage. Yoganandan et al. [26] reported pulmonary contusion, hemorrhage, and laceration caused by rib fractures in an *in vivo* porcine BABT experiment. According to the *in vivo* animal experimental image of left-lung injury shown in their study, in addition to obvious hemorrhage and laceration on the lung tissue surface, a relatively indistinct linear indentation can also be observed in the upper region near the pleura. Its morphology shows a locally continuous distribution and exhibits certain band-like characteristics. Since the original study did not specifically discuss the formation mechanism of this phenomenon, the present study, based on this observation, further used a numerical human thoracic model to analyze the possible causes of localized band-like high-response characteristics in the lung, as well as their potential relationship with rib load transfer and local compression of lung tissue.

In summary, existing studies remain limited in several aspects, including the mechanisms of pulmonary responses under single and multiple impacts, the validation of the physical rationality of numerical human thoracic models, the formation and evolution mechanisms of localized band-like high-response distributions in the lung under blunt impact, and the protective role of the rib cage in cardiopulmonary organs and the associated potential injury risks. This study therefore combines live-fire blunt-impact experiments using a biofidelic thoracic surrogate with a protective-structure-human thorax finite element model. The Type 53 7.62 × 54 mmR armor-piercing incendiary projectile, hereafter referred to as the Type 53 Armor Piercing Incendiary (API) projectile, was selected as the test ammunition to investigate thoracic blunt-impact responses under single-hit and repeated-hit conditions. The human thoracic numerical model with soft tissue encapsulation around the internal organs was validated for physical plausibility. Subsequently, the impact response characteristics of the rib cage-cardiopulmonary system under backplate compressive deformation were investigated, together with the formation mechanisms and evolution patterns of the strip-like response distribution in the lungs, as well as the protection-injury mechanisms of the sternum and costal cartilage. The findings provide a reference for the stiffness design of body armor back plates, the structural optimization of buffer layers, and the development of risk assessment models for blunt thoracic injury.

2 Methods

2.1 Fabrication of the Biofidelic Dummy Model

A biofidelic thoracic dummy model incorporating the heart, lungs, rib cage, and spine was developed in this study to simulate the impact response of the human thorax under impact loading, as shown in Fig. 1. The maximum length, width, and depth of the thoracic model were 54.50, 30.00, and 35.00 cm, respectively.

During fabrication, a commercially purchased medical biomimetic human skeleton made of polyvinyl chloride (PVC) resin was used as the structural framework of the skeletal system. Manickam et al. [27] used a PVC-based C4-C5 segment prototype model for experimental testing and cross-validated the experimental results using finite element analysis, indicating that PVC can be used as a skeletal surrogate material in biomechanical model experiments. As shown in Fig. 2, mineral oil and styrene-ethylene-butylene-styrene (SEBS) polymer powder were weighed according to the designed mass fractions, heated, mixed, and then cast to fabricate soft-tissue structures, including the heart, lungs, and muscles. Xu et al. [28] reported that the stress-strain response of SEBS gel was approximately linear at strains below 0.4, and that its elastic modulus could be adjusted by changing the gel mass fraction to approximate that of different visceral organs, indicating that SEBS gel can be used as a surrogate material for internal organs. In addition, Shen et al. [29] demonstrated that by varying the weight ratio of SEBS powder to mineral oil, SEBS gel can be matched to various human soft tissues, ranging from adipose-like tissues to dense muscle tissues. Therefore, SEBS gel was selected in this study as a tunable soft-tissue surrogate material to simulate the mechanical properties of cardiopulmonary and muscle tissues. In repeated-impact experiments, SEBS gel exhibited higher structural stability and mechanical durability than commonly used soft-tissue surrogate materials, including ballistic gelatin [30–32], polydimethylsiloxane (PDMS) [33,34], and silicone [35].



Figure 1: Biofidelic human thoracic model.

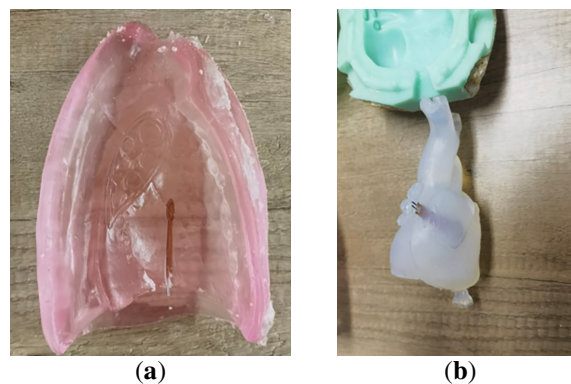


Figure 2: Fabrication of the biofidelic heart and lung: (a) Lung; (b) Heart.

2.2 Sensor Selection and Arrangement

A KD1000HA accelerometer (Yangzhou Kedong, China) and an MVS1010-100-28 Polyvinylidene fluor (PVDF) piezoelectric film sensor (Zhimeikang Technology, China) were employed in this study to satisfy the high temporal-resolution requirements for measuring acceleration and pressure-wave signals in blunt impact tests. Both sensors weighed less than 2 g, which effectively minimized the influence of sensor implantation on the structural integrity of the target and on the test results. The KD1000HA sensor employs a PZT-5 shear-mode piezoelectric structure and has a frequency response range of 1–15 kHz, a resonant frequency of approximately 50 kHz, a sensitivity of 10 mV/g, and an operating temperature range of -55°C to 125°C , which allows stable operation under the elevated-temperature conditions generated by blunt bullet impact. The PVDF film sensor has a thickness of 28 μm , an active area of 10 mm $\times$ 10 mm, a g33 constant of approximately $-330 \times 10^{-3} \text{ V}\cdot\text{m/N}$, and an operating temperature range of -40°C to 80°C . These characteristics make it well suited for accurately capturing and analyzing pressure-wave signals associated with the thoracic response under blunt impact loading.

During the experiment, because the impact signal was sensitive to sensor placement, all sensors were wrapped with adhesive tape and secured at the corresponding locations inside the dummy model. Specifically, the accelerometer was positioned in the T6 segment region of the sternum, as shown in Fig. 3a, while the PVDF film sensors were placed in the corresponding regions of the heart and right lung, as shown in Fig. 4a,b. Correspondingly, the typical structural elements used to extract dynamic responses in the numerical model were kept consistent with the sensor locations, namely point a in Fig. 3b and points b and c in Fig. 4c, to ensure that the measured signals could accurately reflect the response characteristics of key thoracic organs under impact loading.

2.3 Experimental Setup

The typical impact test of a composite ballistic insert followed the military body armor impact standard, as shown in Fig. 5. This study carried out blunt-impact tests on an anthropomorphic surrogate fitted with an SiC/UHMWPE composite ballistic insert by using the Type 53 API projectile [36]. Each test group used the same ballistic insert and the same anthropomorphic surrogate during impact tests at the specified locations. The ballistic insert remained in front of the anthropomorphic surrogate throughout the test. Straps tightly secured the ballistic insert to the anthropomorphic surrogate in order to reduce the gap effect, and no obvious visible gap remained between them.

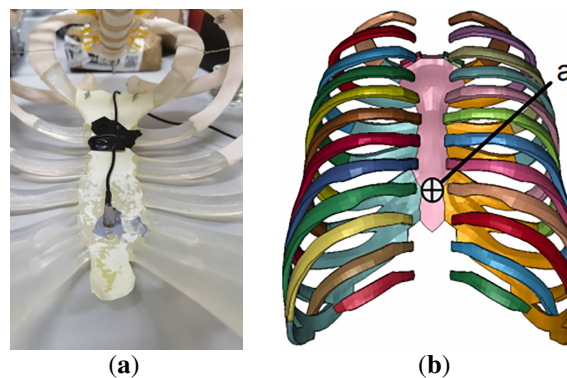


Figure 3: Location of the sternal accelerometer in the experiment (a) and the representative element used for acceleration extraction in the simulation (b).

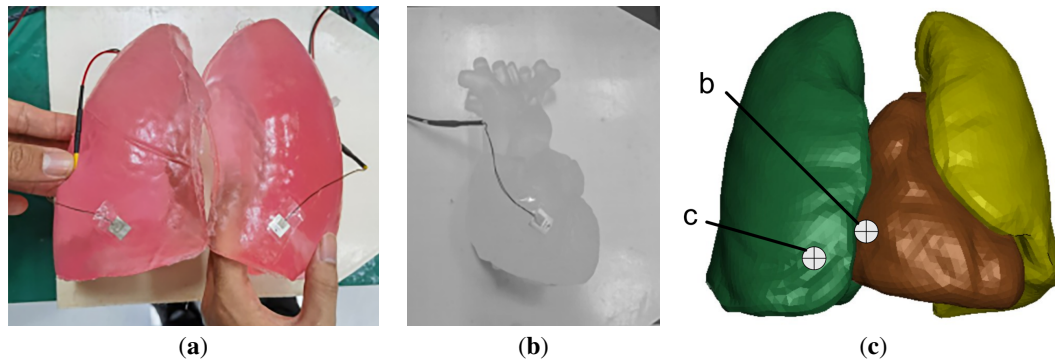


Figure 4: Layout of PVDF piezoelectric film sensors for the lung (a) and heart (b) and locations of representative elements for pressure extraction in the simulation (c).

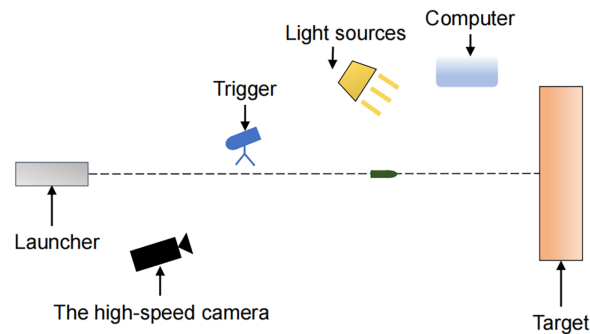


Figure 5: Schematic of the test system.

Fig. 6 shows the overall experimental layout. The assembled surrogate target was subsequently placed on the test bench at a distance of 5 m from the muzzle. The monochrome high-speed camera and the velocity sensor were positioned 15 cm from the muzzle to capture the impact event, while the trigger was placed 50 cm from the muzzle. The shooting distance from the muzzle to the target was 5 m. The charge amplifier used for signal conditioning was located approximately 2 m to the side of the dummy, and the data acquisition system was positioned adjacent to it to minimize interference and attenuation during signal transmission. The bullet impact point was also determined using a laser gun sight.

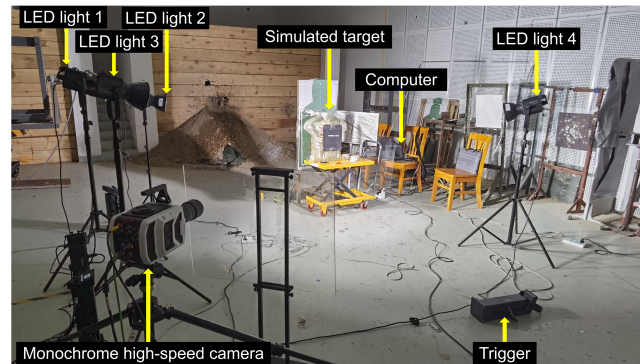


Figure 6: Schematic of sensor arrangement and blunt impact testing on ballistic insert and a biofidelic human thorax surrogate target.

3 Results

3.1 Analysis of Single Impact and Multiple Impact Test Results

This study designed two test groups, denoted as group A and group B, in order to reduce influence of target variability on the experimental results. The impact velocity of each projectile is listed in [Table 1](#).

Table 1: Projectile trigger velocity for each test group.

Group	Ammunition Type	Shot No.	Trigger Velocity (m/s)
A1	53API	1	853.9
A2	53API	2	878.2
A3	53API	3	877.0
A4	53API	4	881.2
B1	53API	1	872.1
B2	53API	2	880.4
B3	53API	3	874.8
B4	53API	4	871.1

3.1.1 Analysis of the Right Lung Pressure Time-History Response Characteristics under Single Impact

Penetration Process and Dynamic Deformation of the Biofidelic Thoracic Surrogate

The validity of the subsequent numerical model results was verified by analyzing the B1 test group corresponding to the numerical simulation condition. The high-speed photographic dynamic deformation process of the body armor and biofidelic thoracic surrogate is shown in [Fig. 7](#). A certain stand-off distance existed between the projectile and the upper-torso surrogate, and the projectile first contacted the ballistic insert at 50 μ s. The armor-piercing incendiary projectile produced an obvious flame jet, while debris generated by ceramic-layer fracture was also observed to scatter outward. Penetration caused the depressed region to expand gradually and led to pronounced deformation of the entire biofidelic thoracic model. The deformation of the biofidelic thoracic model reached its maximum at 400 μ s and then gradually rebounded under the release of its own elastic strain energy.

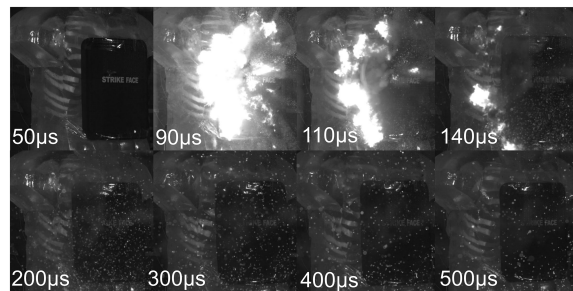


Figure 7: Dynamic penetration process of the Type 53 API projectile impacting the biofidelic human thoracic target.

Analysis of the Right Lung Pressure Time-History Response under Single Blunt Impact

The first shot in each test group was treated as a single-hit blunt-impact case for comparative analysis of the right lung pressure-time response, as shown in [Fig. 8](#). First, both groups A1 and B1 exhibited a rapid initial increase and reached the peak at an early stage of impact, followed by a pronounced decline and oscillatory attenuation process, which is a typical transient dynamic response characteristic. This also indicates that the

protective structure generated strong rebound vibration after impact, which caused significant reflection of the intrathoracic pressure wave. Moreover, each test group exhibited a distinct multi-peak pressure response characteristic. Specifically, within the 70–100 μs interval, the right lung pressure curves under all conditions exhibited the first minor peak, followed by pronounced fluctuations. This may be because the first pressure peak was mainly caused by transmission of the initial pressure wave generated by projectile impact on the protective structure, whereas the subsequent fluctuations were primarily induced by pressure responses associated with rear-face deformation of the protective structure.

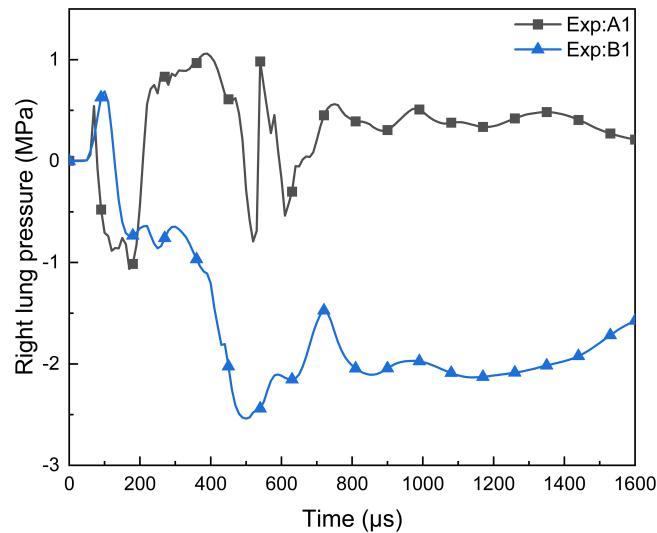


Figure 8: Comparison of right lung pressure time-history response curves under single blunt-impact tests.

3.1.2 Analysis of the Right Lung Impact Response Characteristics under Repeated Impacts

Fig. 9 shows the right lung impact response characteristics of the protected thorax under repeated rifle-bullet blunt impacts. The data from each test group were averaged to obtain the mean experimental pressure curves, and the corresponding right lung pressure-time response results are presented in Fig. 9a,c. The overall fluctuations of the right lung pressure-time curves were relatively large, and the first pressure peak had not yet reached a stable state. The second pressure peak in each test group was therefore selected for analysis, and the corresponding peak-variation curves are shown in Fig. 9b,d.

The comparison between Fig. 9a,c shows that the mean curve suppresses the extreme pressure fluctuations observed in individual tests, resulting in a more stable overall trend. The results in Fig. 9b,d further indicate that, under the protection of the SiC/UHMWPE composite ballistic insert, the amplitude of lung pressure oscillation gradually decreases as the number of impacts from the Type 53 API projectile increases, while the peak values exhibit an overall downward trend. The pressure response therefore evolves from severe fluctuations in the initial stage to a more gradual and stable pattern. This trend may be attributed to the progressive compaction of local structures in the biofidelic dummy under repeated impacts, which gradually stabilizes internal contact state and thus leads to a more stable impact response.

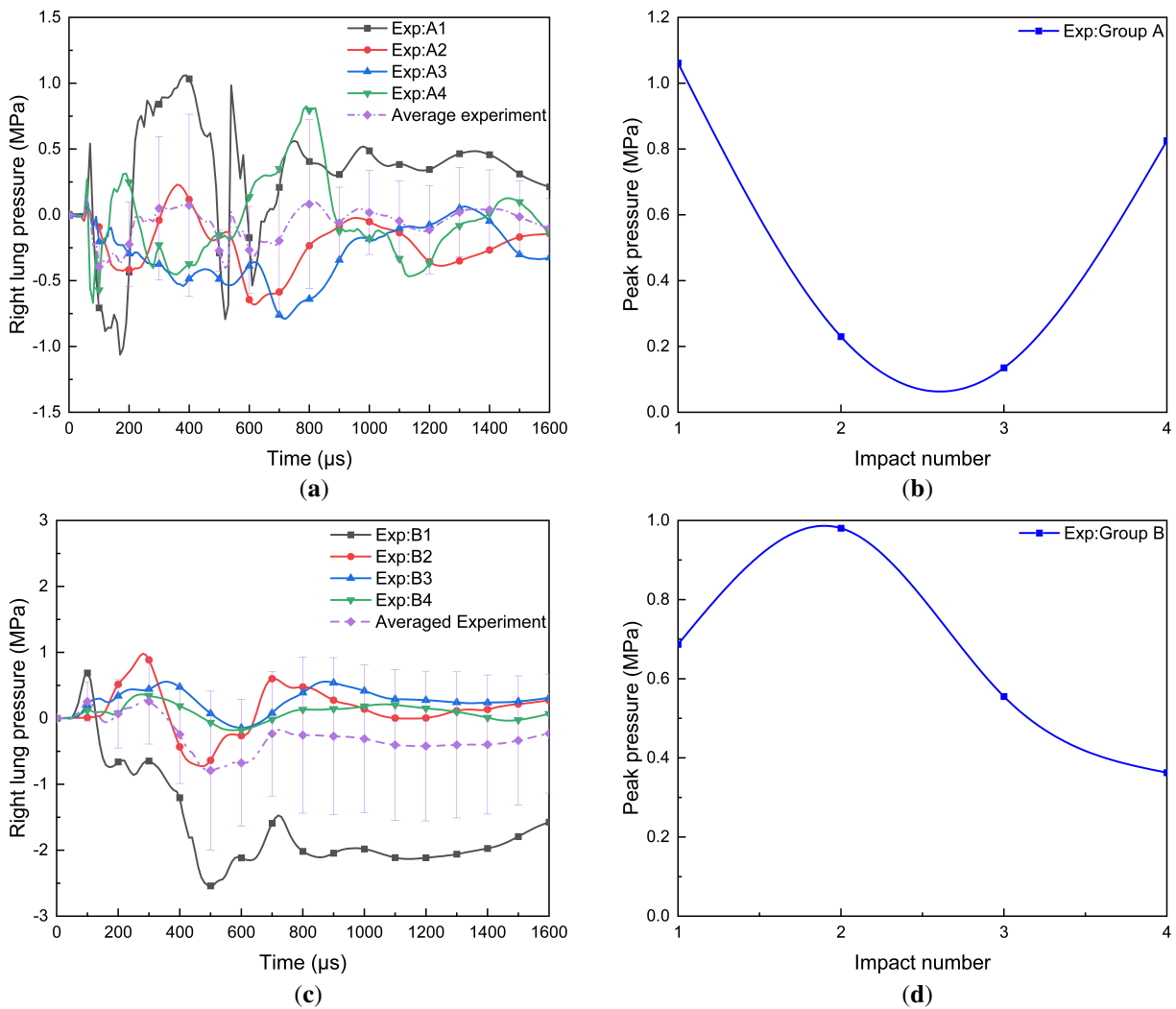


Figure 9: Comparison of right lung pressure responses under repeated blunt-impact tests: (a) pressure time-history response curves of group A; (b) pressure peak curves of group A; (c) pressure time-history response curves of group B; (d) pressure peak curves of group B.

Table 2 presents the statistical results of the right lung pressure peak values in the two groups of repeated blunt impact tests, including the mean and variance. The data in the table shows the right lung pressure peak values in both groups A and B exhibited certain fluctuations under repeated impacts, indicating that the dynamic response of the right lung under repeated impact conditions had obvious dispersion. In contrast, the mean peak pressure of group B was higher than that of group A, while its variance was relatively smaller, indicating the right lung in group B sustained overall higher peak pressures under repeated impacts, and the results of each impact were more concentrated. Correspondingly, the right lung response in group A fluctuated more severely under repeated impacts, indicating relatively poorer stability. Based on the above results, the B1 test condition was selected as the analysis condition for the numerical model. This further indicates the peak response of right lung under repeated impacts did not exhibit a simple linear variation with increasing impact number, possibly because previous impacts altered the local buffering capacity, stiffness distribution, and contact load-transfer paths of the thoracic cavity. Meanwhile, the propagation

boundaries, reflection conditions, and attenuation process of the intrathoracic pressure wave may also change accordingly, thereby exhibiting significant nonlinear characteristics.

Table 2: Right lung pressure peak values from four repeated blunt impacts.

Group	Ammunition Type	Number of Impacts	Peak 1 (MPa)	Peak 2 (MPa)	Peak 3 (MPa)	Peak4 (MPa)	Mean (MPa)	Variance (MPa)
A	53API-SiC/UHMWPE composite ballistic insert	4	1.06	0.23	0.14	0.83	0.56	0.20
B	53API-SiC/UHMWPE composite ballistic insert	4	0.69	0.98	0.56	0.36	0.65	0.07

3.2 Analysis of the Transmission Mechanism of Blunt Impact Responses in the Rib Cage and Cardiopulmonary System

3.2.1 Finite Element Modeling of the Protective Structure-Human Thorax System

A human thoracic finite element model was constructed based on the Toyota THUMS dummy model. The body size of this model was based on a 50th-percentile adult male, AM50, with a height and body mass of 175 cm and 77 kg [37], respectively. The model was simplified and processed by removing local structures with low relevance to the research objective. Only key anatomical components, including the heart, left and right lungs, stomach, spleen, rib cage, spine, and muscle tissues, were retained, as shown in Fig. 10. All models were established using the HyperMesh meshing software. The muscle model was meshed with tetrahedral elements, whereas the other models were meshed with hexahedral elements. The total number of elements was 3,169,955. The material parameters of the muscle [38] and rib cage [39] were modified, and the materials of each structure are listed in Table 3.

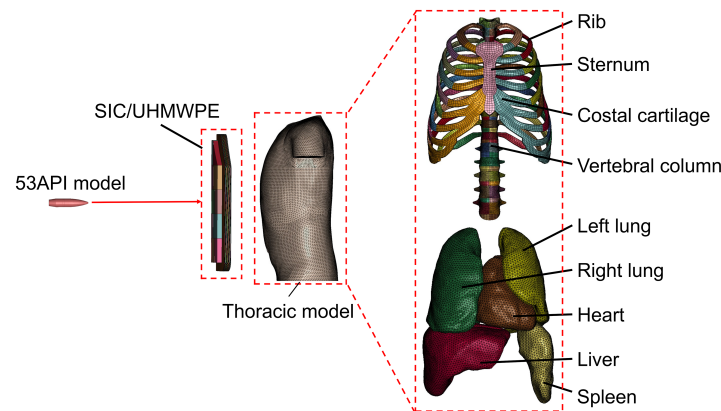


Figure 10: Anatomically detailed finite element model used to investigate the response of the human thorax under the impact loading of the Type 53 API projectile.

Table 3: Material parameters of the human thorax numerical model.

Part	ρ (g/cm ³)	E (GPa)	PR	EPSD	DC	SIGY	ETAN (GPa)	K (GPa)
Ribs	1.08	9.50	0.20	1.00	0.50	1.80×10^{-5}	1.33×10^{-2}	—
Sternum	1.25	9.50	0.25	1.00	0.50	1.80×10^{-5}	1.33×10^{-2}	—

(Continued)

Table 3 (continued)

Part	ρ (g/cm ³)	E (GPa)	PR	EPSD	DC	SIGY	ETAN (GPa)	K (GPa)
Rib cartilage	1.07	2.5×10^{-2}	0.40	1.00	0.50	2.75×10^{-5}	—	—
Vertebral column	1.00	0.04	0.45	—	—	1.80×10^{-5}	1.33×10^{-2}	—
Muscle	1.20	—	—	—	—	—	—	2.90
Heart	1.00	—	—	—	—	—	—	0.10
Lung	1.29×10^{-3}	—	—	—	—	—	—	1.40×10^{-3}
Liver	1.00	—	—	—	—	—	—	4.59×10^{-3}
Spleen	1.00	—	—	—	—	—	—	4.59×10^{-3}

Bone tissues such as the rib cage and spine were modeled using an elastic viscoplastic material model based on continuous damage mechanics theory to characterize their rigid behavior under high-speed impact loading. Specifically, the model was established based on Lemaitre's continuous damage mechanics theory, in which the damage factor D (ranging from 0 to 1) reflects the weakening effect of accumulated microcracks and microvoids within the material on its load-bearing capacity.

$$\tilde{\sigma} = \frac{\sigma}{1 - D} \quad (1)$$

$$\dot{D} = \begin{cases} 0, & \epsilon_{\text{eff}}^p \leq \epsilon_{\text{eff},d}^p \\ \frac{Y}{S} \dot{\epsilon}_{\text{eff}}^p, & \epsilon_{\text{eff}}^p > \epsilon_{\text{eff},d}^p \text{ and } \sigma_1 > 0 \end{cases} \quad (2)$$

here, σ is the nominal stress. $\tilde{\sigma}$ is the effective stress. Y is the damage energy release rate. S is the material damage parameter with a default value of $\sigma_0/200$, and $\dot{\epsilon}_{\text{eff}}^p$ is the equivalent plastic strain rate. Under this condition, when the equivalent plastic strain of the material does not reach the damage threshold $\epsilon_{\text{eff},d}^p$, no damage evolution occurs. By contrast, when the equivalent plastic strain exceeds this threshold and the maximum principal stress is greater than 0, damage begins to accumulate.

Lung tissue was modeled using an elastic model and was approximated as an elastic model. Its shear-carrying capacity was neglected ($G = 0$), and only the volumetric compression response and viscous effects were considered, thereby characterizing the response of lung tissue under impact loading as being dominated by volumetric deformation.

$$K = \frac{E}{3(1 - 2\nu)} \quad (3)$$

$$\dot{p} = -K \dot{\epsilon}_{ii} \quad (4)$$

here, p is the pressure. K is the bulk modulus. $\dot{\epsilon}_{ii}$ is the volumetric strain rate. E is Young's modulus, and ν is Poisson's ratio. In the model, the tensor viscosity coefficient was set to $VC = 0.5$ to suppress numerical oscillations and improve the stability of the impact simulation process.

The heart was modeled using a rubber and foam material model to simulate the deformation response and damage evolution process of human soft tissue under impact loading. This model can effectively characterize the nonlinear compressive behavior of soft tissue materials and the progressive failure response during loading. In this model, the nonlinear response of the material was primarily defined by the uniaxial

loading curve, while the damage evolution was controlled by internal damage parameters. Accordingly, the stress degradation relationship after damage can be expressed as follows:

$$\sigma_{ij} = (1 - D) \sigma_{ij}^0 \quad (5)$$

here, σ_{ij}^0 is the stress tensor in the undamaged state, and σ_{ij} is the actual stress tensor considering damage. When $D = 0$, the material remains intact. When $0 < D < 1$, the material enters the progressive damage stage. When $D = 1$, the material fails completely.

The protective structure–human thoracic finite element model included a protective insert composed of SiC ceramic and a UHMWPE laminate. The specific dimensions were as follows: the ceramic part was $200 \times 250 \times 10 \text{ mm}^3$, the UHMWPE plate was $230 \times 280 \times 12 \text{ mm}^3$, and the radius of curvature of the ballistic insert was 400 mm.

A differentiated meshing strategy was adopted. The $50 \times 50 \times 10 \text{ mm}^3$ ceramic tile at the impact point was discretized using a refined mesh with a minimum element size of 0.3 mm to match the mesh density of the bullet, thereby reducing numerical disturbance and improving computational stability. This region contained 665,600 mesh elements. The remaining ceramic tiles were meshed using a uniform element size of 5 mm. The 12-mm-thick UHMWPE laminate was divided into 40 layers of UHMWPE elements, with every two layers grouped into one part. Mesh refinement was performed in the bullet penetration region, and a total of 2,395,560 mesh elements were finally generated to ensure accurate simulation of the key deformation of the ballistic insert under loading. The projectile model consisted of four parts: the jacket, lead sleeve, steel core, and incendiary agent [40]. The total number of mesh elements was 37,192.

The projectile obliquity angle has little influence on the height and width of the backface deformation in the protective structure [41], and the protective insert was tightly attached to the curved thoracic surface. On this basis, the local normal direction of the thorax at the impact point was defined as the normal projectile incidence direction in this study. The measured projectile trigger velocity in the original experiment was 872.1 m/s. After correcting the incident velocity direction under the 7.2° obliquity condition, the projectile impact velocity in the numerical model was set to 865.2 m/s. The interaction between the rifle projectile and the armor plate was defined using ERODING_SURFACE_TO_SURFACE contact, thereby simulating the possible plastic deformation, erosion failure, and fragmentation of the projectile and protective materials during high-speed impact. Accordingly, the ceramic/UHMWPE composite protective insert structure together with the projectile model constituted the projectile-protective insert interaction module used in the present numerical analysis, as shown in Fig. 11.

The AUTOMATIC_SURFACE_TO_SURFACE contact algorithm was used in the contact modeling of the protective structure-human thorax system to define the interactions between the protective structure and the human thorax, as well as those between the muscle tissue and internal organs, so as to characterize the mechanical interactions and load-transfer processes among different structures under impact loading. The ERODING_SURFACE_TO_SURFACE contact algorithm was used for the interface between the muscle tissue and the rib cage to further account for the possibility of local separation and failure during high-speed impact. The self-contact behavior of all deformable materials was also defined using ERODING_SINGLE_SURFACE to prevent mutual penetration of elements during large deformation. These contact definitions enabled effective simulation of the coupling relationship between the protective structure and the thoracic structures and thereby provided a reliable basis for evaluating the protective performance of the ballistic insert with respect to the human body.

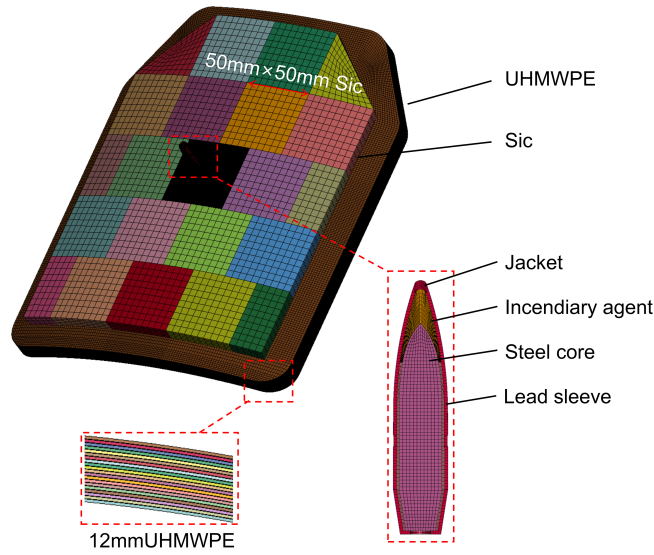


Figure 11: Numerical model of the projectile-ballistic insert interaction.

The BOUNDARY_SPC_SET displacement constraints were applied only to the node sets of the SiC and UHMWPE layers to prevent rigid-body drift of the protective insert during impact and to ensure numerical stability. This treatment improved the stability of the numerical solution process and the reliability of the simulation results.

The ceramic material was modeled using the JH-2 constitutive model, which can describe the evolution of fracture damage during high-speed penetration. In this model, the material strength and pressure parameters were normalized by the corresponding strength and pressure components at the Hugoniot Elastic Limit [42]. Meanwhile, the material strength and damage factor were both expressed as analytical functions of pressure and other state variables, thereby providing the material model with better systematic consistency [43].

$$\sigma^* = \sigma_i^* - D(\sigma_i^* - \sigma_f^*) \quad (6)$$

$$\sigma_i^* = A(P^* + T^*)^N (1 + C \ln \dot{\epsilon}^*) \quad (7)$$

$$\sigma_f^* = B(P^*)^M (1 + C \ln \dot{\epsilon}^*) \leq sfmax \quad (8)$$

here, $\sigma^* = \sigma/\sigma_{HEL}$ is the dimensionless equivalent stress. σ is the true equivalent stress, and σ_{HEL} is the equivalent stress at the Hugoniot Elastic Limit. σ_i^* is the dimensionless equivalent stress of the undamaged material, whereas σ_f^* is the dimensionless equivalent stress of the fully damaged material. The dimensionless pressure $P^* = P/P_{HEL}$ is the pressure at the Hugoniot Elastic Limit. The dimensionless hydrostatic tensile stress $T^* = T/P_{HEL}$, where T is the maximum hydrostatic tensile stress that the material can withstand. The dimensionless strain rate $\dot{\epsilon}^* = \dot{\epsilon}/\dot{\epsilon}_0$, where $\dot{\epsilon}$ is the true strain rate and $\dot{\epsilon}_0 = 1.0 \text{ s}^{-1}$ is the reference strain rate. A , B , C , M , and N are material constants. $sfmax$ is the maximum value attainable by σ_f^* , which was taken as 1.0. It should be noted that when $D = 1$, T^* was set to 0 [44]. The material parameters are listed in Table 4.

Table 4: SiC ceramic material parameters.

ρ (g/cm ³)	G (GPa)	A	B	C	M	N	T (GPa)	$sfmax$	HEL (GPa)	P_{HEL} (GPa)	D_1	D_2	K_1 (GPa)	K_{2-3} (GPa)
3.16	183	0.96	0.35	0	1	1	0.37	0.80	14.57	5.90	0.48	0.48	204.78	0

The UHMWPE fiber bundles in hard body armor are arranged in mutually orthogonal layup directions, and the material therefore exhibits orthotropic mechanical properties. When the x , y , and z directions are taken as their principal elastic axes, the stress-strain relationship can be simplified as follows:

$$\begin{pmatrix} \varepsilon_x \\ \varepsilon_y \\ \varepsilon_z \\ \gamma_{yz} \\ \gamma_{zx} \\ \gamma_{xy} \end{pmatrix} = \begin{pmatrix} \frac{1}{E_A} & -\frac{\nu_{AB}}{E_B} & -\frac{\nu_{AC}}{E_C} & 0 & 0 & 0 \\ -\frac{\nu_{BA}}{E_A} & \frac{1}{E_B} & -\frac{\nu_{BC}}{E_C} & 0 & 0 & 0 \\ -\frac{\nu_{CA}}{E_A} & -\frac{\nu_{CB}}{E_B} & \frac{1}{E_C} & 0 & 0 & 0 \\ 0 & 0 & 0 & \frac{1}{G_{BC}} & 0 & 0 \\ 0 & 0 & 0 & 0 & \frac{1}{G_{CA}} & 0 \\ 0 & 0 & 0 & 0 & 0 & \frac{1}{G_{AB}} \end{pmatrix} \begin{pmatrix} \sigma_x \\ \sigma_y \\ \sigma_z \\ \tau_{yz} \\ \tau_{zx} \\ \tau_{xy} \end{pmatrix} \quad (9)$$

In Eq. (9), E_A , E_B , and E_C denote the elastic moduli in the x , y , and z principal directions, respectively; ν_{AB} , ν_{BC} , ν_{CA} , ν_{BA} , ν_{CB} , and ν_{AC} are the Poisson's ratios between the principal directions; and G_{AB} , G_{BC} , and G_{CA} are the shear moduli in the corresponding planes. The material parameters are listed in Table 5. Here, A , B and C correspond to the x , y , and z material directions, respectively. Only three independent Poisson's ratios are listed, and the remaining Poisson's ratios were obtained from the reciprocal relations for orthotropic elasticity.

Table 5: UHMWPE material parameters.

ρ (g/cm ³)	E_A (GPa)	E_B (GPa)	E_C (GPa)	ν_{BA}	ν_{CA}	ν_{CB}	G_{AB} (GPa)	G_{BC} (GPa)	G_{CA} (GPa)	Y_C (GPa)	Y_T (GPa)	X_C (GPa)	X_T (GPa)
0.97	25	25	8	0.006	0.06	0.06	0.02	3	3	6	6	6	6

The projectile materials were modeled using the Johnson-Cook constitutive model. This model can effectively capture the plastic deformation behavior of most metallic materials under impact loading. For this reason, the Johnson-Cook model was adopted in this study to characterize the constitutive behavior of the materials in each part of the projectile head during penetration [22], as expressed below.

$$\sigma_y = \left(A + B \bar{\varepsilon}^n \right) \left(1 + C \ln \dot{\bar{\varepsilon}}^* \right) (1 - T^{*m}) \quad (10)$$

here, A , B , C , n , and M are material parameters determined experimentally, $\dot{\bar{\varepsilon}}^p$ is the effective plastic strain, $\dot{\bar{\varepsilon}}^* = \dot{\bar{\varepsilon}}^p / \varepsilon_0$ is the dimensionless plastic strain rate, and $T^* = (T - T_r) / (T_m - T_r)$ is the homologous temperature, where T_r is room temperature and T_m is the melting temperature. The Johnson-Cook material parameters of the projectile are listed in Table 6.

Table 6: Johnson-Cook constitutive parameters for bullet parts.

Part	ρ (g/cm ³)	G (GPa)	A (GPa)	B (GPa)	n	C	M	T_r (K)	T_m (K)	D_1	D_{2-5}
Jacket	7.92	77	0.300	0.275	0.150	0.022	1.09	294	1793	2.00	0
Lead sleeve	9.02	7	0.014	0.018	0.685	0.035	1.68	294	600	1.75	0
Steel core	7.85	77	0.792	0.510	0.260	0.014	1.03	294	1793	2.20	0
Incendiary agent	9.02	7	0.014	0.018	0.685	0.035	1.68	294	600	1.75	0

3.2.2 Validation of the Numerical Model

Numerical simulations were performed on the above-established protective structure-human thorax model based on the LS-DYNA explicit dynamic finite element analysis platform. In this analysis, the numerical model adopted the impact location corresponding to the experiment, as shown in Fig. 12, to ensure consistency between the numerical simulation results and the experimental conditions.

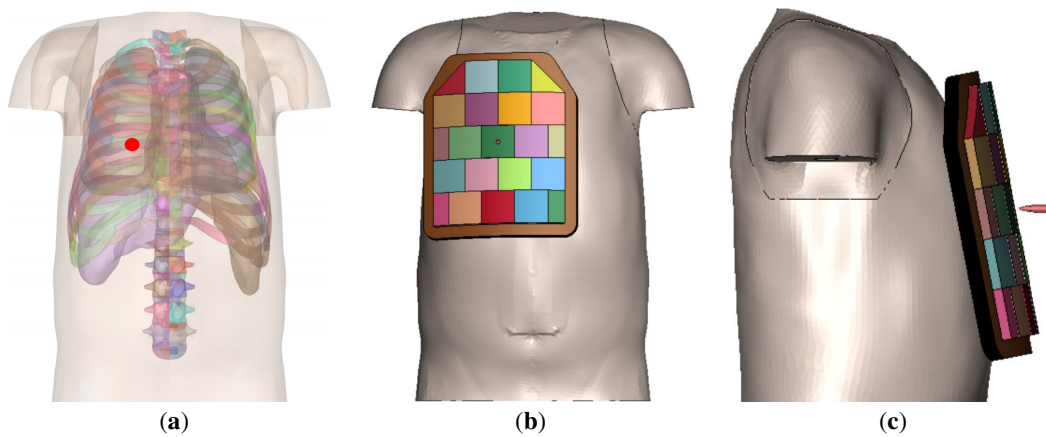


Figure 12: Schematic illustration of the impact location in the numerical model: (a) anatomical impact location; (b) front view of the impact location; (c) side view of the impact location.

Comparative Validation between Experiments and Simulations of Back-Face Bulging in the Composite Ballistic Insert

Fig. 13 compares the back face deformation (BFD) obtained from the numerical simulation with the experimental curves. The numerical simulation curve and the experimental curves showed good agreement in the rising stage, peak stage, and later stable stage. The experimental peak time was 1100 μ s, whereas the simulated peak time was 1200 μ s, with a difference of only 100 μ s. In addition, the average value of the three experimental curves was used as the experimental reference curve. The normalized root mean square error (NRMSE) between the simulated BFD–time curve and the experimental average curve was 1.15%, indicating that the simulation results were highly consistent with the experimental results over the entire time history.

Comparative Validation between Experiments and Simulations of Indentation in the Muscle Material

Fig. 14 shows the experimental and simulated indentation depths of the filling material as a function of time. The final indentation depths of the three experimental groups were 59.59, 60.04, and 60.61 mm, respectively, with an average value of 60.08 mm. The final indentation depth obtained from the simulation

was 58.52 mm, corresponding to an error of approximately 2.59% relative to the experimental average. Meanwhile, both the simulated and experimental curves exhibited a variation pattern of rapid increase followed by gradual stabilization, without obvious nonphysical oscillations or abnormal abrupt changes. This result indicates that the model can stably describe the compression process of the filling material caused by back-face bulging and the transfer process of impact loads toward the thorax.

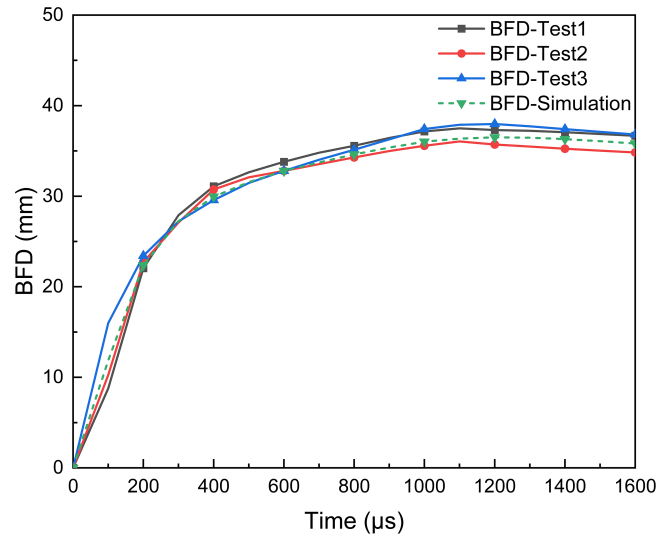


Figure 13: Comparison curves for experimental and simulation validation of bulging in the composite ballistic insert.

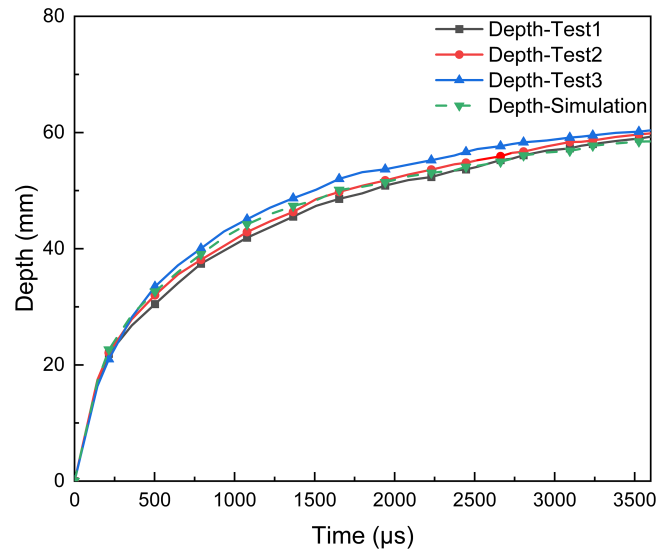


Figure 14: Comparison of indentation depth between the indentation experiment and simulation.

Statistical Characteristics of Repeated-Test Right Lung Pressure Time-History Responses and Comparison with Numerical Simulation

A comparison between the group B experimental results and the numerical simulation results is shown in Fig. 15. In this study, all numerical model results were obtained from the extraction and averaging of response data from multiple representative elements. The overall trends of the pressure curves from the

numerical model and the first-shot B1 group experiment were similar, with both exhibiting transient pressure fluctuations at the initial stage, followed by gradual attenuation and eventual stabilization. This indicates that the model reasonably reproduced the overall evolution process of the pressure response. Meanwhile, both curves exhibited secondary fluctuation features of varying degrees after the primary peak, which may be attributed to the generation of reflected and transmitted waves when the pressure wave propagated to the boundaries of different material structures.

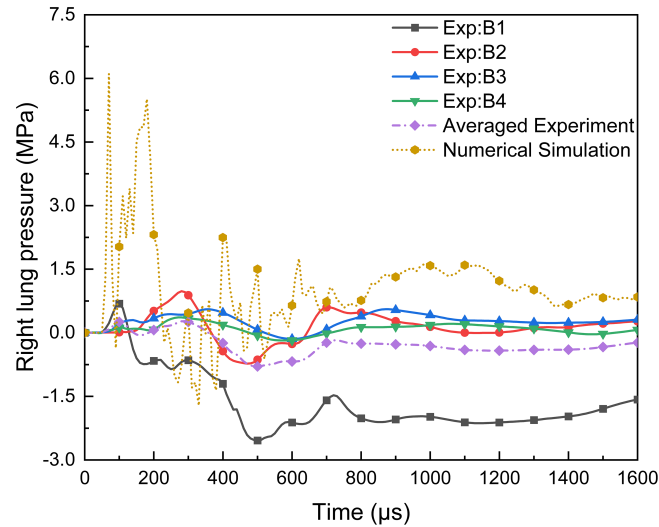


Figure 15: Comparison of the right lung pressure response curves between the group B experiment and the numerical simulation.

Parameter Sensitivity Analysis and Biomechanical Rationality Evaluation of the Right-Lung Pressure Response

The right-lung pressure time history showed a large difference in numerical magnitude between the experimental and numerical results. Therefore, four additional numerical models were constructed in this study. These models used different combinations of rib-cage material parameters and artificial bulk viscosity coefficients. The working conditions were kept consistent among all models. The calculated results were then compared with the corresponding experimental data for validation. Specifically, Model 1 adopted the normal mechanical parameters of rib-cage bone tissue and used the default viscosity coefficients of $Q1 = 1.5$ and $Q2 = 0.06$; Model 2 kept the material parameters unchanged and only reduced the viscosity coefficients to $Q1 = 0.1$ and $Q2 = 0$, which are significantly lower than the reasonable value range in biomechanical simulations; Model 3 kept the material parameters unchanged and only equivalently assigned the rib-cage material as muscle tissue; and Model 4 simultaneously equivalently assigned the rib-cage material as muscle tissue and reduced the viscosity coefficients. Among the above four numerical models, the remaining structural modeling, material parameters, contact relationships, and loading conditions were kept consistent.

Fig. 16 shows that the experimental results and those of the numerical models were basically consistent in the evolution trend of the pressure wave, and all exhibited an obvious initial positive pressure peak followed by an unloading response. Compared with Numerical Model 1, Numerical Model 2 showed only a small change in the positive pressure peak when only the viscosity coefficients were reduced, whereas the negative pressure peak increased from -0.869 to -1.266 MPa, representing an increase of 45.68% relative to Numerical Model 1. This indicates that the viscosity coefficients mainly affected the unloading response after pressure-wave transmission. Under the condition that only the rib-cage material parameters were changed,

the positive pressure peak in the right lung in Numerical Model 3 decreased from 16.570 to 5.993 MPa, which was 63.83% lower than that in Numerical Model 1, whereas the negative pressure peak changed only slightly. This indicates that rib-cage stiffness had a more direct influence on the pressure-wave peak. After Numerical Model 4 simultaneously adopted muscle-equivalent material and reduced viscosity coefficients, the positive pressure peak further decreased to 6.175 MPa, and the negative pressure peak increased markedly to -2.227 MPa. Compared with the experimental negative pressure peak of -2.529 MPa, the relative error was only 11.94%, indicating that the predicted negative pressure response of the right lung during the unloading stage changed significantly after the rib-cage material parameters and numerical damping were adjusted simultaneously.

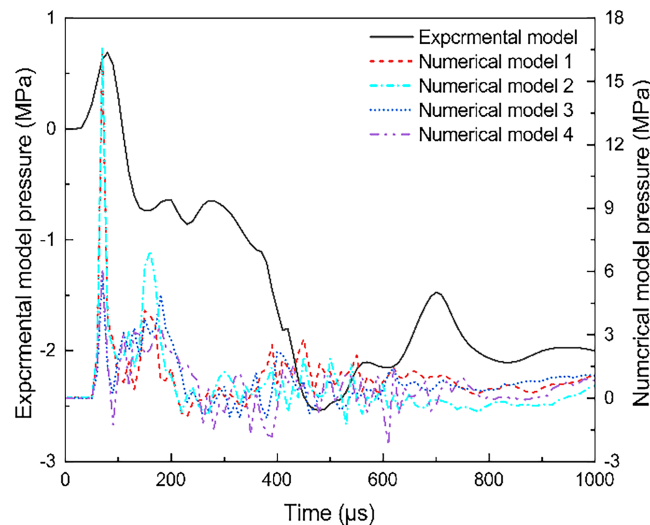


Figure 16: Comparison of right lung pressure responses between the experimental results and the numerical models.

The relatively low positive pressure peak of the right lung measured in the experiment may be related to the arrangement of the pressure sensor. In the experiment, the sensor was attached to the lung surface and was encapsulated and fixed after casting with the muscle material. When the pressure wave propagated through the lung tissue–sensor interface and the surrounding equivalent soft-tissue medium, a certain degree of amplitude attenuation may have occurred. Therefore, the artificial bulk viscosity coefficients mainly affected the negative pressure response and local oscillations during the unloading stage, whereas the rib-cage stiffness had a more direct influence on the positive pressure peak of the right lung. Although abnormal parameter combinations may approach the experimental results in terms of local peak values, their material parameters and artificial bulk viscosity coefficient settings lack biomechanical rationality. In contrast, Model 1 adopted normal rib-cage material parameters and default artificial bulk viscosity coefficients. Although differences in pressure magnitude still existed between the simulation and experimental results, its parameter settings were more consistent with the real physiological and mechanical characteristics of the thoracic structure and could maintain a more reasonable pressure-wave transmission process, load-transfer continuity, and overall response trend. Therefore, Model 1 was more suitable as the model for subsequent analysis of the right-lung pressure response and the load-transfer patterns inside the thoracic cavity.

Comparative Validation of Human Thorax Numerical Models with and without Internal Filling

Fig. 17 presents sectional views of the finite element models of the human thorax with and without internal cavities. This model employed Hyper Mesh software to fill the internal voids of the human thoracic

model with an equivalent muscle-like soft tissue material inward from the skin interface, thereby enclosing the internal organs and tissues. It also removed the original muscle tissue of a certain thickness to enhance the model's ability to transmit impact loads within the thoracic cavity under impact loading.

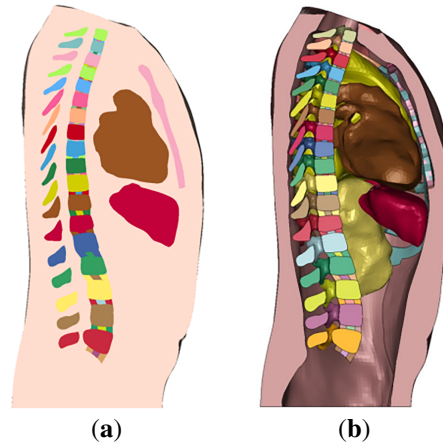


Figure 17: Comparison of human thorax finite element models with and without internal cavities: (a) modified model without internal cavities; (b) original model with internal cavities.

A comparison was made between the original human thorax model, which retained a certain muscle thickness while preserving the cavities among the thoracic organs, and the modified thorax model with internal filling. The sternal acceleration response can more directly characterize the inertial response of the overall thoracic structure and the transmission characteristics of impact loading, and it is also more sensitive to variations in the internal filling state of the thoracic cavity. The acceleration response at the T6 joint of sternum was therefore extracted from both numerical models for comparison, as shown in Fig. 18.

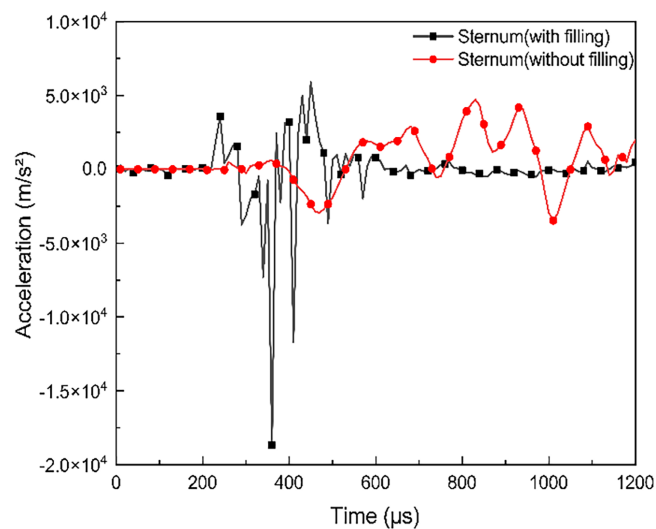


Figure 18: Comparison of acceleration time-history curves between thoracic models with and without internal filling.

The thoracic cavity-filled model responded clearly at 220 μs and reached a peak negative acceleration of approximately $-1.87 \times 10^4 \text{ m/s}^2$ at 360 μs , indicating faster load transmission to the sternum. In contrast, the unfilled model showed a weaker, delayed response with prolonged oscillations, suggesting that internal

gaps enhanced wave reflection and scattering. Removing these gaps improved load transmission continuity and produced a faster, more concentrated sternal response. Thus, the modified model is more suitable for studying thoracic responses under blunt projectile impact.

3.2.3 Analysis of the Protective Mechanism of the Rib Cage under Impact-Induced Pressure Waves

The pressure response during the blunt projectile impact on the protective insert can be divided into two categories: the first is the impact pressure wave generated after the projectile strikes the ceramic layer and rapidly transmitted to the laminate; the second is the pressure response caused by compressive deformation of the ceramic layer under projectile loading, which acts on the laminate and induces compression-dominated loading. Fig. 19 shows the pressure-wave transmission process before and after backplate deformation of the protective structure. The periods of 0–16, 17–39, 39–80, and 81–1600 μs are defined as stages I, II, III, and IV, respectively. Among these stages, stages I and II correspond to the early impact response process before significant backplate deformation occurs, whereas stages III and IV correspond to the pressure, displacement, and equivalent stress response processes of the rib cage and cardiopulmonary tissues after the development of backplate compressive deformation.

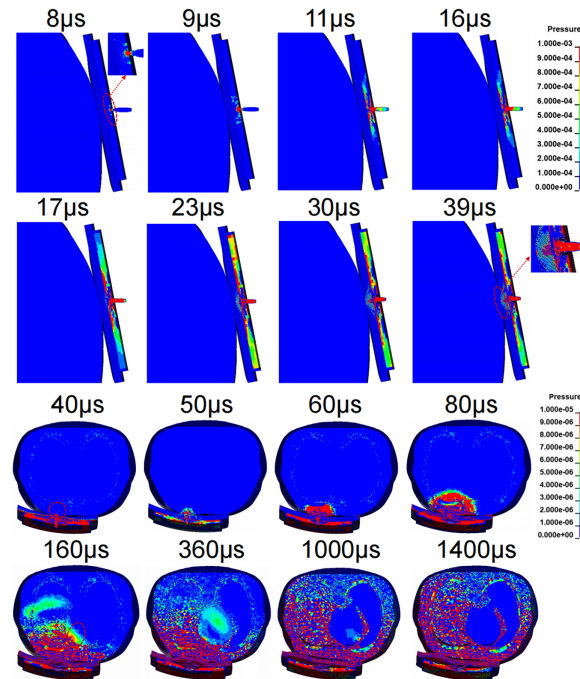


Figure 19: Pressure-wave transmission contours in the protective insert and the thorax behind it before and after backplate deformation.

Fig. 19 further illustrates the pressure-wave propagation process in the rib cage and cardiopulmonary tissues under the compressive deformation of the backing plate. The pressure wave first developed in the regions adjacent to the rib cage, then propagated through the right lung-heart-left lung tissues on the impacted side, and gradually diffused over time to form a broader response zone. To reduce computational cost and improve computational efficiency, the overall process was solved using a time step of 10 μs . The overall impact response time-history curves for the four stages are shown in Fig. 20. Detailed response characteristics of the relevant regions are presented in the following analysis.

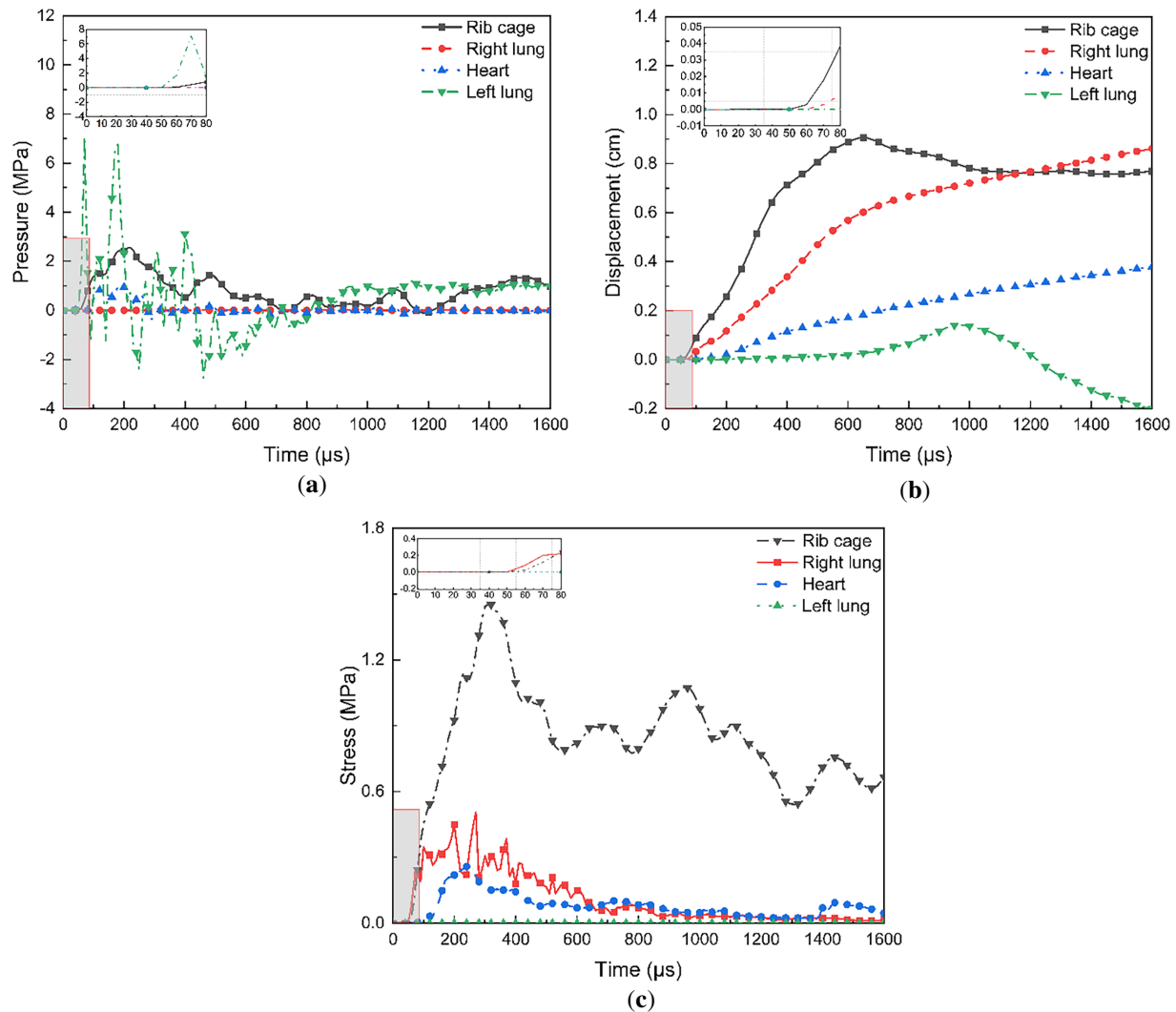


Figure 20: Comparative curves of the pressure, displacement, and equivalent stress responses of the rib cage and cardiopulmonary tissues throughout the entire deformation stage of the protective insert backplate: (a) pressure time-history curves; (b) displacement time-history curves; (c) equivalent stress time-history curves.

Stages I and II: Analysis of the Pressure Response Characteristics of the Protective Insert before Compressive Deformation of the Backplate

Fig. 19 shows the evolution of the protective structure from a ceramic-layer-dominated response to the progressive involvement of the laminate within 0–39 μs . In stage I, the projectile first comes into contact with the ceramic layer, producing a rapid local pressure concentration in the impact region and initiating the initial impact response within the ceramic layer. At 8 μs , the projectile had just come into contact with the ceramic layer, generating a distinct pressure wave. During 9–11 μs , the localized pressure region expanded along the in-plane direction of the ceramic layer. By 16 μs , however, no continuous or distinct propagation path of the impact pressure wave had yet formed within the laminate. It should be noted that, based on the previous work of our research group, Zhu et al. [45] systematically analyzed the early-stage shock-wave transmission mechanism in ceramic/UHMWPE composite protective structures using a coupled Smoothed Particle Hydrodynamics and Finite Element Method (SPH-FEM) numerical model. The

results showed that multiple compression waves were generated within the ceramic after projectile impact, exhibiting pronounced exponential attenuation in both the in-plane and impact directions. In contrast, the present model differs from that study in terms of structural composition, contact definitions, and the physical spacing between components, and therefore could not effectively capture similar high-frequency transient impact-pressure-wave features. Consequently, no new characteristic shock-wave propagation phenomenon was observed in stage I.

Stage II involved compressive deformation of the ceramic layer under the continued action of the projectile, accompanied by progressive transmission of the pressure wave from the ceramic layer into the laminate. At 17 μs , an initial pressure response appeared in the laminate due to compression of the ceramic layer. This response then progressively propagated into the laminate and continued to develop. At 30 μs , the pressure response within the laminate further intensified, forming a relatively continuous high-pressure region near the impact side. By 39 μs , a distinct stress concentration region had formed within the laminate, and the pressure response had further expanded, indicating that the laminate was about to enter the deformation stage. The numerical results of Zhu et al. [45] further indicated that the ceramic-induced compressive longitudinal waves were primarily transmitted to the first equivalent layer of the laminate. They subsequently propagated within the laminate along the fiber and impact directions, further inducing compressive-wave responses that expanded in the in-plane transverse direction and around the impact region. The early-stage response in the present study was dominated by a low-frequency pressure response transmitted from ceramic compressive deformation to the laminate, rather than by a clearly resolved high-frequency transient compressive-wave propagation process. Accordingly, this study focuses on the thoracic response characteristics in Stages III and IV, where the response is dominated by backplate compressive deformation. Further optimization of the protective structure-human thorax model is still required to more accurately capture early-stage impact pressure waves and evaluate their influence on thoracic responses.

Stage III: Analysis of the Pressure, Displacement, and Stress Response Characteristics of the Rib Cage and Cardiopulmonary Tissues during the Initial Stage of Backing-Plate Compression of the Protective Insert

Fig. 21 illustrates the impact response process of the rib cage during 39–80 μs after deformation of the backing plate of the protective insert. At 39 μs , the overall response of the rib cage was weak, and only initial pressure, displacement, and stress changes appeared in the local impacted-side region, indicating that load transfer caused by backing plate compressive deformation was still at the initial stage. At 54 μs , a local pressure concentration region had appeared in the costal cartilage, indicating that the compressive effect generated by deformation of the backing plate of the protective insert had been transmitted to the rib cage. The sternum and ribs also continued to displace inward during this process. At 80 μs , the stress in the sternum increased markedly and a high-stress concentration region appeared, while stress distribution also began to emerge in the impacted-side region of the costal cartilage, indicating the process of stress transmission generated by pressure-wave action on the rib cage.

Fig. 22 illustrates the impact response process of the cardiopulmonary tissues during 39–80 μs after deformation of the backing plate of the protective insert. At 39 μs , the displacement response of the right lung exhibited localized band-like high-response regions, while the stress response also showed a certain degree of spatially nonuniform distribution. This may be attributed to differences in stiffness and structural composition among the ribs, intercostal tissues, and lung tissue. Under impact loading, the ribs underwent slight transient inward displacement and produced local compression on the adjacent lung tissue, causing local loads to be transmitted non-uniformly along the rib distribution direction and ultimately resulting in localized band-like high-response characteristics in the lung tissue. By 54 μs , distinct pressure and stress concentration regions had appeared in the right lung, indicating that the pressure wave generated by rear-face deformation of the protective insert had further propagated into the thoracic cavity. At 60 μs , the

original banded displacement response changed little overall, but a distinct displacement concentration region appeared in the impacted area of the right lung. This may be because the global surface response formed by pressure-wave propagation within the thoracic cavity remained relatively stable over a short period. Meanwhile, compression caused by continued plate deformation had begun to transmit into the thoracic cavity. At 80 μs , localized regions of pressure and stress concentration caused by compression had formed in the heart. This indicates that the compressive effect induced by deformation of the rear surface of the protective insert had further propagated into the thoracic cavity.

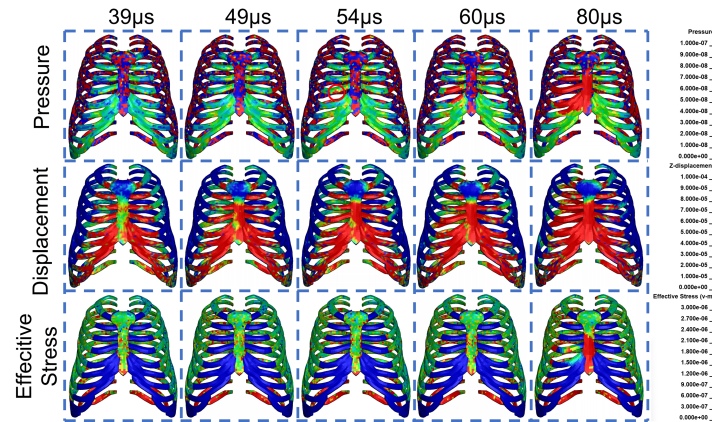


Figure 21: Pressure, displacement, and equivalent stress contours of the rib cage during 39–80 μs backface deformation of the protective insert.

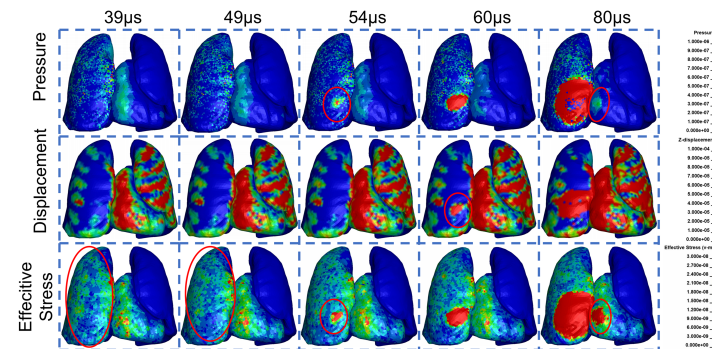


Figure 22: Pressure, displacement, and equivalent stress contours of the cardiopulmonary tissues during 39–80 μs backface deformation of the protective insert.

Stage III corresponds to the initial stage of compressive deformation of the protective insert backplate. During this stage, the load was mainly transmitted inward along the contact path between the protective structure and the anterior rib cage. The sternum and costal cartilage are important components of the anterior rib cage and are highly representative of the early response and its evolution. Therefore, the response characteristics of the sternum and costal cartilage were analyzed in this stage. Fig. 23 presents the evolution of the impact responses of the sternum, costal cartilage, and cardiopulmonary tissues during stages I, II, and III within 0–80 μs . Localized responses could still be observed during the first two stages due to numerical noise. At 56 μs , the right lung pressure increased markedly, indicating that the compression generated by deformation of the protective insert backplate had begun to act on the thoracic cavity interior. The displacement of the costal cartilage began to increase linearly at 70 μs , and the stress in the sternum also

increased rapidly from 72 μs . These results indicate that during the initial compression stage of the protective insert backplate, the rib cage attenuated the impact load and thereby reduced direct injury to the lungs and heart.

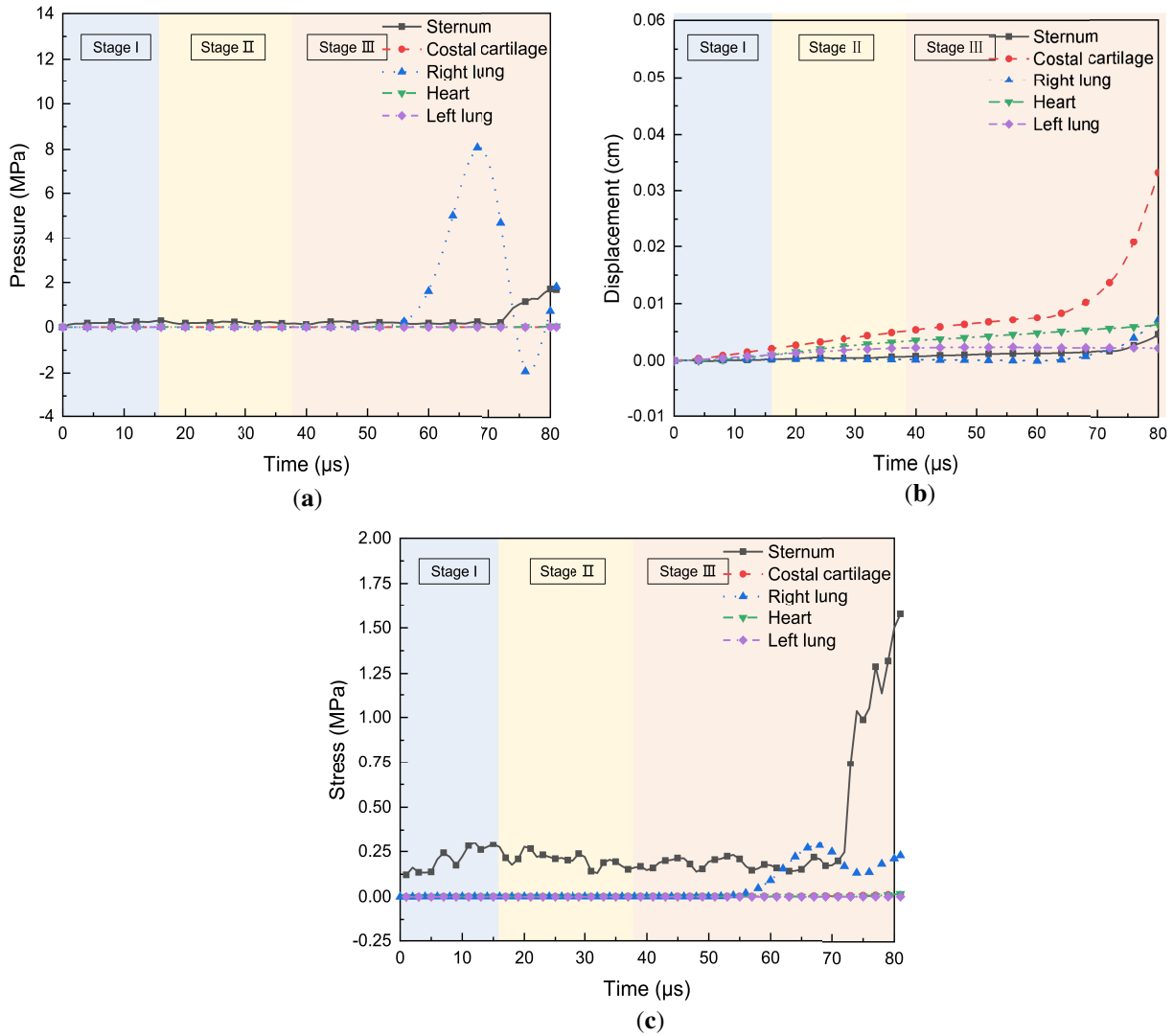


Figure 23: Comparison of the pressure, displacement, and equivalent stress responses of the sternum, costal cartilage, and cardiopulmonary tissues before deformation of the protective insert backplate: (a) pressure time-history curves; (b) displacement time-history curves; (c) equivalent stress time-history curves.

Fig. 24 presents the fitting results of the displacement response curves of the sternum, costal cartilage, heart, and lungs during the initial stage under backing plate compressive deformation. A unified $y(x) = a + bx + ce^{dx}$ linear-exponential function was constructed for fitting because the linear term can reflect the overall evolution trend of the displacement response, whereas the exponential term can characterize nonlinear enhancement or hysteretic amplification under impact loading. Owing to the limited number of data points representing stage-dependent variations in the actual left lung curve, the R^2 value was relatively low. Nevertheless, the fitting still captured the overall time-dependent trend and principal characteristics of left lung displacement.

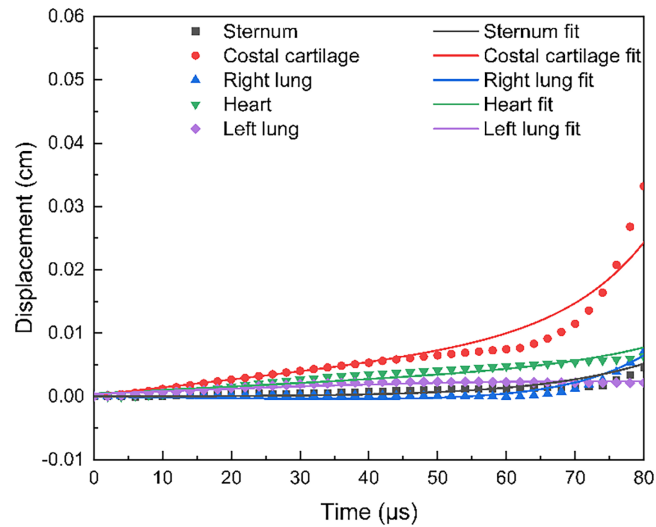


Figure 24: Displacement time fitted curves for the sternum, costal cartilage, and cardiopulmonary tissues during the initial stage of backface compressive deformation.

Table 7 parameters and the fitted curves in Fig. 24 together indicate the roles of the fitting parameters in the displacement response. The left lung, which exhibited a larger constant term a , showed a relatively higher overall initial level of the fitted curve. This suggests that a larger constant term a may correspond to a higher baseline offset of the fitted displacement curve. For the sternum and right lung, where $a = 0$, the constant baseline offset component was absent. However, the initial fitted displacement was still determined by $y(0) = a + c$. Therefore, parameter a may mainly reflect the baseline offset of the fitted curve, while the initial displacement level is governed by the combined effect of a and c . The values of b for the sternum and right lung were both less than 0, corresponding to relatively small changes in the early and middle stages of the curves, whereas the values of b for the costal cartilage, heart, and left lung were all greater than 0, corresponding to an overall increasing trend over time. This suggests parameter b may represent the linear evolutionary trend of tissue displacement with time, reflecting the overall direction of growth or suppression of tissue displacement under backplate compressive deformation. For parameter c , the values for the sternum, costal cartilage, heart, and right lung were all greater than 0, and all exhibited varying degrees of late-stage elevation. In contrast, the value for the left lung was less than 0, with a value of -1.0×10^{-3} , and its curve showed a relatively small overall increase with no obvious nonlinear enhancement. Parameter c may correspond to the amplitude direction of the exponential term, reflecting the degree of enhancement or suppression of the nonlinear displacement response. For parameter d , the sternum had the largest value of 0.63. However, because its c value was relatively small and b was negative, the curve showed elevation only in the late stage. The d value of the costal cartilage was lower than that of the sternum, but under the combined effects of positive b and positive c , its displacement increased continuously and was rapidly amplified in the late stage. Both c and d for the right lung were small, resulting in relatively mild changes during the early and middle stages, with elevation appearing only later. For the left lung, because c was negative and d was small, the late-stage nonlinear enhancement was the weakest. This suggests parameter d may correspond to the development rate of the exponential term over time, reflecting the rate at which the nonlinear response is established. Parameters c and d may also exhibit a coupling relationship. When c is positive and has a certain magnitude, a larger d may correspond to pronounced late-stage nonlinear growth. For tissues with a small or negative c , an increase in d alone may not necessarily produce significant displacement amplification.

Table 7: Parameters and R2 for displacement fitting functions of the sternum, costal cartilage, and cardiopulmonary tissues.

Equation	$y(x) = a + bx + ce^{dx}$				
Parameters	a	b	c	d	R^2
Sternum	0	-2.0×10^{-6}	3.5×10^{-5}	0.63	0.96
Costal cartilage	2.0×10^{-4}	1.2×10^{-4}	1.5×10^{-5}	0.09	0.93
Right lung	0	-1.6×10^{-5}	9.0×10^{-6}	0.08	0.99
Heart	4.6×10^{-4}	5.3×10^{-5}	1.0×10^{-5}	0.07	0.98

The displacement differences among tissues under the same fitting function were mainly determined by b , c , and d . Parameter b may reflect the linear displacement evolution trend, parameter c may reflect the magnitude and direction of the nonlinear response, and parameter d may reflect the establishment rate of the nonlinear response. The results showed that the costal cartilage was the first to exhibit a pronounced displacement response and had a larger displacement amplitude, whereas the responses of the internal organs were relatively delayed in time and exhibited limited overall amplitudes. The right lung and heart exhibited faster response growth in the later stage, accompanied by larger amplitude changes. In contrast, the left lung, which was more strongly buffered by the surrounding tissues, showed slower response growth and a more gradual overall variation process.

Stage IV: Analysis of the Pressure, Displacement, and Stress Response Characteristics of the Rib Cage and Cardiopulmonary Tissues under Large Deformation of the Protective-Insert Backing Plate

Fig. 25 illustrates the impact response process of the rib cage from 81 to 1600 μ s under significant backplate deformation of the protective insert. At 100 μ s, the displacement and stress were mainly concentrated in the costal cartilage region on the impacted side. Subsequently, the displacement and stress responses gradually propagated along the sternum and bilateral ribs. The overall response of the anterior rib cage continued to intensify, and the stress distribution area progressively expanded. This indicates that the impact load was mainly transmitted through the sternum and costal cartilage regions to the surrounding structures, ultimately affecting the deformation and stress distribution of the entire rib cage.

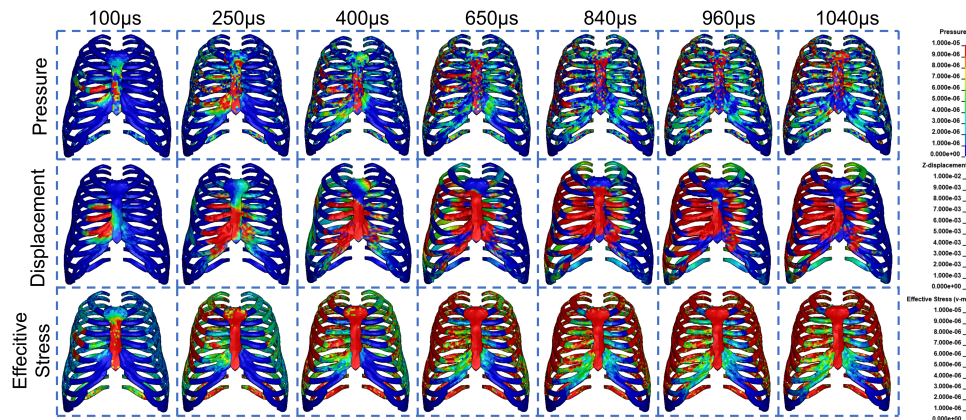
**Figure 25:** Pressure, displacement, and equivalent stress contours of the rib cage during 81–1600 μ s after backface deformation of the protective insert plate.

Fig. 26 shows the impact response process of the heart and lung tissues from 81 μ s to 1600 μ s under significant compressive deformation of the protective structure backplate. At 100 μ s, the right lung on the

impacted side was the first to exhibit pronounced pressure, displacement, and stress responses, whereas the overall response of the left lung remained relatively weak. Subsequently, the response region within the right lung gradually expanded, while the response in the heart region continued to intensify, with the overall displacement and stress distributions continuously developing. The results indicate that the transmission of impact load within the heart and lung tissues exhibited a clear temporal sequence and ultimately affected the deformation and stress distribution of the overall cardiopulmonary tissues.

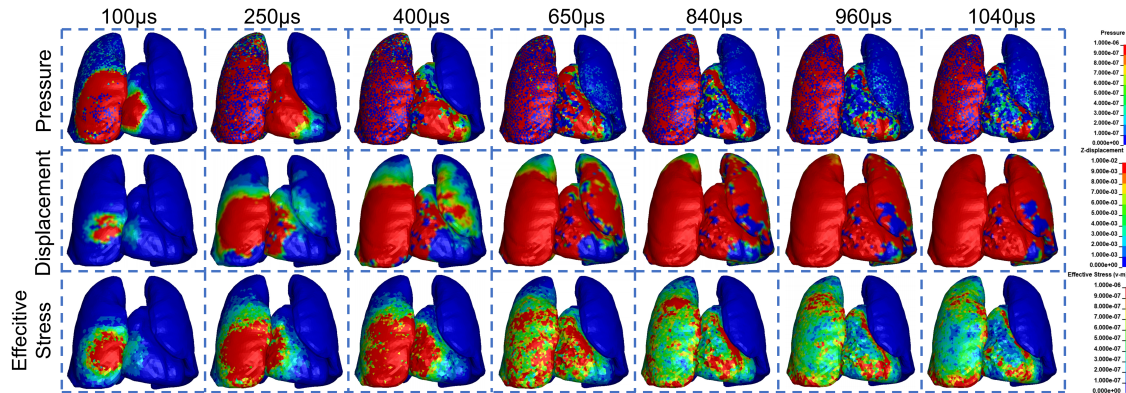


Figure 26: Pressure, displacement, and equivalent stress contours of cardiopulmonary tissues during 81–1600 μ s after backface deformation of the protective insert.

The impact load became further concentrated and acted on the representative load-bearing region of the rib cage as backface compressive deformation developed markedly in stage IV. Therefore, the costal cartilage corresponding to the projectile impact location was selected as the representative region to characterize the response of the key rib cage region under pronounced compressive deformation and its influence on cardiopulmonary tissue responses. Fig. 27 presents the time-history curves of impact responses at representative elements of rib cage and cardiopulmonary tissues. The pressure of the rib cage was overall higher than that of cardiopulmonary tissues, with pronounced fluctuations occurring during 81–450 μ s, followed by a gradual transition to a relatively stable state. The pressure response of the left lung exhibited severe fluctuations. This may be because it was located in the indirect transmission region of the impact load, where reflections from thoracic boundaries and wave superposition more readily induced oscillatory pressure responses. The right lung exhibited a pronounced displacement response at the initial stage, with its displacement amplitude continuously increasing to 0.60 cm at 650 μ s, after which the growth rate markedly decreased and gradually approached a stable state. This indicates that the impact load was preferentially transmitted to the lung tissue on the impacted side (right lung) during the early stage, resulting in significant deformation. The displacement response of the left lung was markedly delayed and showed a smaller amplitude, reaching a maximum value of 0.14 cm at 960 μ s, which was only 23% of the peak value of the right lung. The displacement response of the heart was intermediate between those of the bilateral lungs, indicating that it occupied a central load-bearing position within the thoracic cavity. The displacement response exhibited a sequentially delayed transmission pattern from the right lung to the heart and then to the left lung. The stress response first acted on the rib cage. As indicated in Stage III, it rose rapidly within 50–60 μ s, reached a peak value of 1.46 MPa at 310 μ s, and then decayed rapidly. The right lung reached its maximum stress of 0.51 MPa at 270 μ s, representing a 65.3% reduction relative to the peak stress of the rib cage, whereas the peak stress of the heart was reduced by 82.2% relative to that of the rib cage. These results indicate that the rib cage reduced the stress directly transmitted to the lungs and heart, thereby protecting the internal organs from excessive impact.

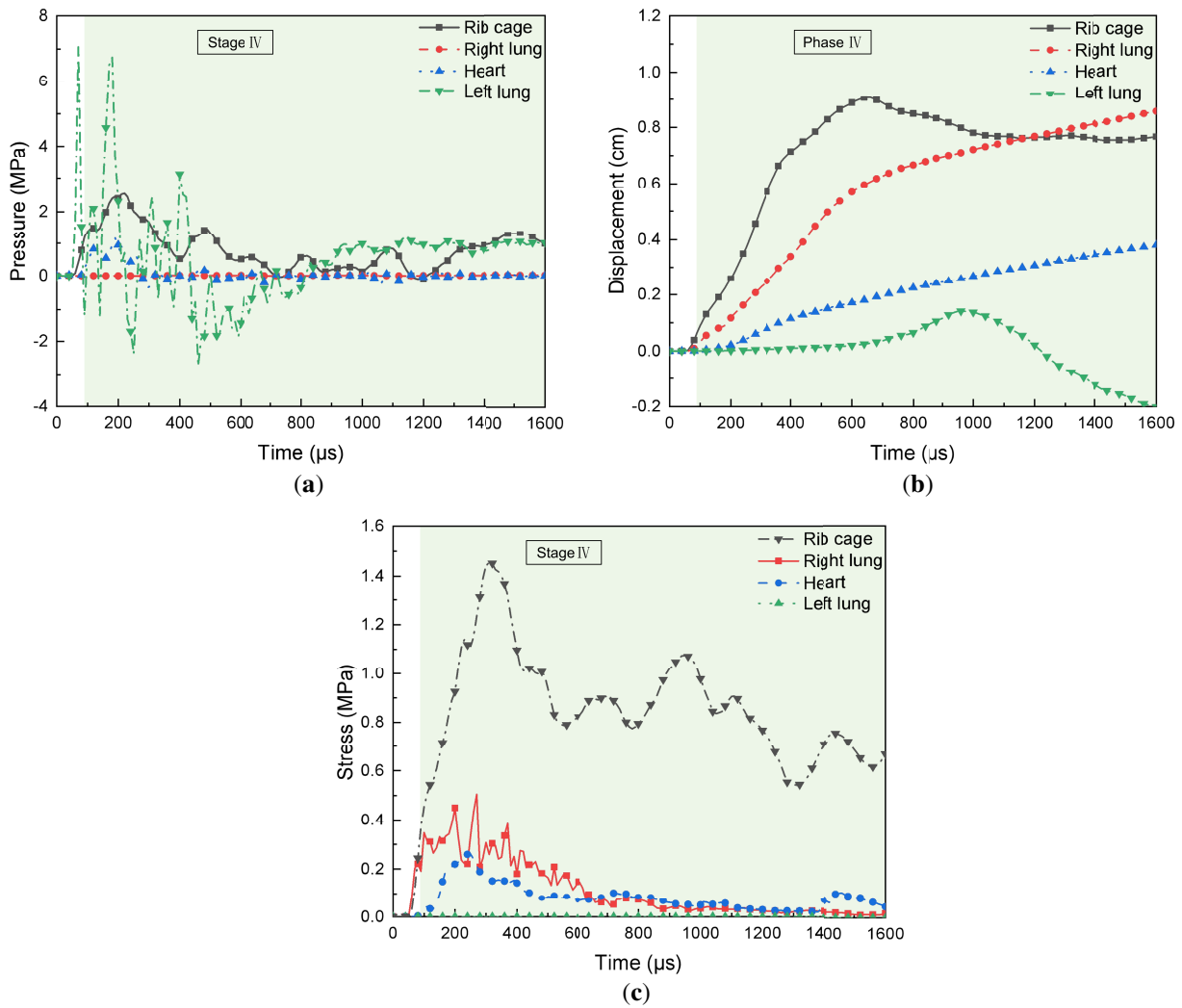


Figure 27: Comparison of the pressure, displacement, and equivalent stress responses of the rib cage and cardiopulmonary tissues during 81–1600 μs after deformation of the protective insert backplate: (a) pressure time-history curves; (b) displacement time-history curves; (c) equivalent stress time-history curves.

As shown in Fig. 27c, the stress evolution of the rib cage, right lung, and heart exhibited a common pattern, characterized by a rapid initial rise to a primary peak, followed by gradual decay and the retention of a certain residual stress at the later stage. Therefore, a unified power-exponential function, as listed in Table 8, was constructed for all three tissues. The corresponding curves are shown in Fig. 28.

Table 8: Parameters and R^2 for stress fitting functions of the rib cage and cardiopulmonary tissues.

Equation	$\sigma(x) = A(1 - e^{-x/t_r})^p e^{-x/t_d} + C(1 - e^{-x/t_c})$						
Parameters	A	t_r	p	t_d	C	t_c	R^2
Costal cartilage	2.71	96.85	5.00	306.37	0.72	365.72	0.80
Right lung	0.94	88.69	2.64	285.19	0.01	200.00	0.90
Heart	1.50	51.62	30.00	136.97	0.06	215.97	0.86

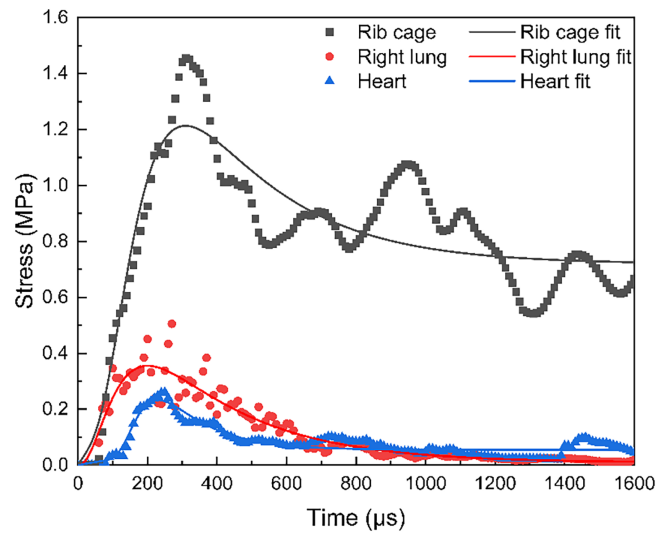


Figure 28: Fitted stress time curves for the rib cage, heart, and lungs under backplate compression of protective insert.

The rib cage exhibited the largest A value of 2.71 and the highest primary peak amplitude in the fitted curve, whereas the right lung showed the smallest A value and the lowest peak, suggesting that A may play a dominant role in governing the transient response amplitude. The heart had the smallest t_r value, corresponding to a shorter stress rise stage and a more concentrated primary peak, suggesting that t_r may determine the time scale of early-stage stress buildup. For parameter p , the heart showed the largest value of 30.00, substantially higher than those of the rib cage and right lung, corresponding to the steepest pre-peak curve and a more concentrated primary peak, whereas the right lung had the smallest p value and a more gradual pre-peak rise. These findings suggest that p may control the steepness of the peak initiation stage. The rib cage exhibited the largest t_d value of 306.37, followed by the right lung, while the heart had the smallest value of 136.97. This trend is consistent with the slower post-peak decay of the rib cage and right lung and the faster decay of the heart shown in Fig. 27, indicating that t_d may govern the duration of post-peak attenuation. For parameter C , the rib cage had the largest value, corresponding to the highest residual stress level in the later stage, whereas the C value of the right lung was close to zero, indicating difficulty in maintaining sustained residual stress after the primary peak. These results suggest that C may govern the amplitude level of residual stress. The right lung exhibited the smallest t_c value of 200.00, which corresponded to a faster buildup of the residual term during the late-stage residual stress formation process in Fig. 27. Although the rib cage had the largest t_c value, its residual stress level remained relatively high in the later stage because it also had the largest C value. These results suggest that t_c may determine the time scale of residual stress formation.

The morphological differences among the stress curves of different tissues under the same fitting function were mainly determined by A , t_r , p , and t_d . Specifically, A may dominate the intensity of the primary peak response, t_r may determine the time scale of the early-stage peak formation, p may control the steepness of the pre-peak rising phase, and t_d may govern the duration of the post-peak decay process. Correspondingly, the results indicated the rib cage, which was located close to the primary load-transfer path and served a supporting role, exhibited higher transient responses and residual stresses. By contrast, the more compliant right lung and heart showed lower stress amplitudes and weaker persistence.

Local Thoracic Injury Risk Evaluation Index

Nsiampa et al. [46] pointed out that the experimentally measured global thoracic displacement is often an averaged response and cannot fully reflect the local displacement in the impact region. In addition, both the displacement measurement location and measurement direction can affect the result of $VC_{\max}$. Therefore, this study selected node sets on the outer surface and inner wall of the muscle layer. These node sets were located directly opposite the center of back-face bulging of the ballistic insert. The relative displacement between the two node sets along the impact direction was then calculated. Based on this displacement, the local viscous response index VC_{local} was established. This index was used to quantitatively evaluate the local compression response and potential injury risk in the impact region. Here, D_{local} denotes the change in relative displacement between the two node sets along the impact direction and is defined as the chest-wall compression. $D_{0,\text{local}}$ denotes the initial distance between the outer surface and inner wall of the muscle layer along the impact direction, which was 33.71 mm, and C_{local} represents the local normalized compression. The calculation formulas are as follows:

$$VC_{\text{local}}(t) = V_{\text{local}}(t) \times C_{\text{local}}(t) \quad (11)$$

$$C_{\text{local}}(t) = \frac{D_{\text{local}}(t)}{D_{0,\text{local}}} \quad (12)$$

$$V_{\text{local}}(t) = \frac{dD_{\text{local}}(t)}{dt} \quad (13)$$

Table 9 presents the calculation results of the local compression response of the muscle layer in the impact region and the VC_{local} index. The maximum local compression of the muscle layer in the impact region was 1.22 mm, corresponding to a maximum local compression ratio of 3.62%. This indicates that the muscle layer underwent a certain degree of local compression under the back-face compression of the ballistic insert, but the compression amplitude was small relative to its initial thickness. The peak local compression velocity was 17.24 m/s and occurred at 70 μs , indicating that a short-duration rapid compression process occurred between the outer surface and inner wall of the muscle layer during the initial impact stage. Because the local compression ratio was relatively low, the final $VC_{\text{local,max}}$ was 0.249 m/s. In classical whole-thorax VC studies [47], $VC_{\max} = 1.0$ m/s is often used as a reference level for severe thoracic injury risk, corresponding to approximately a 25% risk of AIS 4+ thoracic injury; $VC_{\max} = 1.3$ m/s is often reported as corresponding to approximately a 50% severe thoracic injury risk level. The $VC_{\text{local,max}} = 0.249$ m/s obtained in this study was markedly lower than the above whole-thorax VC risk reference levels, indicating that although short-duration rapid compression occurred in the impact region, the intensity of the viscous response jointly characterized by the local compression amplitude and compression velocity was relatively low.

Table 9: Local compression response and viscous response index of the muscle layer in the right-side impact region.

Key Indicators	Result	Time to Peak (μs)
$D_{\text{local,max}}/\text{mm}$	1.221	200
$C_{\text{local,max}}/\%$	3.621	200
$V_{\text{local,max}}/\text{m/s}$	17.238	70
$VC_{\text{local,max}}/\text{m/s}$	0.249	80

Impact Response Characteristics of the Anterior and Posterior Surfaces of the Sternum and Costal Cartilage and Analysis of Protective Mechanisms and Potential Injury Risks

Fig. 29 compares the pressure, acceleration, and equivalent stress responses on the impact-facing and posterior surfaces of both structures, in order to further reveal the protective mechanisms and potential injury risks of the two structures during load transfer. The pressure on the impact-facing surface of the sternum exhibited a relatively high amplitude and high-frequency fluctuations within 50–210 μs , with a maximum pressure of approximately 8.698 MPa. The pressure peak on the posterior surface was only 39.3% of that on the impact-facing surface and showed a time delay, indicating that the sternum had a relatively obvious pressure attenuation effect. The acceleration on its impact-facing surface was dominated by a short-duration high-frequency response, with a peak value of $9.43 \times 10^5 \text{ m/s}^2$. The posterior surface was affected by overall inertial motion and bending deformation, and its peak appeared earlier and was higher. The equivalent stress rapidly entered a quasi-stable fluctuation stage of approximately 2.315 MPa, and the stress on the posterior surface decreased in the later stage. This indicates that the sternum had a certain energy regulation and dispersion effect during load transfer, while local microdamage and internal dissipation may also have occurred.

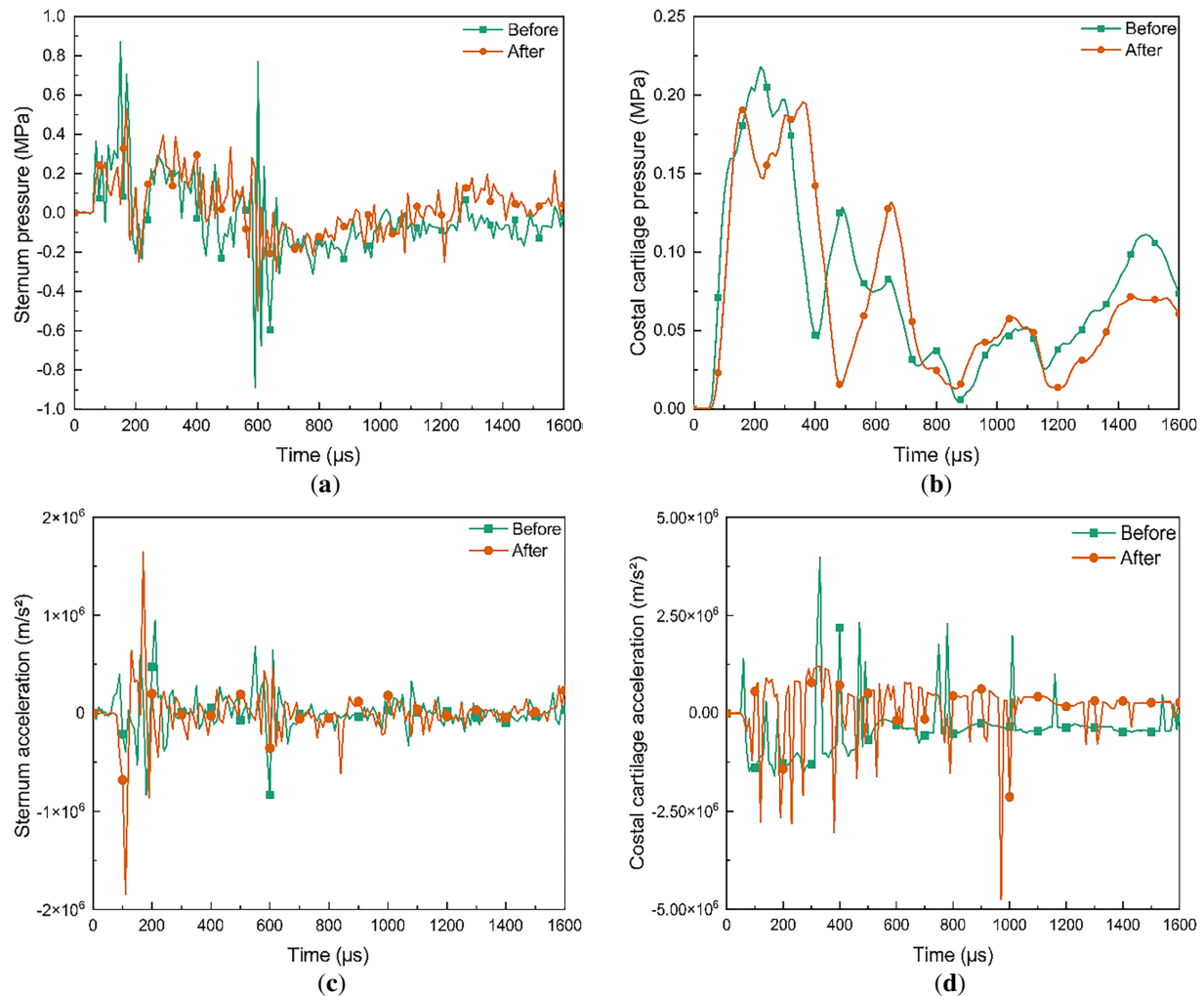


Figure 29: (Continued)

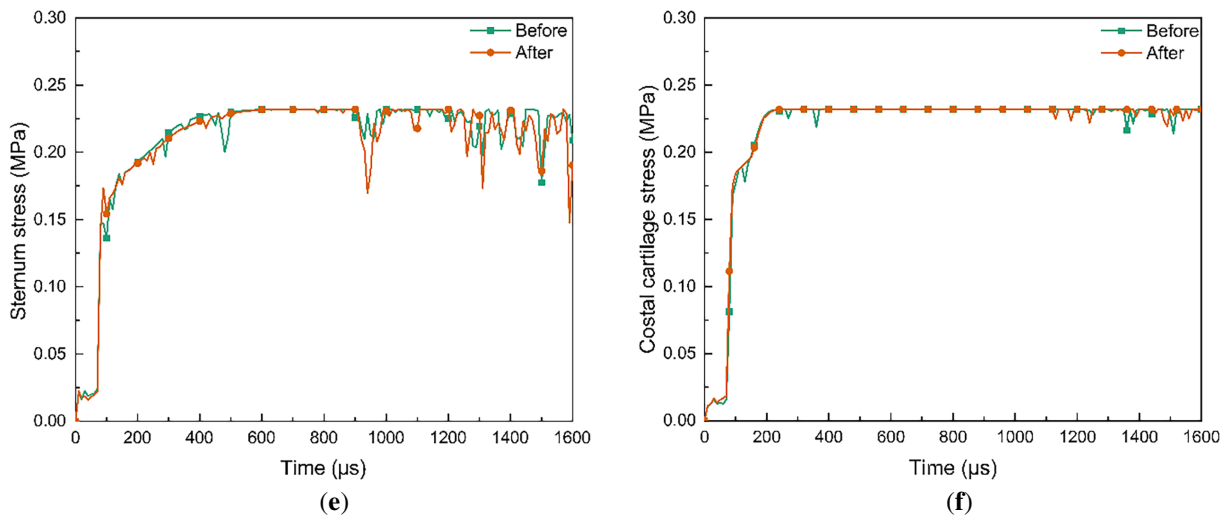


Figure 29: Pressure, acceleration, and stress time-history curves of the front and back surfaces of the sternum and costal cartilage: (a) sternum pressure; (b) costal cartilage pressure; (c) sternum acceleration; (d) costal cartilage acceleration; (e) sternum stress; (f) costal cartilage stress.

For the costal cartilage, the pressure peak on the impact-facing surface was approximately 2.180 MPa, and the pressure attenuation on the posterior surface was approximately 10.8%. This indicates that its pressure attenuation effect along the thickness direction was weaker than that of the sternum, and the pressure response could be transmitted more rapidly to the posterior surface. Its acceleration response showed obvious alternating positive and negative oscillations, reflecting that the costal cartilage exhibited a certain inertial response and bending–compression coupled deformation under impact loading. The equivalent stresses on the anterior and posterior surfaces both increased rapidly during the initial impact stage and quickly entered a relatively stable stage, with only a small difference between them. This indicates that the costal cartilage mainly participated in energy dispersion through compliant deformation and vibration response, but sustained vibration and a small attenuation amplitude may lead to dynamic response accumulation and potential injury risk.

Impact Response Characteristics of the Sternum–Soft Tissue and Costal Cartilage–Soft Tissue Coupling Interfaces and Analysis of Protective Mechanisms and Potential Injury Risks

This study further investigated the load-transfer relationships at the sternum–soft tissue and costal cartilage–soft tissue interfaces. It also analyzed the response differences and potential injury risks of the sternum and costal cartilage in their protective roles. The anterior and posterior soft-tissue surfaces in contact with the two structures were selected as characteristic locations. The time-history responses of pressure, acceleration, and equivalent stress were comparatively analyzed, and the results are shown in Fig. 30.

In the sternum–soft tissue coupling system, the pressure of the anterior soft tissue reached its peak at 250 μ s. The posterior pressure peak appeared with a delay and was only 54% of the anterior peak, accompanied by negative-pressure rebound, indicating that the sternum–soft tissue interface exhibited certain attenuation, delay, and fluctuation characteristics during load transfer. The acceleration response was mainly concentrated within 90–200 μ s after impact and then decayed rapidly, showing a short-duration high-frequency response. The equivalent stress results showed that the stress in the anterior soft tissue increased first and then gradually decayed. Although the posterior stress peak was delayed, it remained at a certain level during the middle and later stages, indicating that load redistribution occurred between the sternum and the adjacent soft tissues.

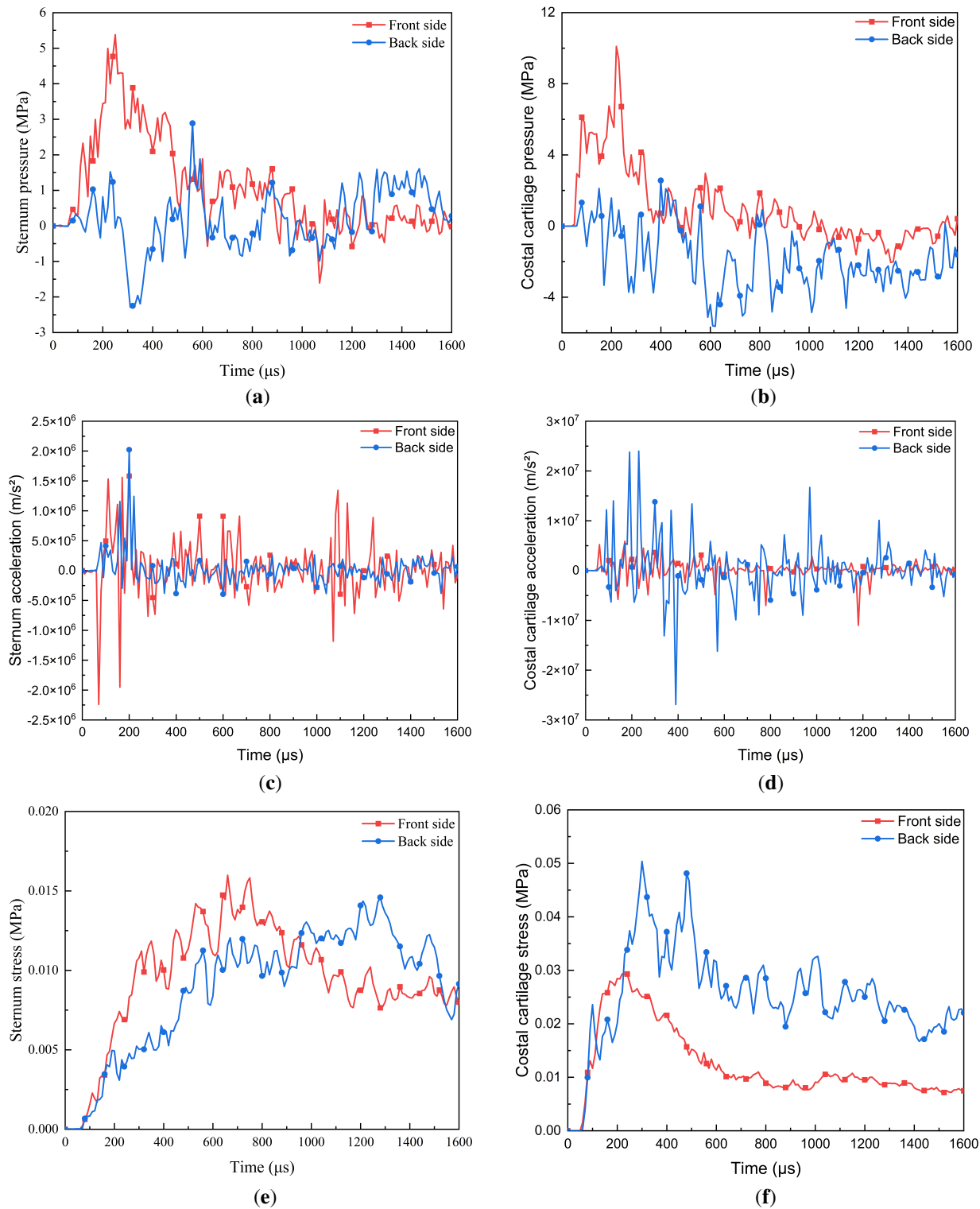


Figure 30: Pressure, acceleration, and stress time-history curves at the front and back soft-tissue surfaces of the sternum and costal cartilage: (a) sternum pressure; (b) costal cartilage pressure; (c) sternum acceleration; (d) costal cartilage acceleration; (e) sternum stress; (f) costal cartilage stress.

In the costal cartilage–soft tissue coupling system, the pressure of the anterior soft tissue reached its peak at approximately 220 μs , with a value of about 0.10 MPa. The posterior pressure peak was only 25% of the anterior peak and lagged by approximately 180 μs , accompanied by negative-pressure fluctuations, indicating that reflection and oscillation of the pressure response occurred at the costal cartilage–soft tissue interface and within the soft tissue. The acceleration response increased from $5.9 \times 10^6 \text{ m/s}^2$ on the anterior side to approximately $2.7 \times 10^7 \text{ m/s}^2$ on the posterior side, indicating that the costal cartilage region was dominated by medium- to low-frequency responses and was accompanied by certain bending and compressive deformation. Overall, the sternum mainly played a role in load attenuation and regulation, whereas the costal cartilage participated more in load dispersion and redistribution through compliant deformation and vibration response.

Comparative Analysis of the Differentiated Protective Mechanisms of the Sternum and Costal Cartilage

Fig. 31 compares the pressure, acceleration, and stress time-history responses of impact loads along the transmission path of the sternum and costal cartilage, namely, the front soft tissue, the impact-facing surface, the back-facing surface, and the back soft tissue. Along the sternal pathway, the response peaks were mainly concentrated on the impact-facing surface and exhibited attenuation, delay, and redistribution during transmission toward the posterior surface and the posterior soft tissue. This indicates that the sternum mainly played a role in rigid shielding and load regulation, although local stress concentration and inertial response may lead to certain potential injury risks. Along the costal cartilage pathway, the attenuation amplitudes of the response quantities were relatively small and were accompanied by sustained vibration, alternating positive and negative fluctuations, and bending–compression coupled deformation. This indicates that the costal cartilage mainly participated in load dispersion through compliant deformation and vibration response, while dynamic response accumulation may also occur. Overall, the sternum and costal cartilage participated in impact load regulation through rigid shielding and flexible energy dissipation, respectively, jointly forming a differentiated synergistic protective mechanism of the rib cage.

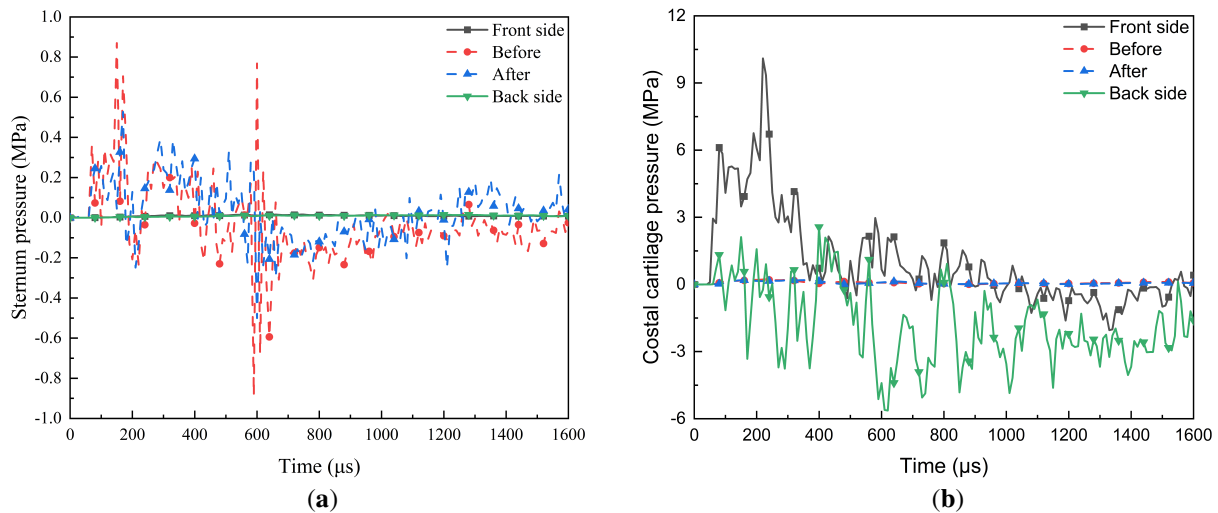


Figure 31: (Continued)

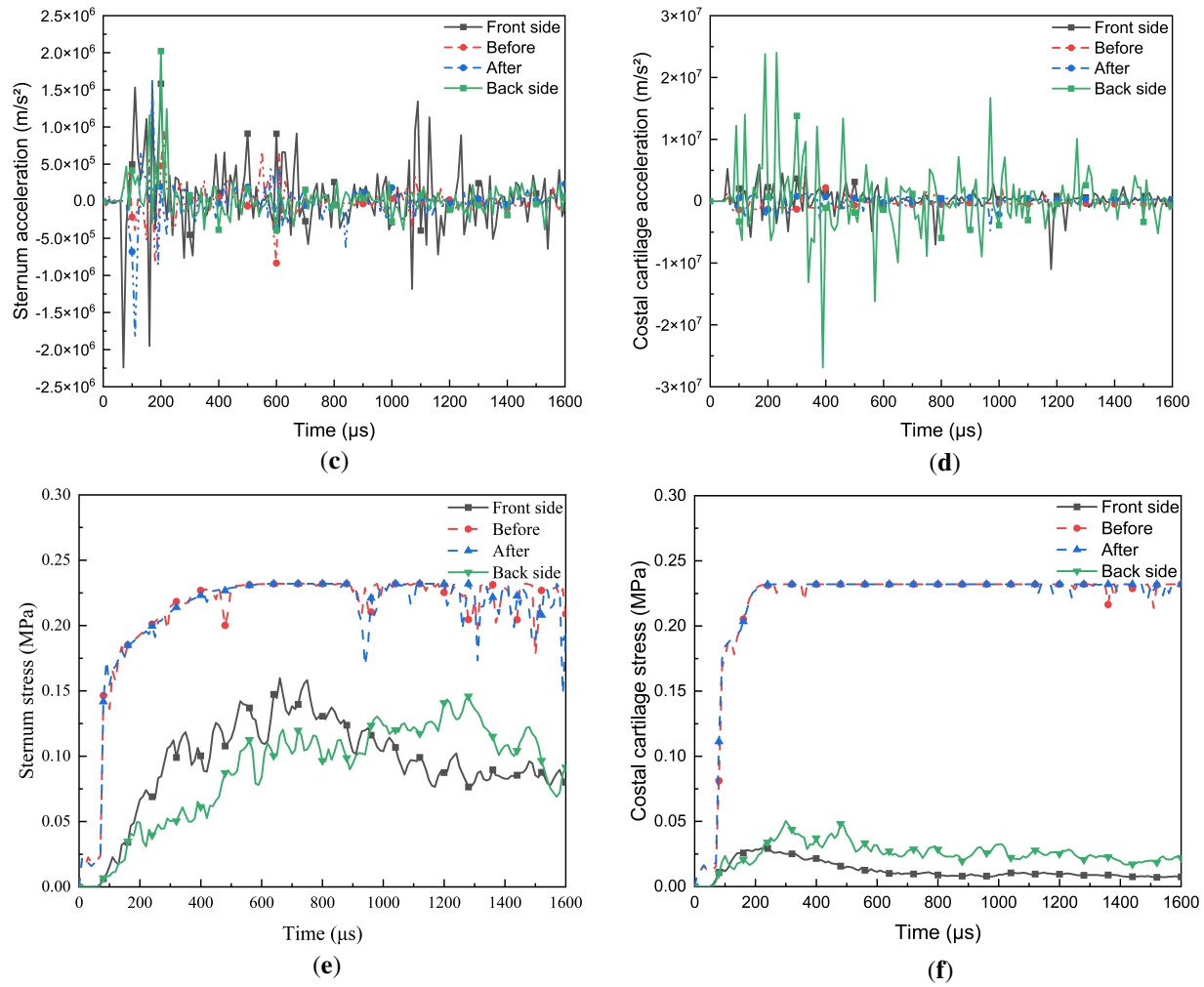


Figure 31: Pressure, acceleration, and stress time-history curves along the transmission paths from the front side to the back side of the sternum and costal cartilage: (a) sternum pressure; (b) costal cartilage pressure; (c) sternum acceleration; (d) costal cartilage acceleration; (e) sternum stress; (f) costal cartilage stress.

4 Discussion

This study further indicates that the internal thoracic response under BABT conditions cannot be explained solely by the back-face deformation of the ballistic insert or by a single external indicator. This finding extends previous studies. The internal response should instead be jointly analyzed by considering transient back-plate bulging, chest-wall compression, rib-cage load regulation, and the internal responses of the cardiopulmonary organs. Gilson et al. [15] mainly focused on the back-face deformation of ballistic inserts and its relationship with blunt injury risk. Such studies provide an important external deformation basis for BABT evaluation. However, back-face deformation itself can hardly directly reflect the propagation path of loads after entering the thoracic cavity, the pressure responses of organs, or the localized high-response distribution in tissues. In contrast, based on the validation of back-face bulging height and indentation depth of the filling material, this study further analyzed the right-lung pressure, cardiopulmonary tissue displacement, and stress responses, thereby addressing the insufficient understanding of internal thoracic responses when evaluation relies only on back-plate deformation.

Differences in the right-lung pressure response under single and multiple blunt-impact conditions reflected the variation patterns of load-transfer mechanisms under different impact scenarios. Under single impact, the right-lung pressure increased rapidly during the initial impact stage and reached the first peak at an early time, followed by a decrease and oscillatory attenuation. This may be because the initial response was mainly controlled by the transient pressure wave, whereas the subsequent variation gradually shifted to being controlled by the compression effect caused by back-plate deformation. Under multiple-impact conditions, the right-lung pressure response exhibited nonlinear variation with increasing impact number. This may be because repeated loading caused compaction and residual deformation in the local region of the biomimetic thorax, thereby changing the local buffering capacity, stiffness distribution, and contact state, and further affecting the propagation, reflection, and attenuation processes of pressure waves inside the thoracic cavity. This result is consistent with the conclusion of Koohbor et al. [14] that material damage accumulation under repeated impacts can alter the impact response. It also supplements the findings of Kote et al. [48] and Yoganandan et al. [49] regarding the site dependence and index dependence of BABT responses from the perspective of pulmonary pressure, indicating that the internal thoracic pressure response under multiple blunt impacts has obvious nonlinear characteristics.

This study performed experimental–numerical comparisons using the BFD of the composite ballistic insert and the indentation depth of the muscle-equivalent filling material. For BFD, the experimental peak time was 1100 μs , whereas the simulated peak time was 1200 μs , with a time difference of 100 μs . Using the average of the three experimental BFD curves as the reference, the root mean square error (RMSE) and NRMSE of the simulated BFD–time curve were 0.427 mm and 1.15%, respectively. For indentation depth, the experimental average was 60.08 mm, whereas the simulated value was 58.52 mm, with an absolute difference of 1.56 mm and a relative error of approximately 2.59%. These results indicate that the model can adequately describe the main processes of transient back-face deformation of the ballistic insert, compression of the filling material, and impact-load transfer toward the thorax. However, the right-lung pressure magnitude still showed an experimental–numerical discrepancy, which may be related to the arrangement of the pressure sensor. In the experiment, the sensor was attached to the lung surface and fixed by subsequent muscle-material casting. When the pressure wave propagated through the lung tissue–sensor interface and the surrounding equivalent soft-tissue medium, amplitude attenuation may have occurred. Further parameter sensitivity analysis showed that the artificial bulk viscosity coefficients (Q1) and (Q2) mainly affected the negative pressure response and local oscillations during the unloading stage, whereas the rib-cage stiffness had a more direct influence on the positive pressure peak of the right lung. Although some abnormal parameter combinations may approach the experimental results in terms of local peak values, their rib-cage material parameters and artificial bulk viscosity coefficient settings deviate from the physiological and mechanical characteristics of the real thoracic structure. Therefore, the model using normal rib-cage material parameters and default artificial bulk viscosity coefficients provides more reliable overall response trends, load-transfer behavior, and structural rationality. Compared with existing human finite element models, the use of soft-tissue materials to encapsulate the visceral organs improved the continuity of intrathoracic load transfer. Validation based on back-face bulging height and filling-material indentation depth further confirmed the model's capability to describe load transmission under back-plate compressive deformation.

Under transient loading, the displacement responses of the rib cage and the cardiopulmonary tissues during the initial stage under backplate deformation were fitted using a linear-exponential function $y(x) = a + bx + ce^{dx}$. The fitting results showed that the function captured the temporal and amplitude differences in displacement responses among different tissues. Under the same fitting function, the differences in displacement responses among tissues were mainly determined by b , c , and d . Parameter b may reflect the linear displacement evolution trend, parameter c may govern the magnitude and direction of the nonlinear

response, and parameter d may represent the establishment rate of the nonlinear response. In terms of the response pattern, the costal cartilage responded earlier and exhibited larger displacement amplitudes, whereas the internal organs showed relatively delayed responses. At the later stage, the right lung and heart displayed more pronounced growth, while the left lung changed more gradually. The impact process continued to develop as backplate deformation gradually increased, and the overall inward displacement of the rib cage became more pronounced. As the impact process continued to develop, backplate deformation gradually increased. The displacement, pressure, and stress responses of the lungs and heart were further intensified during this stage. A unified power-exponential function $\sigma(x) = A(1 - e^{-x/t_r})^p e^{-x/t_d} + C(1 - e^{-x/t_c})$ was constructed for stress responses. The fitting results showed that the morphological differences among the stress curves of different tissues were mainly determined by A , t_r , p , and t_d . Specifically, A may dominate the intensity of the primary peak response, t_r may determine the time scale of the early-stage peak formation, p may control the steepness of the pre-peak rising phase, and t_d may govern the duration of the post-peak decay process. The results further indicated that the function was able to describe the common characteristics of the stress responses of the rib cage and cardiopulmonary tissues, namely a rapid early stage rise to a peak, followed by decay and the retention of a certain residual stress.

The displacement and stress contours of the right lung in the numerical simulation showed certain localized band-like high-response characteristics. This may be because, under the impact load, the ribs underwent slight transient inward displacement and compressed the adjacent lung tissue, causing nonuniform load transfer along the rib orientation and thereby forming localized band-like high-response regions in the lung tissue. Studies by Jaffin et al. [24], Marro et al. [25], Yoganandan et al. [26], and others have shown that pulmonary injury under thoracic impact or BABT conditions may exhibit nonuniform spatial distribution characteristics. However, these studies mostly remained at the level of describing injury phenomena and discussed less the relationship between such injuries and rib load-transfer pathways. The numerical results of this study provide a possible explanation for this type of localized high-response distribution. Specifically, the rib cage may not only play an overall protective role but may also affect the spatial distribution of high-response regions in the lung through local geometry and load-transfer direction.

The maximum local compression of the muscle layer in the right-side impact region was 1.22 mm, the maximum local compression ratio was 3.62%, and the maximum local compression velocity was 17.24 m/s, corresponding to a maximum local viscous response index $VC_{\text{local,max}}$ of 0.249 m/s. This result indicates that, under the current protective conditions, although a short-duration rapid compression process occurred in the muscle layer of the impact region, the overall compression amplitude and rate-related response level were relatively low, suggesting a relatively low local potential injury risk in this region.

The local load caused by back-plate bulging did not directly act on the cardiopulmonary organs during its propagation into the thoracic cavity, but first acted on the anterior side of the rib cage. The sternum had relatively high stiffness and mainly contributed to load reflection, shielding, and rapid regulation during the early impact stage. The pressure peak in the posterior soft tissue was only 54% of that in the anterior soft tissue, indicating that the sternum could attenuate, to some extent, the impact load directly transmitted to the lungs and heart. In contrast, the costal cartilage showed greater compliance, and the pressure peak on its posterior surface was attenuated by approximately 10.8% compared with that on its impact-facing surface, indicating that it tended to participate in impact energy dispersion through local deformation and vibration response. Meanwhile, the sternum may be accompanied by local stress concentration and enhanced inertial response, whereas the costal cartilage may exhibit bending-compression coupled deformation and a relatively high local deformation response.

The above results indicate that the evaluation of thoracic blunt injury should not focus only on the maximum back-plate deformation, but should also comprehensively consider the load-transfer path

inside the thoracic cavity, the duration of load action, and the local tissue response during back-plate compression. Although excessively high local stiffness is beneficial for suppressing back-plate deformation, it may aggravate stress concentration in the chest wall or bony structures. Although an appropriately flexible design is beneficial for impact energy dispersion, it may prolong the load-action duration and increase sustained compression or vibration responses.

This study still has certain limitations. The current analysis was mainly conducted under specific projectile-type and protective-structure conditions, and the applicability of its conclusions to different projectile types, different protective structures, and different body sizes still requires further validation. Meanwhile, this study investigated the thoracic response under back-plate compressive deformation, but did not systematically analyze the specific influence of impact pressure waves on the thorax. Future work should further combine more working-condition analyses and model parameter optimization to improve the reliability of predicting local responses of thoracic organs under BAPT conditions.

5 Conclusions

This study addressed several unresolved issues, including the insufficient understanding of thoracic responses under single and multiple impacts, the limited validation of the physical rationality of numerical human thoracic models, the unclear spatial distribution and evolution of localized high-response regions in the lung under back-plate compression, and the load-buffering effect and potential injury risk of the rib cage for cardiopulmonary organs. Live-fire blunt-impact experiments on a biomimetic thoracic target were combined with numerical simulations using a gap-filled human thoracic finite element model. The study investigated the transmission patterns and impact response characteristics of the rib cage and cardiopulmonary organs under non-penetrating rifle-bullet impact protected by a SiC/UHMWPE composite ballistic insert. The load-transfer characteristics across the sternum and costal cartilage, as well as the corresponding protective mechanisms and potential injury risks, were systematically analyzed. The main conclusions are as follows:

- (1) Under single and multiple blunt impacts, the right-lung pressure response exhibited staged evolution characteristics. Under single impact, the response showed rapid fluctuations in the initial stage, followed by oscillation and gradual stabilization. Under multiple impacts, the peak pressure showed nonlinear fluctuations and did not increase linearly with the number of impacts.
- (2) After soft-tissue materials were used in this model to encapsulate the visceral organs, the continuity of load transfer was significantly enhanced. The model was validated using the back-face bulging height and the indentation depth of the filling material, thereby better highlighting the continuity and structural rationality of intrathoracic load transfer under back-plate compressive deformation.
- (3) During the initial establishment of back-plate deformation, the displacement responses of the sternum, costal cartilage, and cardiopulmonary tissues were described by a linear-exponential function $y(x) = a + bx + ce^{dx}$, with b , c , and d governing the linear trend, nonlinear response magnitude/direction, and nonlinear establishment rate, respectively. After significant back-plate compression, the stress responses of the rib cage, right lung, and heart were characterized by a power-exponential function $\sigma(x) = A(1 - e^{-x/t_r})^p e^{-x/t_d} + C(1 - e^{-x/t_c})$, where A , t_r , p , and t_d , controlled the peak intensity, early peak formation, pre-peak rising steepness, and post-peak decay duration.
- (4) The maximum local compression of the muscle layer was 1.22 mm, and $VC_{\text{local,max}}$ was 0.249 m/s, further quantifying the local compression response of soft tissue under back-plate bulging. Rib-cage protection of visceral organs was mainly achieved through the synergistic effect of rigid shielding by the sternum and flexible energy dissipation by the costal cartilage. The posterior sternum pressure peak was only 39.3% of that on the impact-facing surface, and the stress peaks in the right lung and

heart were attenuated by 65.3% and 82.2%, respectively, relative to those in the rib cage, indicating the sternum could significantly weaken the direct impact load; the costal cartilage promoted energy dispersion through bending–compression coupled deformation.

The results of this study can provide references for the back-plate stiffness design of ballistic inserts, optimization of buffer-layer structures, and evaluation of thoracic blunt injury risk. However, the localized band-like response of the lung still requires experimental validation, and the applicability of the conclusions under different projectile types, protective structures, and boundary conditions still needs to be extended. Future work will combine model optimization, multi-point sensor measurements, and more working-condition analyses to improve the reliability of predicting local responses of thoracic organs.

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Availability of Data and Materials: The data that support the findings of this study are available from the corresponding author, Yihui Zhu, upon reasonable request.

Ethics Approval: Not applicable.

Conflicts of Interest: Ang Wang, Wei Pang, and Wenchao Chen are affiliated with AVIC Jincheng Unmanned Systems Co., Ltd. The authors declare that this affiliation does not constitute a conflict of interest affecting the objectivity or integrity of this research. All other authors declare no conflicts of interest.

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