Correlating the extent and location of pulmonary contusion to vehicle crash parameters

K.A. Danelson[1,2], A.A. Weaver[1,2], F.S. Gayzik[1,2], J.D. Stitzel[1,2]
1. Wake Forest University School of Medicine
2. VT-WFU School of Biomedical Engineering and Sciences

ABSTRACT

Pulmonary contusion is a common injury following motor vehicle crash (MVC). The estimated mortality rates for this injury range from 10% to 20%. Another important clinical implication of this lesion is the potential for development of Acute Respiratory Distress Syndrome (ARDS). Studies have correlated an increased risk of ARDS with a pulmonary contusion extent exceeding 20% of the total lung volume. The mechanism of injury for pulmonary contusion is a mechanical insult that leads to an inflammatory response. To better predict possible injury to an occupant following a MVC, this study correlates the resulting percent injury to the lung (inflammatory response) to crash parameters (mechanical insult).

The Wake Forest University CIREN database was queried to extract all occupants sustaining pulmonary contusion in a near-side crash. CIREN was selected because of the extensive medical history of the occupant, imaging studies of the injury, and in-depth crash investigations. Specific crash characteristics selected were the vehicle type, object struck, change in velocity due to the impact, principal direction of force of the crash, and vehicle components contacted by the occupant. Additionally, the extent of the crush was calculated as the area of the crush. The radiological studies were segmented to determine the percent volume of high radiopacity lung as compared to the total volume of the lung. The mean impact severity for the cases with pulmonary contusion below 20% was 36.14 kph, with a range of 13 to 68 kph. In comparison, the mean severity for cases above this value was 40.9 kph, with a range of 25 to 56 kph. Using the same grouping of above or below 20% volume, maximum crush was a significant predictor of the pulmonary contusion higher volume group with a p-value of 0.0427. Additionally, the effect of side airbags was considered by examining NASS. In near-side cases without airbag deployment, in crashes with delta-V's close to NCAP (43 to 63 kph), the incidence of pulmonary contusion was

2.52%. In comparison, the near-side cases with airbag deployment had a pulmonary contusion rate of 2.22%, a 14% decrease. This difference is small; however, pulmonary contusion rates with side impact airbags should be studied further.

These types of correlations can be used in the future to more accurately predict potential patient outcomes given basic crash characteristics. Since pulmonary contusion is based on an inflammatory response, the ability to predict possible outcomes would allow physicians to develop an appropriate treatment plan that focuses on the prevention of more severe outcomes, such as ARDS, through early interventions in high risk patients.

INTRODUCTION

Motor Vehicle Crashes (MVCs) are the leading cause of blunt chest trauma (Shorr et al. 1987) with pulmonary contusion (PC) as the most common injury following this type of insult (Clark et al. 1988; Allen et al. 1996). The associated mortality rate for this injury ranges from 10% to 20% of individuals (Hoff et al. 1994). Additionally, even non-fatal occurrences of this injury can result in long term diminished respiratory function (Kishikawa et al. 1991). Miller et al. correlated the percent injured lung to the possibility of developing Acute Respiratory Distress Syndrome (ARDS). In this study, patients with isolated pulmonary contusion were evaluated, and the percent pulmonary contusion was calculated from computed tomography (CT) scans. The results indicated that if 20% of the lung was injured, the incidence of ARDS sharply increased. Seventy-eight percent of the patients with PC volume above 20% developed ARDS (Miller et al. 2001). The significance of these findings is that the volumetric measurement of pulmonary contusion can predict possible clinical outcomes to appropriately plan treatment for these patients.

Pulmonary contusion is not a well understood injury. In MVC impact scenarios, the initial injury mechanism is a mechanical insult to the chest. This results in stress waves that injure the alveoli in the lungs (Fung et al. 1988). There are three postulated mechanisms for injury to these structures: a spalling effect at the air/tissue interface, an inertial effect, and an implosion effect (Cohn 1997). Spalling occurs due to a shearing or bursting at the air/tissue interface. The inertial effect is when the low density lung material is stripped from attached vessels through relative motion within the lung. The implosion effect is the overexpansion of the gas in the alveoli as the stress wave passes. While the injury is initiated by a mechanical insult, the inflammatory response of the lungs also determines the extent of the injury (Windsor et al. 1993; Marshall 2001). Due to this secondary response, the ultimate size of the lesion is not maximal until 24 to 48 hours after the impact (Cohn 1997). Understanding the effect of the initial response as well as the following inflammatory response is critical in the identification of risk factors that would predispose an individual to this injury. The research for this study will address the initial mechanical insult for this injury.

There have been previous studies correlating crash parameters to the possibility of pulmonary contusion. These studies have demonstrated that higher severity crashes and crashes involving near-side impact, defined as an impact to the side closest to the occupant, were more likely to result in pulmonary contusion (O'Connor et al. 2009). The Crash Injury Research and Engineering Network (CIREN) database was studied by Gayzik et al. to elucidate potential factors that would predispose a patient to pulmonary contusion. This study found that while the number of side impact cases that resulted in pulmonary contusion was similar to the number of frontal cases that resulted in pulmonary contusion, the total number of frontal crashes, with or without pulmonary contusion, was much higher. Therefore, the number of side impact crashes resulting in pulmonary contusion was disproportionately high. Also, this study found that the injured lung was correlated to the side of the vehicle impacted (Gayzik et al. 2009). Because this lesion has a delayed onset, knowing the crash factors that can predispose a patient to pulmonary contusion could allow physicians to develop methods to treat the disease before it is fully developed.

METHODS

The initial dataset examined was only Wake Forest University CIREN occupants. The selection parameters for study inclusion were the presence of pulmonary contusion, a left or right side impact, and the impact on the same side as the occupant (a near-side impact). After narrowing the number of subjects based

on the selection parameters there were 9 individuals in the dataset. Using the radiologic studies from CIREN case occupants, the percent volume of high radiopacity lung was quantified in Mimics (Materialise, Leuven, Belgium) using the method developed by the Virginia Tech / Wake Forest University Center for Injury Biomechanics and described by Weaver et al. (Weaver et al. 2009) . This semi-automated method of segmentation relies on the difference in Hounsfield unit (HU) values between the air in the lungs and opaque injured tissue. Uninjured tissue was clearly delineated with HU values between -1024 and -562 HU. Radiopaque tissue had a HU value greater than -562 and could be a variety of lung pathology including atelectasis or pulmonary contusion. This difference in HU values clearly delineated lung tissue from other thoracic tissue and high radiopaque tissue from healthy tissue.
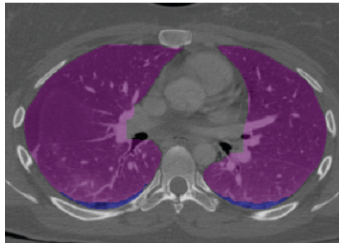

Figure 1: Total lung mask before (purple) and after (blue sections added) manual editing
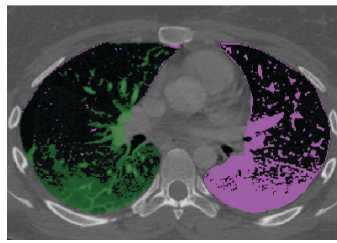

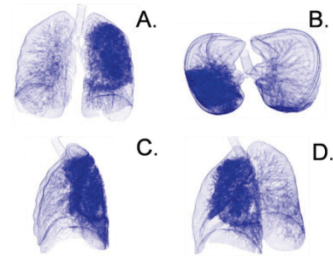**Figure 2: Right (green) and left (purple) radiopaque lung masks**

Figure 5: 3D reconstruction of uninjured and radiopaque lung (dark areas).

Views: A. Anterior/Posterior, B. Superior/Inferior, C. Lateral/ Medial, D. Oblique

The first step in this process was the general selection of all lung tissue using the automatic thresholding command included in the software. An exemplar image of the total lung mask is shown in purple in Figure 1. From this mask, areas of pnuemothorax were subtracted from the total lung mask. This mask was then manually adjusted to remove areas that are not lung parenchyma, such as bronchioles and blood vessels. Finally areas that were high radiopaque tissue within the lung were manually added into the mask. Next, the areas of radiopaque lung were identified in a similar fashion, through automatic segmentation followed by manual editing. The radiopaque lung mask for the right and left lungs (prior to manual editing) are shown in the green and purple masks, respectively, in Figure 2. The final result was three masks (total lung, healthy lung, and radiopaque lung) on each of the two-dimensional slices of the CT scan. The percent volume of high radiopacity was calculated based on the ratio of high radiopacity lung to total lung. These volumes can be three-dimensionally displayed, as shown in Figure 3. In this figure, the dark blue colored portion is radiopaque lung, and the more transparent light blue is healthy lung.

The second phase of the study was to correlate the injury percentages and lesion locations, based on the segmentation study, to the subject and crash data included in the CIREN database. The specific crash data of interest are the change in velocity (delta-V), principal direction of force, Collision Deformation Code (CDC), extent of crush, occupant location relative to crush, components contacted, restraint status, airbags present, vehicle type and occupant parameters. Occupant parameters include height, weight, and age. The delta-V is a measure of the change in the velocity of the vehicle and is a commonly selected metric to evaluate the severity of the crash. The CDC is a seven digit alpha-numeric code that describes the area and extent of damage to the vehicle. The extent of crush and crush location relative to the occupant is a measure of the amount of intrusion and its location relative to the injured organ. It was postulated that delta-V, principal direction of force, crush extent, occupant location relative to crush, CDC, components contacts, or a combination of a number of them, will be the best predictors of pulmonary contusion. Restraint status, airbags present, vehicle type and occupant parameters are included in the analysis since they are postulated to be covariates in the relationship of crash severity and the extent of pulmonary contusion. For example, given the same crash configuration and severity, an occupant without a side airbag would be more likely to sustain more severe injuries than the airbag protected occupant.

RESULTS

The mean impact severity for the cases with pulmonary contusion volume below 20% was 36.14 kph, with a range of 13 to 68 kph. In comparison, the mean severity for cases above this value was 40.9 kph, with a range of 25 to 56 kph. When these results are plotted, there is general trend of increasing volume of pulmonary contusion with increasing delta-V, as shown in Figure 4. Using the same grouping of above or below 20% volume, maximum crush was a significant predictor of the pulmonary contusion volume group with a p-value of 0.0427. This parameter was the only significant predictor of pulmonary contusion for this subset of CIREN data.
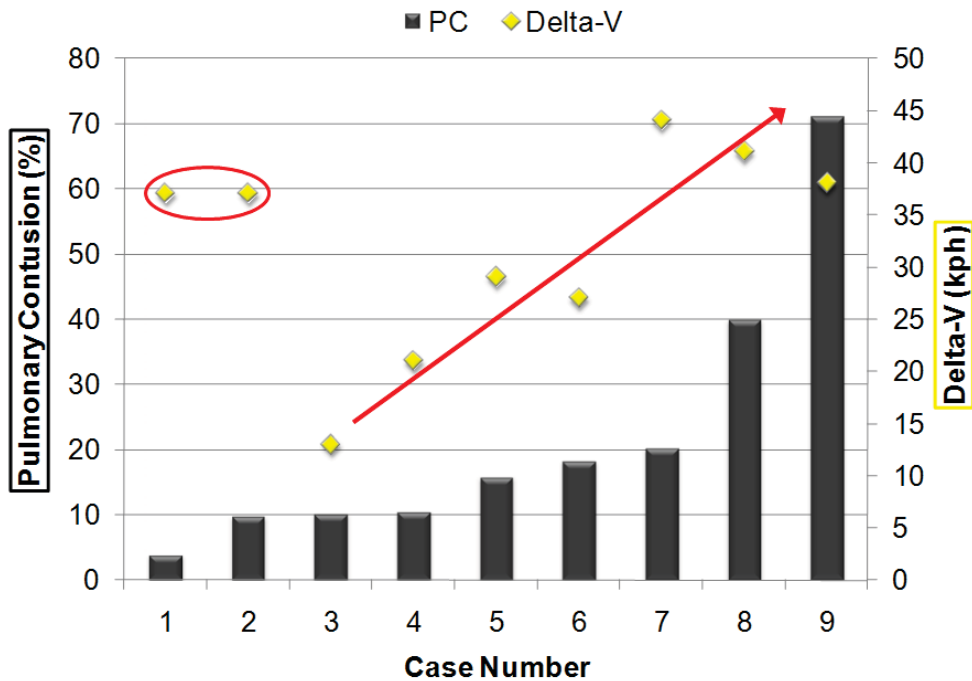

Figure 4: Plot of delta-V and percent pulmonary contusion for the WFU CIREN case occupants. Two possible outliers are circled in red. Case number 1 and 2 were young occupants with a high volume of high radiopacity lung and low change in velocity.
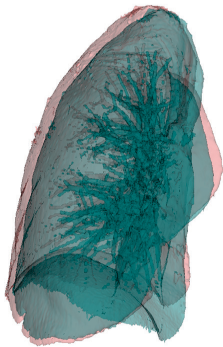
DISCUSSION

Figure 5: Registration of the trauma scan lung to the lung atlas.

Future work will determine the location of pulmonary contusion in these patients using a lung atlas to co-register trauma scans against. A grossly normal CT scan of an average male, taken to evaluate possible pulmonary embolism, has been selected from the hospital database. The patient selected did not have any blunt chest trauma, pulmonary embolism, or pulmonary pathology. With the assistance of a trained radiologist that specializes in chest CT, the lobes were segmented. To put the segmented trauma scans in the same coordinate system as the atlas, rigid and affine registration transformations will be used to morph the trauma scan to the atlas. An initial registration of the lung, by side, was successful as shown in Figure 5. This figure shows the result of the right lung trauma scan (teal) registered to the lung atlas (pink). For this registration, the trauma scan was morphed to the atlas space. This placed both lungs in the same coordinate system. In future work, this morphing will allow for a better comparison of lesion location between different individuals in the study.

The effect of side airbags was considered in these cases by examining NASS to determine if they are a confounding variable for the analysis. In near-side cases without airbag deployment and with a delta-V close to NCAP testing speeds (43 to 63 kph), the incidence of pulmonary contusion was 2.52%. In comparison, the near-side cases with airbag deployment had a pulmonary contusion rate of 2.22%, a 14% decrease. This difference is small; however, pulmonary contusion rates with side impact airbags should be studied further. For the study with the full dataset, these results indicate that the presence of a deployed airbag will have to be considered during the analysis.

The initial dataset used was small and future work will expand the analysis to include all CIREN occupants with radiology uploaded to the database. Initial selection of these individuals has identified 109 near-side occupants with pulmonary contusion from the January 2005 (the first year with required scan entry into the database) to July 2010.

CONCLUSIONS

The correlation between crash parameters and the volume of high radiopacity lung can be used in the future to more accurately predict potential patient outcomes given basic crash characteristics. The Wake Forest University CIREN center cases with pulmonary contusion were analyzed and the results suggested a possible trend of increased volume of high radiopacity lung with an increase in the change in velocity of the MVC. Since pulmonary contusion is based on an inflammatory response, the ability to predict possible outcomes would allow physicians to develop an appropriate treatment plan that focuses on the prevention of more severe outcomes, such as ARDS, through early interventions in high risk patients.

ACKNOWLEDGEMENTS

REFERENCES

ALLEN, G. S. and COATES, N. E. (1996). Pulmonary contusion: a collective review. Am Surg 62(11): 895-900.

CLARK, G. C., SCHECTER, W. P. and TRUNKEY, D. D. (1988). Variables affecting outcome in blunt chest trauma: flail chest vs. pulmonary contusion. J Trauma 28(3): 298-304.

COHN, S. M. (1997). Pulmonary contusion: review of the clinical entity. J Trauma 42(5): 973-979.

FUNG, Y. C., YEN, R. T., TAO, Z. L. and LIU, S. Q. (1988). A hypothesis on the mechanism of trauma of lung tissue subjected to impact load. J Biomech Eng 110(1): 50-56.

GAYZIK, F. S., MARTIN, R. S., GABLER, H. C., HOTH, J. J., DUMA, S. M., MEREDITH, J. W. and STITZEL, J. D. (2009). Characterization of crash-induced thoracic loading resulting in pulmonary contusion. J Trauma 66(3): 840-849.

HOFF, S. J., SHOTTS, S. D., EDDY, V. A. and MORRIS, J. A., JR. (1994). Outcome of isolated pulmonary contusion in blunt trauma patients. Am Surg 60(2): 138-142.

KISHIKAWA, M., YOSHIOKA, T., SHIMAZU, T., SUGIMOTO, H., YOSHIOKA, T. and SUGIMOTO, T. (1991). Pulmonary contusion causes long-term respiratory dysfunction with decreased functional residual capacity. J Trauma 31(9): 1203-1208; discussion 1208-1210.

MARSHALL, J. C. (2001). Inflammation, coagulopathy, and the pathogenesis of multiple organ dysfunction syndrome. Crit Care Med 29(7 Suppl): S99-106.

MILLER, P. R., CROCE, M. A., BEE, T. K., QAISI, W. G., SMITH, C. P., COLLINS, G. L. and FABIAN, T. C. (2001). ARDS after pulmonary contusion: accurate measurement of contusion volume identifies high-risk patients. J Trauma 51(2): 223-228; discussion 229-230.

O'CONNOR, J. V., KUFERA, J. A., KERNS, T. J., STEIN, D. M., HO, S., DISCHINGER, P. C. and SCALEA, T. M. (2009). Crash and occupant predictors of pulmonary contusion. J Trauma 66(4): 1091-1095.

SHORR, R. M., CRITTENDEN, M., INDECK, M., HARTUNIAN, S. L. and RODRIGUEZ, A. (1987). Blunt thoracic trauma. Analysis of 515 patients. Ann Surg 206(2): 200-205.

WEAVER, A. A., GAYZIK, F. S. and STITZEL, J. D. (2009). Biomechanical analysis of pulmonary contusion in motor vehicle crash victims: a crash injury research and engineering network (ciren) study - biomed 2009. Biomed Sci Instrum 45: 364-369.

WINDSOR, A. C., MULLEN, P. G. and FOWLER, A. A. (1993). Acute lung injury: what have we learned from animal models? Am J Med Sci 306(2): 111-116.