AN EXPERIMENTAL INVESTIGATION OF THE BIOKINETIC PRINCIPLES GOVERNING

NON-PENETRATING IMPACT TO THE CHEST AND THE INFLUENCE OF THE RATE OF BODY

WALL DISTORTION UPON THE SEVERITY OF LUNG INJURY

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ABSTRACT

Experimental impacts to the lateral thoracic wall of anaesthetised pigs by nonpenetrating projectiles of mass range 0,069 kg to 3,0 kg within an overall velocity range 5.8 m/s to 81.2 m/s have allowed empirical models to be derived to predict the magnitude of the peak distortion of the chest wall under loading by the free-flying projectile (Pmax) and the time taken for Pmax to be attained after initial contact of the projectile upon the thoracic wall (Time-95). Pmax can be described as a function of projectile momentum, body weight and effective contact diameter; Time-95 is dependent upon projectile mass and body weight and is independent of projectile velocity. The severity of pulmonary contusion is dependent not only upon the peak relative thoracic wall distortion, but also upon the time taken to reach the peak distortion, Time-95. The particular projectiles used in this study resulted in peak chest wall distortions being attained within the time range 1 ms to 22 ms after contact. The short duration impacts could result in serious pulmonary injuries at very small thoracic compressions, conversely, very large chest compressions produced relatively minor lung injury if the time to peak distortion was of long duration. Biophysical phenomena that may account for these rate-dependent effects are discussed.

INTRODUCTION

The contact of a non-penetrating (NP) projectile upon the torso will result in an inward distortion of the body wall; the nature and severity of the internal injury is dependent not only upon the peak distortion attained but must also be dependent upon the rate of the distortion. The motion of the body wall is the primary physical phenomenon responsible for injury and for any particular projectile impact, this motion results from the interaction of the body wall and the NP projectile. It follows therefore that the capacity for injury of a NP projectile impact is not solely dependent upon say, the kinetic energy of the projectile, it is a function of the biomechanical tolerance of the body wall and of morphometric features of the projectile such as impact diameter, deformability in addition to the obvious factors of mass and velocity. The biomechanical properties of the various sites on the torso such as lateral thorax or anterior abdomen are different, however, the ability to withstand the impact forces at a particular impact site is dependent upon the body weight or age of the animal.

The rapid distortion of the thoracic or abdominal wall may produce injuries to soft tissue that can be grouped as either DIRECT or INDIRECT

DIRECT injuries are produced generally adjacent to the point of impact by shear and distortion resulting from the displacement of the overlying body wall. Pulmonary contusions and some bowel contusions are examples of this type of injury. The propagation of stress waves or even shock waves under certain circumstances may also contribute directly to primary injury.

INDIRECT injuries are produced by the gross motion of the organ within the body cavity, motion that is tempered by the inertia of the organ and by the sites of fixation or attachment (1). Strain induced at these sites may lead to laceration, notable examples being aortic/left subclavian artery rupture following impact to the anterior chest, some splenic and bowel injuries and the tearing of the insertion of the gall bladder on the liver by differential motion of these two bodies. Indirect injuries generally occur subsequent to the direct injuries produced by body wall deformation.

The general aim of this study was to study the biomechanical principles of impact to the lateral thorax and the aetiology of DIRECT contusion injury to the lung. The pathophysiological consequences of the pulmonary injury were also studied but will not be discussed in this paper.

The severity of injury at a particular impact site such as the lateral thorax is dependent upon the magnitude of the distortion of the thoracic wall and the rate of the distortion. The influence of the rate of distortion upon the severity of pulmonary injury has been a principal topic in this study. Jonsson et al (2) studied the response of the thoracic wall of the rabbit to blast and impact exposure and were able to demonstrate the critical inter-relationship of thoracic deformation and the rate of deformation (the index of the rate of distortion was the peak velocity attained by the chest wall). The range of peak chest wall velocities studied was 2 - 20 m/s with relative chest wall deformations within the range 0.05 - 0.60. They defined a transition zone between 5 m/s and 10 m/s within which the lung injuries changed character. At peak chest wall velocities <5 m/s the lung was not significantly contused even with relative deformations up to 0.5, however, the lung could be lacerated. If the chest wall velocity exceeded 15 m/s then relative deformations of only 0.15 - 0.20 could produce severe contusions and lethal injuries.

Lau and Viano (3) studied the relative severities of bronchiolar and alveolar lung injury at fixed impact velocities of 5, 10 and 18 m/s and absolute body wall displacements between 0.2 cm and 4.5 cm in rabbits. Bronchiolar contusion was prevalent at impact velocities less than 6 m/s; alveolar injuries were predominant at velocities greater than 15 m/s. More recently, Viano and Lau (4) have proposed a 'viscous tolerance criterion' as a predictor of the risk of thoracic injury. Analysis of impact experiments on rabbits with peak chest wall velocities within the range 5 - 22 m/s and relative chest wall compressions within the range 0.04 - 0.55 led them to propose the maximum of the product of the chest wall velocity and the relative deformation as an index of injury. This criterion has been extended to abdominal injury (5).

The specific aims of our study on thoracic impacts were to:

1) Identify the kinetic characteristics of free-flying projectiles and the morphometric features of the body and chest that determine the magnitude of the distortion of the chest wall and the <u>rate</u> of the chest wall distortion.

2) Determine the sensitivity of the severity of pulmonary injury to the relative distortion and to the time to peak distortion.

3) Describe qualitative changes in the type of thoracic pathology resulting from broadly equivalent thoracic distortions occuring at different rates.

It will be demonstated later that the time to peak thoracic distortion is a function of the mass of the projectile. Projectiles of markedly different mass have been used to vary this time interval to assess the influence of chest distortion rate upon the severity of pulmonary injury.

To achieve the aims outlined above, anaesthetised pigs within the weight range 22 - 74 kg were subjected to NP impacts over the right lateral thorax by 3.7 cm diameter projectiles with a mass range of 0.069 - 3.0 kg at velocities within the overall range 5.8 - 81.2 m/s. This mass range resulted in time to peak thoracic distortions within the range 1 ms to 22 ms. The severity of the pulmonary pathology was assessed 3 hours after impact.

METHODS

A list of abbreviations is presented in TABLE 1 and the range of biokinetic conditions used in the experiments is summarised in TABLE 2.

TABLE 1: List of abbreviations.

Abbreviation Units Description

| М | kg | mass of the projectile | | | | |
|------------|-----|--|--|--|--|--|
| D | cm | impact diameter of the projectile | | | | |
| V | m/s | pre-impact velocity of the projectile | | | | |
| Pmax | cm | maximum transient inward deformation of the body wall | | | | |
| Time-95 | ms | time taken for the projectile to attain a 95% reduction in its pre-impact velocity after contact | | | | |
| W | kg | body weight of the pig | | | | |
| LAT and AP | cm | lateral and anteroposterior torso depth respectively at the impact site | | | | |
| Qi | | quotient of lung injury (actual lung weight/predicted normal lung weight) | | | | |

TABLE 2: RANGE of projectile kinetic characteristics, pig weight and response of the lateral thoracic wall to the non-penetrating impact loading

All projectiles had a diameter of 3.7 cm. 'n' is the number of experiments.

| PROJECTILE | | PIG WEIGHT | LATERAL THORACIC WALL DISTORTION | | | |
|------------|-----------|---------------|----------------------------------|-------------|--------------|----|
| MASS | VELOCITY | | ABSOLUTE | RELATIVE | TIME TO PEAK | |
| M, kg | V, m/s | W, kg | Pmax, cm | Pmax/LAT | Time-95, ms | n |
| 3.0 | 5.8-12.0 | 37-59 | 4.7-12.2 | 0.223-0.590 | 15.7-22.0 | 5 |
| 1.0 | 13.8-24.1 | 37-54 | 6.2-10.8 | 0.295-0.537 | 9.0-11.1 | 6 |
| 0.38 | 15.6-38.6 | 32-53 | 4.2-7.4 | 0.200-0.400 | 3.8-6.8 | 5 |
| 0.14 | 23.7-65.3 | 22-74 | 3.2-6.0 | 0.168-0.352 | 2.0-4.0 | 19 |
| 0.12 | 43.0-69.7 | 37-54 | 3.8-6.1 | 0.188-0.293 | 1.8-2.9 | 5 |
| 0.069 | 43.6-81.2 | 28-52 | 2.5-4.6 | 0.119-0.237 | 1.0-1.6 | 7 |

Animal preparation

Sedated pigs were anaesthetised through an ear vein with sodium pentobarbitone and were then tracheotomised. A catheter was inserted into the femoral vein for subsequent administration of anaesthetic. In some animals, high frequency response tourmaline pressure transducers were inserted by peripheral cutdown into the thoracic aorta and vena cava.

The impact site was chosen to overlie the diaphragmatic lobe of the right lung; this point was defined externally as mid-right flank on the same transverse section as the xiphisternum. Animals were suspended horizontally in a specially constructed stainless steel frame over a compressed-air driven projectile launcher. The left lateral torso was restrained to obviate motion of the animal during the higher momentum impacts. The partial pressure of oxygen in arterial blood of the anaesthetised pig was monitored for 3 hours (results not presented) and the pig was then sacrificed by exsanguination whilst still under the influence of anaesthetic.

The pig was subjected to a detailed post-mortem examination. The severity of lung injury was assessed quantitatively as an increase in lung weight (and by measurement of the contusion volume). The actual weight of the lungs from the injured animal was compared to the predicted normal lung weight for a pig of that body weight (predicted from the relationship between body weight and lung weight of seventeen CONTROL pigs within the body weight range 23-79 kg). The ratio (injured lung weight/predicted normal lung weight) was described as the "Quotient of lung injury", Qi. Each set of lungs was awarded an injury severity score ranging from 1 to 4 based upon its Qi; TABLE 3 defines the Qi limits.

TABLE 3: Qi limits of the four severity grades used in the quantitative assessment of the severity of pulmonary contusion.

| QUOTIENT OF INJURY, Qi | GRADE | DE SCRIPTION | SYMBOL in Figure 5 | |
|------------------------|-------|-----------------|-----------------------|--|
| <1.2 | 1 | Minor/Uninjured | | |
| >1.2 and <1.5 | 2 | Moderate | Δ | |
| >1.5 and <1.9 | 3 | Severe | | |
| >1.9 | 4 | Very Severe | • | |

Biokinetic techniques

Impacts were photographed by high-speed cine cameras at a framing rate between 1000 and 5000 pps depending upon the nominal velocity of the free-flying projectile. Computer-aided analysis of high speed film allowed measurement of

- : impact velocity (V)
- : the maximum transient deformation of the chest wall under the contact area of the projectile (Pmax). This was achieved by digitizing the rear of the projectile upon contact with the chest and assumes that the projectile does not deform upon contact and that the chest wall does not overrun when the projectile comes to rest. Separate high-speed cine-radiographic experiments studying pulmonary distortion failed to demonstrate overrun of the thoracic wall.

: the time taken for the projectile to attain a 95% reduction in its pre-impact velocity after contact with the chest (Time-95).

The six projectiles had a diameter of 3.7 cm and were of mass (M) 0.069, 0.12, 0.14, 0.38, 1.0 and 3.0 kg; (reference is also made briefly to data acquired using a 0.14 kg projectile with an effective contact diameter of 10.0 cm.) The range of impact velocities and the body weight of the pigs is shown in TABLE 2.

RESULTS

Factors governing the time to peak thoracic deformation

The motion of the lateral thoracic wall under impact loading is shown in FIGURE 1 for a 0.14 kg projectile. Pmax was 5.3 cm under these impact conditions (V = 58.8 m/s, W = 41 kg, D = 3.7 cm) and the time to peak distortion (Time-95) was 2.1 ms. The range of Pmax and Time-95 measured with the various projectiles is summarised in TABLE 2 and it will be shown later that Time-95 had a critical influence on the severity of lung injury. Time-95 was found to be dependent upon the mass of the projectile and inversely dependent upon the body weight of the pig. Time-95 was independent of initial impact velocity. The dependence of Time-95 upon M has a theoretical basis if the thorax is considered to be a simple linear spring/mass system; the contact of the projectile increases the effective mass of the body wall changing the natural frequency of the mass-spring-mass system crudely representing the thoracic wall, lung and mediastinum respectively. The body weight of the pig is taken in this context as an index of the biomechanical properties of the body wall and the inverse relationship between Time-95 and this index of body wall stiffness is not surprising.

FIGURE 2 shows Time-95 as a function os (projectile mass/pig weight) on LOGe/LOGe axes. The data may be fitted linearly (r = 0.98) and the equation of this line may be transformed to the power function

Abbreviations and units are shown in TABLE 1. Distortions taking place over short periods of time with low mass projectiles produce much greater severity of injury than greater distortions taking place over long periods of time with the high mass projectiles. The kinetic factors that govern the magnitude of the thoracic wall distortion will be discussed in the next section.

Factors relating the projectile kinetics and the peak thoracic deformation The absolute thoracic deformation (Pmax) and the relative deformation (Pmax/LAT) must be a function of the mass and velocity of the projectile. As the resultant distortion is the result of the interaction between the projectile and the body wall, the biomechanical properties of the thoracic wall must also influence the degree of distortion. Pmax will be inversely dependent upon body weight (in animals of the same strain and fat content) and at equivalent impact severities, an animal of low body mass will undergo a greater thoracic distortion under the impact site than an animal of higher body weight.

Pmax (and Pmax/LAT) was found to be a function of the <u>momentum</u> of the projectile, not its kinetic energy. The maximum chest compression (Pmax) is shown as a function of (momentum/lateral chest depth) in FIGURE 3 on LOGe/LOGe axes. The data is fitted by a straight line (r=0.96) and the equation of the line may be transformed to the relationship LAT in this context is an index of the biomechanical tolerance of the pig's thorax and implicity indicates the effective chest mass and 'stiffness' of the thoracic wall. Equation 2 was constructed with 3.7 cm projectiles of mass range 0.069 kg to 3.0 kg, velocity range 5.8 m/s to 81.2 m/s and pig weight range 22 kg to 74 kg (TABLE 2).

Previous studies investigating the biomechanical principles of NP projectile impacts to the anterior chest (6) had used an additional projectile of mass 0.14 kg with a rectangular contact area of 3.7 cm x 10.0 cm. Increasing D to 10.0 cm resulted in a lower Pmax compared to the distortion produced by 3.7 cm projectiles at broadly equivalent impact severities. This is not surprising as the impact force was applied over a larger surface area. Additional experiments were performed in which the 0.14 kg projectile with an effective contact diameter of 10.0 cm was propelled against the right lateral thorax; the 10.0 cm dimension was aligned in a cranial/caudal orientation traversing ribs. Although the details of these experiments will not be reported now, this data is included in FIGURE 4 defining the relative chest wall distortion (Pmax/LAT) as a function of ((M*V)/(W*D)) on LOGe/LOGe axes. Dividing the projectile momentum by the contact diameter allows the 3.7 cm and 10.0 cm data to be described by a common straight line of correlation coefficient 0.96. This relationship transforms to the expression

$$Pmax/LAT = 1.29 * (((M*V)/(W*D))^{0.529})$$
3

The empirical models outlined above (and others not described) constructed from experiments whose biokinetic details provided a large range of projectile mass, velocity and pig weight permit quantitative predictions of the absolute response of the lateral chest wall under a wide variety of NP projectile impact conditions. The results of this motion of the thoracic wall in terms of the pulmonary injury produced by compression, shear and the propagation of stress phenonemena will be considered in the next section.

The response of the lung to the distortion of the body wall

The common acute response of the lung to NP mechanical injury is to accumulate blood (a pulmonary contusion) usually in the absence of gross parenchymal laceration. The presence of blood within alveoli may produce a physiological shunt resulting in a reduction of the partial pressure of oxygen in arterial blood (PaO2). Haemodynamic and permeability changes within lung may result in a self reinforcing cascade of fluid accumulation, further reduction of PaO2, infection and the ultimate demise of the victim. Small contusions may be well tolerated; the lung is able to regulate (reduce) the blood supply to small haemorrhagic areas and maintain a normal PaO2.

The pulmonary contusions produced in this series of experiments ranged from a Qi of approximately 1 (uninjured lungs) to a Qi of 2.73 showing haemorrhagic contamination of the whole right lung and a significant proportion of the left lung. Qualitatively the pattern of injury seen in these experiments, irrespective of the time to peak distortion, may be summarised thus:

- : ribs were fractured in nearly all impacts
- : contusions were confined to the right lung. Recent experiments using high-speed cine-radiography of lateral thoracic impacts showed only minor

distortions of the mediastinum and transmission of significant strain to the left lung was not evident (paper in preparation). Contamination of the left lung in the current experiments was only seen in those animals with very severe haemorrhagic contaminations of the right lung; the blood in the left lung was considered to result from reflux of blood from the injured right lung.

: laceration of the parenchyma was seen only following impacts of the 1.0 kg and 3.0 kg projectiles resulting in large thoracic distortions over extended time periods (10 - 20 ms). The lacerations were quite small and resulted from trapping of the parenchyma between displaced ribs.

Both Time-95 and Pmax/LAT had a critical influence upon the severity of the pulmonary contusions. At the extremes, high mass/high momentum projectiles produced great distortions over long periods of time resulting in relatively minor lung injury whereas small mass projectiles resulting in quite small distortions over very short periods of time produced serious injuries. As an example of the former:-

a pig impacted by a 3.0kg projectile at 9.6 m/s suffered a relative chest distortion of 0.505 occuring over 22.0 ms. In spite of the fact that locally the chest was compressed to half its normal diameter, the pulmonary injuries were not severe - a few scattered patechiae on the right diaphragmatic lobe (impact point) and two small lacerations with associated confirmed contusion where fractured/displaced ribs had injured the parenchyma. There was very little haemorrhagic contamination of the lung (Qi = 1.05).

however,

a pig impacted by a 0.069 kg projectile at 53.5 m/s suffered a relative chest distortion of only 0.184 and the peak distortion was reached in only 1.6 ms. The right lung was grossly contused with almost total involvement of the right diaphragmatic lobe and extensive contusions were evident on the posterolateral surfaces of the middle and apical lobes. This was a notable and severe haemorrhagic contamination of the lung (Qi = 1.79), equivalent to 260 g of blood in a lung of normal predicted weight 330 g.

These two examples are not the extremes of injury but are presented simply to illustrate a point. Notwithstanding the severity of pulmonary haemorrhagic contamination, the only qualitative difference seen with high-rate and low-rate distortions was a larger number of fractured ribs in each pig subjected to the very large distortions, and the parenchymal trapping described above.

FIGURE 5 presents the relationship between Pmax/LAT, Time-95 and Qi for all the 3.7 cm diameter impact data. The range of Pmax/LAT and Time-95 produced by each projectile is shown in TABLE 2. The ordinate in FIGURE 5 is in fact the reciprocal of the time to 95% reduction in projectile velocity, 1/Time-95 and the abcissa is the peak relative body wall distortion. The severity of lung injury, Qi is summarised for each animal by assigning a symbol to represent the injury grade deduced from Qi (TABLE 3).

The data can be seen to lie broadly in three bands, Grade 1 injuries, Grade 2 and 3 combined and Grade 4 injuries. It can be seen from this Figure that

those animals suffering relative chest distortions over time intervals greater than approximately 10 ms had minor pulmonary contusions in spite of the very severe chest compressions produced (up to 0.6). Shortening the time to peak distortion resulted in a greater incidence of serious and very serious injuries although very serious Grade 4 contusions were only evident if the value of Time-95 was less than approximately 4 ms (the greatest value of Time-95 showing Grade 4 injuries was 3.8 ms). In the most extreme case of Grade 4 injuries produced by very short duration impacts, a Qi=1.94 was produced by a Pmax/LAT of only 0.157 (Pmax = 3.3cm) occurring within 1 ms.

Physical phenomena responsible for the rate dependence of lung injury Consideration of the mechanistic reasons for the dependence of the severity of lung injury upon Time-95 could implicate the following phenomena:

- : shear strain of the underlying lung parenchyma with the propensity to tear small blood vessels dependent upon the magnitude and rate of distortion of the tissue. Quite large distortions of parenchyma would be expected with this low-frequency phenomenon.
- : stress waves resulting from the distortion of the chest wall with possible stress concentration at interfaces of different density and at regions within the thorax that may result in multiple reflections (for example the medial border of the lung). These waves can be considered to be compression waves of high-frequency.
- : shock waves generated by the acceleration of parenchyma to velocities in excess of the velocity of sound in the tissue. These waves would initially possess effectively instantaneous pressure wavefronts.

It is well known from studies of the behaviour of inanimate materials that solid material will break when strained too greatly or too quickly. Although this is a convenient explanation for the rate dependent effects of lung injury particularly at the low impact velocity and high Pmax conditions where shear strain may be a notable component, the extent of lung injury found with the high velocity impacts (low Time-95) and the minor physical distortions of the body wall imply that other physical phenomena may be involved. The chest wall under impact loading acts as a piston and projects a pressure pulse into the thoracic contents, the pressure being an index of the average stress field. We (and others (2)) have measured the pressures within the chest and bronchi under impact loading and have found it to be a fairly smoothed low-frequency stress wave. Gauges in bronchi measure only the pressure in the bronchi not in the true parenchyma; compression waves in the tissue itself may be quite intense resulting in high local forces at the capillary level associated with quite small distortions of tissue. These forces and small distortions (compared to quite gross distortions associated with shear waves) may be implicated in the laceration of small capillaries.

The velocity of sound within the lung is around 15 - 30 m/s (2). Acceleration of tissue particles by an advancing chest wall at velocities greater than this could give rise to true shock phenomena close at the impact site with propagation of the shock wave into the parenchyma. Most of the initial chest wall velocities produced by the low mass projectiles used in this study exceeded this velocity of wave propagation in the lung. Although the shock is likely to be damped by the lung fairly quickly, significant pressure fluctuations may be transmitted widely in the parenchyma with dissipation of energy at boundaries and at regions of stress concentration such as pleural niches. Implosion of alveoli and spalling at the alveolar level have been implicated but the absence of high frequency components in the pressure waves measured in bronchi have implied to others that the spalling hypotheses are unlikely (2) and are simply speculative.

Detection of the actual sites of capillary disruption in lungs (impact site, contralateral border of the lung, interlobular septae, pleural borders and other sites predisposing to stress concentration and energy transfer) would allow informed speculation of the relative contribution of the phenomena outlined above to the pulmonary injury witnessed at the various energy transfer rates. Identification of microvascular laceration by histological sampling of an organ such as the lung is extremely difficult. The gross appearance of a pulmonary contusion defines simply the presence of blood not necessarily the sites of mechanical injury to capillaries or larger vessels. Blood has a remarkable ability to reflux throughout the lung and we believe that a significant proportion of the volume of a pulmonary contusion is simply blood from a site of mechanical injury contaminating normal parenchyma. Quantitative histological experiments are underway to attempt to locate these sites.

In summary, it is obvious that impact injury to the thorax may excite a combination of wave modes whose relative significance will be dependent upon the rate of distortion of the body wall; the different wave phenomena may result in differing types and severities of injury to lung. A theoretical study of the possible phenomena is underway using the present empirical data as a foundation.

CONCLUSIONS

1) The body wall subjected to a loading by NP impact with a free flying projectile will distort asymptotically with respect to time to a maximum body wall distortion. The distortion is transient.

2) The time taken to achieve maximum body wall distortion is governed by the mass of the projectile and inversely dependent upon the body mass of the pig.

3) The time to peak distortion is independent of projectile velocity.

4) The maximum thoracic distortion (either absolute or relative) is dependent upon the momentum of the projectile.

5) Impacts at the same site upon pigs of different biomechanical tolerances resulting from varying effective mass of the chest wall and rib strength can be expressed simply in terms of the mass of the animal or chest dimensions. With the pig model, dividing by the mass of the animal can be used to normalise impact severities to compare impacts upon targets or different weight.

6) The severity of pulmonary contusion is dependent not only upon the peak relative thoracic deformation but also upon the time taken to achieve the peak distortion.

7) Serious pulmonary injuries may be produced by quite small thoracic wall distortions if the distortion occurs over a short period of time (1 - 2 ms). Impacts producing severe chest wall distortions of extended duration (10 - 20 ms) produce less severe pulmonary injuries.

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FIGURE 1: The displacement of the chest wall with respect to time for a 0.14 κ g projectile. The first differential of the curve fitted to this data is also shown.







FIGURE 3: The maximum absolute chest wall distortion, Pmax as a function of ((M*V)/LAT) plotted on LOGe/LOGe axes for the impact of 3.7 cm projectiles.







FIGURE 5: The severity of lung injury related to Pmax/LAT and the reciprocal of the time to peak distortion (1/Time-95). The severity of lung injury is expressed as a symbol: minor injury (\triangle), moderate (\triangle), severe (\square) and very severe (\square). The Qi limits for these grades are shown in TABLE 3. This Figure only plots data acquired with 3.7 cm diameter projectiles.