An assessment of the factors associated with fatal outcomes for motor vehicle occupants in South Australia (1981-2020)

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Abstract

Vehicle occupants account for a large majority of the fatalities resulting from motor vehicle crashes. Despite the overall downward trend in occupant fatalities in Australia in recent decades, the number of occupant fatalities has begun to plateau, suggesting that there is a subset of crashes that remain lethal. The long-term objective of the *National Road Safety Strategy (2021-2030)* is to achieve zero road fatalities by 2050 and, therefore, more in-depth research into fatal crashes is needed. Current road crash databases are often engineering-focused and lack data that relate to injuries, in turn limiting the extent to which factors that contribute to fatal injury can be investigated.

The aim of this research was to highlight the benefits of combining data from a police-reported crash database with fatal injury information obtained from coronial autopsy reports. A database of 2800 occupant fatalities combining variables from the Traffic Accident Reporting System (TARS) and coronial autopsy reports was established. Several publications are presented within this thesis that sample from the larger combined dataset. One publication compares the trends in methamphetamine detection and blood alcohol concentration above 0.05g/100mL in driver fatalities, finding an upward trend in methamphetamine detection and a significant downward trend in BAC detection between 2008 and 2018. The presence of significant cardiac disease was also assessed in a sample of fatally injured drivers, finding no significant difference in prevalence between the drivers and a control sample of fatally injured passengers. The presence of seat belt markings was investigated in two publications. Increasing body mass index (BMI) was found to be associated with the presence of seat belt marking as was bilateral pelvic fracture; however, no other injuries were predictive of seat belt markings. Secondly, season of year, as a proxy measure for the amount of clothing worn, changed the incidence of seat belt markings. During winter months the incidence of seat belt markings significantly decreased, suggesting that clothing imposition may mask cutaneous

trauma. Increasing age was found to be significantly associated with the number and pattern of rib fractures. Finally, a case series reporting limb amputations in vehicle-related fatalities found that higher impact speed was associated with limb amputation.

Several other research questions explored the interactions between various occupant, crash and injury factors to further expand our understanding of these factors and how they contribute to patterns of fatal injury. Two common themes emerged, the first being that increasing age is strongly associated with injuries such as rib fractures and cervical and thoracic spinal fractures but, unexpectedly, was not associated with other injuries such as atlanto-occipital fracture. The other was BMI, with high BMI being protective against some abdominal injuries while conversely increasing other injuries. There were also three review publications, including an overview of classification of cause of death, vehicle-related suicides and lethal airbag injuries. The results found in this thesis also highlight the complexities of gathering data retrospectively. The amalgamation of various crash-related databases can provide greater understanding of contributing factors, with the aim of reducing and eliminating motor vehicle fatalities.

Thesis Declaration – Statement of Originality

I certify that this work contains no material which has been accepted for the award of any other degree or diploma in my name, in any university or other tertiary institution and, to the best of my knowledge and belief, contains no material previously published or written by another person, except where due reference has been made in the text. In addition, I certify that no part of this work will, in the future, be used in a submission in my name, for any other degree or diploma in any university or other tertiary institution without the prior approval of the University of Adelaide and where applicable, any partner institution responsible for the joint-award of this degree. I acknowledge that copyright of published works contained within this thesis resides with the copyright holder(s) of those works. I also give permission for the digital version of my thesis to be made available on the web, via the University's digital research repository, the Library Search and also through web search engines, unless permission has been granted by the University to restrict access for a period of time. I acknowledge the support I have received for my research through the provision of an Australian Government Research Training Program Scholarship.

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This Ph.D. thesis is dedicated to the victims of road crashes in South Australia.

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"If I have seen further, it is by standing on the shoulders of giants" – Sir Isaac Newton

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Publications arising from this thesis

O'Donovan, S, Van Den Heuvel, C, Baldock, MRJ & Byard, RW 2020, 'Injuries, death and vehicle airbag deployment', *Medicine, Science and the Law*, vol. 60, no. 2, pp. 147-149, DOI: 10.1177/0025802419892392.

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Thesis Structure

This thesis investigates several aspects of motor vehicle occupants including factors which contribute to a fatal crash, how the patterns of fatal injury of vehicle occupants have changed over time and how occupant characteristics and crash factors influence patterns of fatal injury. This thesis contains ten published research articles, of which three are review articles and seven are original research articles.

The overarching structure of the thesis is as follows: a preliminary analysis of a police-reported crash database to establish the gap in injury data that exists in such databases; a materials and methods chapter; three chapters reviewing the literature, which also incorporate a number of original publications to demonstrate their contribution to the current body of knowledge; an overall data analysis and results chapter, also incorporating two original research articles; and a general discussion. References are provided at the end of each chapter. A number of appendices are also included.

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Abbreviations

MVC	Motor vehicle collision
TARS	Traffic Accident Reporting System
NRSS	National Road Safety Strategy
FSSA	Forensic Science SA
CASR	Centre for Automotive Safety Research
IHD	Ischaemic Heart Disease
CAA	Coronary artery atherosclerosis
CM	Cardiomegaly
SA	South Australia
MA	Methamphetamine
BAC	Blood alcohol concentration (g/100mL)

Chapter One: South Australian motor vehicle fatalities

1.1 Introduction

Motor vehicle fatalities remain a leading cause of death in South Australia (SA). In keeping with road fatality trends across Australia, there has been gradual decline in road fatalities in SA that is attributable to improvements in vehicle design and road infrastructure, emergency response times and a variety of other road safety countermeasures. Despite the decline, the mortality and morbidity associated with motor vehicle crashes (MVCs) continue to present a significant challenge.

Earlier road safety philosophy emphasised driver responsibility and error as the primary causes of road crashes, but this approach has been superseded by the internationally recognised 'Safe System' (Cornelissen et al. 2015; Woolley et al. 2018). A 'Safe System' acknowledges human fallibility (i.e., human error is inevitable within the road system), and that the system must be designed to minimise the impact of these errors (Corben et al. 2010; Green et al. 2021; Turner et al. 2016). The fundamental concept underpinning this philosophy is the finite nature of the human body's tolerance to crash impacts, and the ultimate measure of the system's performance is fatal and serious injury outcomes (Green et al. 2021; Turner et al. 2016). SA has adopted the aims of the *National Road Safety Strategy 2021-2030* (NRSS) to reduce annual road fatalities by 50% and serious injury by 30% by 2030, with the ultimate objective of zero road fatalities by 2050 (*South Australia's Road Safety Strategy to 2031* 2021).

Data collection and research are two crucial elements of achieving the Safe System's aims. As acknowledged in the NRSS 2021-2030, current fatal crash databases are well-resourced and comprehensive, providing the when, where, what, who and why of crashes to assess the performance of current safety countermeasures and inform future safety strategies (Imprialou & Quddus 2019). However, most of these databases are engineering-focused and

seldom include information regarding the specific injuries sustained in fatal MVCs. The paucity of integration between crash and injury data limits research capacity to explore the relationships that may exist between injuries and crash factors, and to validate possible mechanisms or patterns of injury across large samples of real-world data.

As the frequency of fatal MVCs decreases, it is likely that more detailed information will be required that combines crash and injury data to assess the performance of the Safe System and to identify the main factors that continue to contribute to fatalities. The remainder of this introductory chapter will explore and examine the crash data contained within SA's primary road crash database, the Traffic Accident Reporting System (TARS), to identify current limitations of the TARS database and identify the gaps within TARS that could be addressed through the collection of detailed injury data.

1.2 South Australian motor vehicle fatalities

A road fatality in SA is defined as a road user involved in a road crash who dies within 30 days because of injuries sustained in the crash. There are several categories of road user including motorcyclist, pillion passenger, bicyclist, pedestrian, and vehicle occupant. The present thesis and body of research will focus exclusively on motor vehicle occupants. There are two reasons for this: firstly, vehicle occupants comprise the largest proportion of road users in crashes (Bureau of Infrastructure and Transport Research Economics 2022). Secondly, the current research involves in-depth analyses of fatal injury patterns; combining road user groups for such analyses would be problematic, as the mechanisms of injury across road user groups will vary significantly.

The age range for the current research was also restricted to include only motor vehicle occupants aged 18 years or older. Paediatric vehicle occupants were excluded for several reasons, including their distinct and specific crash exposure risks, purposes for travelling in the vehicle and patterns of injury (O'Donovan et al. 2018). Younger children

effectively have no control as to how and why they may be present in a vehicle and are more vulnerable to extraneous factors that could confound any conclusion that could be drawn from the data (e.g., proper restraint use appropriate for the child's age being the responsibility of the driver, primarily the parent). Paediatric motor vehicle occupants also experience different patterns of injury due to the relative size of the child compared to an adult. This difference is particularly evident with seat belts whereby the smaller size of the child affects the performance and function of the seatbelt (Byard & Noblett 2004). Furthermore, the physical and physiological response to an injury may differ. Infants and young children are highly susceptible to head trauma but may have a higher injury tolerance to chest impact due to the elasticity of the paediatric thorax (Tepas 1996). Therefore, for the remainder of this thesis, the term "motor vehicle fatalities" and discussion of trends associated with motor vehicle fatalities refer specifically to motor vehicle occupant fatalities aged 18 years and older.

1.3 Traffic Accident Reporting System (TARS) database

The primary source of crash-related information for this research is the Traffic Accident Reporting System (TARS). Established in 1981, crashes reportable to the South Australia Police (SAPOL) are entered into a database and forwarded to the South Australian Department for Infrastructure and Transport (DIT). The crash information is processed, de-identified and stored in TARS. For this research, TARS was accessed through the Centre for Automotive Safety Research (CASR) at the University of Adelaide under the University of Adelaide Human Ethics Committee (see Ethics Statement 2.2).

TARS includes all levels of crash severity, from property damage only, to fatal crashes. For a crash to be included in TARS, it must involve one or more of the following characteristics: at least one of the persons involved must have been injured, $3000 or more worth of damage must have been caused to the vehicles, or one of the vehicles must have

been towed away. The only injury severity variable provided by TARS is the 'crash severity', that is associated with increasing injury severity in the following order: property damage only <$3000, property damage only >$3000, treatment by a private doctor, treated at hospital, admitted to hospital and fatality. TARS also contains information relating to the nature of the crash (e.g., the type of crash and weather conditions), casualty characteristics (e.g., age, sex, and position in vehicle), vehicle specifics (e.g., vehicle type and year of manufacture), safety countermeasures (restraint use), and time and location of the crash.

Cases of deliberate vehicle collision, as in vehicular suicides or homicides, are not included in the TARS database. As these collisions are deliberate, they are also not counted as part of the South Australian road fatality statistics. Acute medical episodes that result in a fatality but involve only a minor MVC are also excluded from TARS. Finally, collisions that have occurred on non-government or private roads, or on properties such as car parks, driveways, or rural properties are also excluded.

1.4 TARS variables

Several considerations, coding amendments and exclusions were applied to the TARS sample to allow for consistent comparisons across occupant characteristics and crash factors. Crashes without injury (property damage only <$3000, property damage only >$3000) were excluded from analysis. For a number of variables, several categories were grouped together. Crash types that involved hitting a pedestrian, animal or an object on road were combined. Rear seated positions in vehicles were combined to form a single category due to the limited additional information that TARS provides, while the driver and front seat passengers remained distinct categories. Distinction was made between the driver and all other seated occupants due to the active role drivers play and the different in-vehicle structures adjacent to the driver (e.g., the steering wheel). While the middle and left seat remained differentiated, the middle seat is only available in some vehicles, is rarely used and has different safely

properties (e.g., usually a supplemental added seat rather than one built into the car). Distinguishing between the occupant positions in rear seats would only be useful if point of impact information were available but this is absent in TARS.

1.5 Fatalities compared to other crash severities using TARS

Motor vehicle fatalities have been declining in SA between 1995 and 2020 despite increases in population, vehicles registered and licence holders (*Road Crashes in South Australia: Statistical Summary of Road Crashes & Casualties in 2020* 2020). Such a decline could be due to a reduction in the risk of a lethal injury given a crash or could be due to a decline in crash numbers but with the risk of death per crash remaining the same. To investigate the decline in motor vehicle fatalities, the proportion of casualty severity was plotted against the total number of crashes over time, for all crashes resulting in some degree of injury to at least one vehicle occupant (private doctor, treated at hospital, admitted to hospital and fatality) (Figure 1.1).

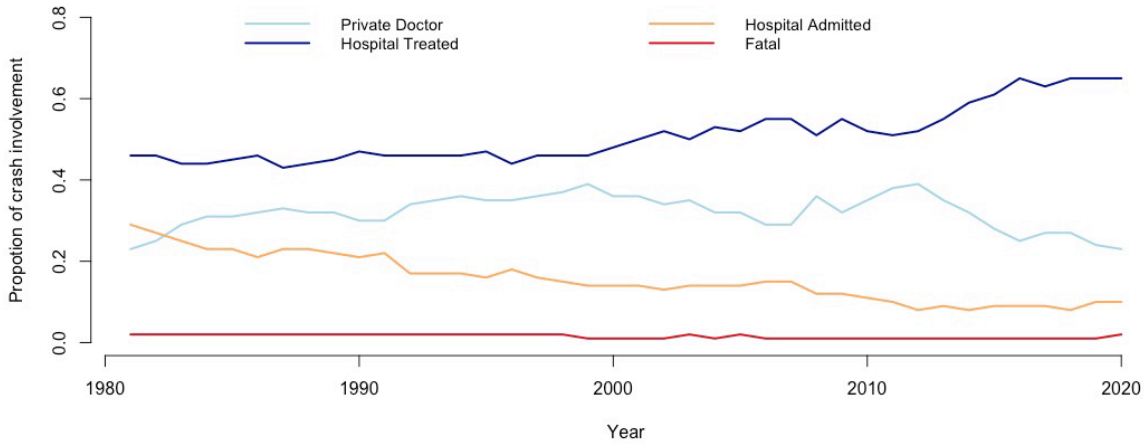

Figure 1.1 Proportion of injury crashes by crash severity (1981-2020)

As seen in Figure 1.1, the proportion of fatalities remained steady with a slight decline from the late 1990s onwards. Hospital admission rates have been steadily declining, although appear to have plateaued between 2010 and 2020. This is likely due to the adjustment in the data collection methods used by SAPOL in 2008, as SAPOL officers began verifying formal admissions to hospital. By 2012, this practice was standard across the state and improved the

classification of hospitalised occupants (*Road Crashes in South Australia: Statistical Summary of Road Crashes & Casualties in 2020* 2020). These improvements in classifications likely contributed to the sharp rise in hospital treated occupants from the early 2010s. Interestingly, the rise in hospital treated occupants also appears to match the decline in the proportion treated by private doctors. It should be noted that following the improvement in injury classification methods in 2012, hospital treated, and private doctor treated cases are the only two casualty severities that do not require verification. Although verification of hospital admission cases results in differentiating these from hospital treated cases for the more seriously injured cases, there is no formal method of differentiating private doctor and hospital treated casualties for the less seriously injured cases. The classification decision is based on what the SAPOL officer observed at the scene, which may negatively impact the accuracy of classification.

1.6 Assessing the utility of TARS

The steady proportion of fatal crashes and declining proportion of hospital admissions (Figure 1.1) suggests that there may crashes which remain lethal despite road safety improvements. This raises the question of which factors distinguish serious crashes resulting in hospital admission from fatal crashes. To investigate this, a preliminary analysis using the TARS database was performed. As the focus of this preliminary analysis is on a cross-sectional comparison between injury crashes and fatal crashes rather than variable changes over time, a shorter and more recent time frame was selected for this analysis (1995-2020). TARS variables that were selected for this analysis included casualty age, casualty sex, position in vehicle, restraint status, type of crash, whether the crash was inner Adelaide (capital city of SA), outer Adelaide or country, speed limit of the road where the crash occurred, road geometry, road alignment, traffic controls (e.g., traffic light, stop sign, rail

crossing or no controls), type of vehicle, year of vehicle manufacture, vehicle movement and number of vehicle occupants.

To evaluate occupant and crash factors within TARS that distinguish between serious injury crashes and fatalities, a logistic regression model was developed from a training dataset (a 60% randomised sample of the data), and the accuracy and validity of the model was assessed using a testing dataset (the remaining randomised 40% of the data). A stepwise logistic regression model ($\alpha = 0.05$) was performed on the training dataset using R studio statistical software (Version 1.4). Model selection was determined by Bayesian Information Criterion (BIC). A confusion matrix was then calculated using the test data to evaluate the predictive performance of the logistic regression model. The results of the logistic regression and confusion matrix are summarised below; for full model output, see Appendix A.

The model showed that increasing age was associated with an increased probability of fatality when compared to serious injury. Occupants who were male, not wearing a seatbelt or ejected from the vehicle were also more likely to be fatally injured. The speed limit of the road and the type of crash were also significant predictors (Appendix A Table A1.1). Post hoc analysis of the speed limit, with Tukey correction to control for family-wise error rate, revealed that speed limits over 70 km/h were consistently more likely to result in fatalities than lower limits (Appendix A Table A1.3).

Interestingly, the area of crash was not included in the final model output suggesting that the distance from a city or major town, and therefore from medical facilities, either does not predict fatality or is being captured by another variable in the model (e.g., speed limit). A limitation to be noted with the analysis is the inability to ascertain whether these factors are predicting involvement in a high impact severity crash or greater injury risk.

1.7 Limitations of TARS

The test dataset was then used to evaluate the accuracy of the model in predicting fatalities. The model had an ostensibly high accuracy of approximately 89% but a third of fatalities were incorrectly classified as serious injury cases (Appendix A Table A1.2). It is likely that the model accuracy is affected by the majority of the sample consisting of serious injury crashes. Fatal crashes, compared to serious crashes, are infrequent and, as the model demonstrates, the data available in TARS appear insufficient for identifying the determinants of a fatal versus serious injury crash. Therefore, a model to differentiate fatalities from serious injuries would benefit from the inclusion of other variables not included in TARS. One such type of variable would be the injuries sustained by the occupant. With TARS only having a single indicator for overall injury severity, there would be a clear benefit to incorporating injury information alongside other more engineering-focused variables. This additional information, likely to be useful for differentiating fatalities from serious injuries, would also be useful for identification of countermeasures and contributing to the development of road safety strategies.

As TARS is compiled from police-reported crashes, the quality and accuracy of the data are determined by the reporting officer. Some variables such as seat belt wearing may be difficult to determine, as the officer is likely to arrive at a collision scene after vehicle occupants have exited or been removed from the vehicle and the officer may be unable to ascertain confidently whether a seat belt was worn. For such cases, the officer will often code seatbelts as being “Fitted” in the vehicle without classifying them as having been “Worn” or “Not Worn”. Crash type is another variable that has intrinsic limitations, whereby the crash type is characterised only by the initial collision with the first ‘unit’ (e.g., another vehicle, pedestrian, or tree). If multiple vehicles are involved with multiple impacts, this cannot be represented easily in TARS.

TARS also lacks an accurate estimate of travelling speed for crash-involved vehicles. Determining speed at the time of collision is difficult and therefore not routinely performed in less serious cases. For fatal crashes in which a charge could potentially be brought against one of the drivers, police will use sophisticated computerised reconstruction techniques to determine travelling speed, or for certain vehicles will download data from the Event Data Recorder. In the majority of TARS cases, however, only very broad estimates of travelling speed are provided. Other factors that influence crash severity and outcomes such as level of cabin intrusion and direction and point of impact are also not recorded in TARS.

The majority of TARS information is accurate but there are several common factors of interest for the present research for which TARS data can be inaccurate due to errors in data collection or unattainable due to the nature of fatal crashes (age, sex, restraint wearing status, position in vehicle). This is an inherent limitation in all retrospective data analysis. For the present research, age and sex will be determined conclusively using the coronial autopsy data to correct some of these discrepancies. There are also key variables that were not recorded in TARS until more recent years. For example, seat belt wearing was only recorded from the early 1990s and the vehicle age was not routinely recorded in earlier years.

1.8 Summary

The most significant limitation of TARS is the lack of information about the associated injuries. Casualty severity is the only parameter that indicates injury severity and, as the above analysis has shown, crash information alone is inadequate for identifying what differentiates fatal crashes from serious injury crashes. Preliminary analysis demonstrated that older age and male sex independently increase fatality risk, but the current model does not indicate whether this risk is related to the likelihood of fatal crash involvement or the risk of fatality from injuries. These concepts will be explored within this thesis and how the

integrated fatal crash and injury data can be used to assess both crash involvement and injury risk factors.

1.9 Objectives of the research

Motor vehicle fatalities, despite declining in number, remain a significant challenge. The preliminary evaluation identified several limitations of the South Australian TARS database and showed that analyses using primarily crash characteristics and basic occupant characteristics fail to distinguish between fatality and serious injury crashes. This highlights the gap in data collection whereby databases consisting of predominantly crash data with minimal injury data are insufficient to answer some of the complex questions that exist in road safety research.

To address the gap in fatal injury data, the main objective of the current research was to collect injury information in cases of fatal crashes from coronial autopsy reports and crossmatch with corresponding crash data from TARS to develop a combined dataset. Subsequent publications and analysis use the combined dataset to evaluate factors which contribute to fatal crashes and explore the existing relationships between various occupant characteristics, crash factor sand fatal injury patterns.

The integration of fatal injury data with crash data allowed for specific research questions that were used to explore the relationships between injury patterns, occupant characteristics and crash factors in fatal South Australian occupant MVCs. Fatal crashes, by their nature, are complex events with several factors that can contribute to fatal outcomes and, therefore, the overarching research hypothesis of this thesis was that *occupant characteristics, crash factors and safety countermeasures can influence fatal crashes and alter the patterns of fatal injury*.

Throughout the course of this research several other issues relating to occupant fatal MVCs arose that warranted further investigation in the form of independent descriptive

reviews and studies. The following outlines the various reviews and studies that were addressed within the scope of this thesis with relevant study objectives and methodological criteria:

Descriptive Reviews

Title: *Classification of cause of death in motor vehicle crashes*

Objective: To review the various causes of death that are associated with MVCs as determined at autopsy. Typically, cause of death in MVCs has been attributed to blunt force trauma but throughout the course of the research, several other causes of death were noted.

Title: An overview of *suicides related to motor vehicles*

Objective: To review the various forms of vehicle-related suicide. Deliberate MVCs are not recorded in TARS. It was therefore pertinent to review the various methods of suicide that involve a motor vehicle to highlight further the difficulties that occur in making this determination. Representative cases were identified, and a review was conducted of the various methods that may be employed.

Title: *Injuries, death and vehicle airbag deployment*

Objective: As the presence and deployment of an airbag is not recorded in TARS, a review was conducted to evaluate the lethal mechanisms associated with airbags. Airbags are a relatively new safety device in vehicles. Despite their demonstrated effectiveness in injury prevention, lethal injuries have occurred from airbag misfire, malfunction, or defect.

Primary Research Studies

Title: *Methamphetamine and alcohol detection in vehicle-driver fatalities in South Australia: A 10-year survey (2008-2018)*

Objective: To examine the most recent trends in the detection of methamphetamine and a blood alcohol concentration above 0.05g/100mL in drivers involved in fatal crashes. Drivers were identified using the TARS database and the toxicological results from the fatal injury data were obtained to study the trends in detection between January 2008 and December 2018. It was predicted that alcohol detection would be declining in response to safety countermeasures, but that methamphetamine would be increasing, mirroring increased community use.

Title: *Cardiac disease and driver fatality*

Objective: To determine the incidence of severe coronary artery atherosclerosis (CAA) and cardiomegaly (CM) in driver fatalities attributed to trauma compared to a control group of fatally injured passengers. Drivers were identified using the TARS database and prevalence of CAA and CM were extracted from fatal injury data.

Title: *Seat belt injuries and external markings at autopsy in cases of lethal vehicle crashes*

Objective: To explore the relationship between external cutaneous seat belt marking and underlying injuries at autopsy. The presence of cutaneous markings was assessed in cases from 2014 to 2018 and related to underlying injuries. It was expected that seat belt markings may indicate the presence of underlying abdominal, chest and occasionally cervical spinal injury.

Title: *Seasonal variation in cutaneous seat belt markings in fatal vehicle crashes*

Objective: To determine if season of year affects the incidence of seat belt marking at autopsy. It was predicted that the incidence of seat belt marking would decrease in the winter months as occupants would be wearing more clothing.

Title: *Lethal mechanisms in cases of inverted suspension from lap component of seat belts*

Objective: To report and review a unique presentation of postural asphyxia caused by seat belts.

Title: *Upper and lower limb amputation in vehicle-related fatalities*

Objective: To determine the incidence of upper and lower limb amputation in vehicle collisions. The frequency of limb amputation in MVCs is rare, with the majority of cases in the clinical literature consisting of post-injury amputation of the upper limbs following severe limb injury.

Title: *Fatal blunt chest trauma: an evaluation of rib fracture patterns*

Objective: To examine the relationship between rib fractures and various occupant and crash characteristics. Combined TARS and fatal injury data were selected between 2000 and 2020 with supplemental data collected from autopsy reports to obtain the number, side and location of ribs fractured. Age was expected to be one of the strongest predictors of rib fracture and the number of ribs fractured.

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Chapter Two: Materials and Methods

2.1 Introduction

The Materials and Methods outlined in this chapter have been applied to retrospective data collection and analyses pertaining to the work presented and published in this thesis. Individual studies may have required only a subset of the data, restricted time frames or additional information from the coronial autopsy reports, which are outlined in each relevant paper in subsequent chapters.

There are several factors that modify, reduce, or increase injury risk and severity for occupants in MVCs. These factors relate to crash characteristics, occupant demographics, vehicle design, safety devices and technologies such as seat belts and airbags (further explored in the subsequent chapters). These factors have improved or changed with time and, therefore, the pattern of injuries experienced by vehicle occupant has also likely changed. The aim of the research was to develop a dataset of mixed retrospective sources of crash and injury data to investigate the change in injury patterns and to explore the relationships between injury patterns, demographic factors, and crash characteristics.

The methodology was designed to account for the complexities of cross-institutional data collection by maximising data capture through dual coronial autopsy data sources and minimising missing or unknown variables through data cross-matching at multiple intervals.

2.2 Ethics Statement

This research was conducted in accordance with National Health and Medical Research Council (NHMRC) *National Statement on Ethical Conduct in Human Research 2007* and approved by the University of Adelaide Low Risk Human Research Ethics Review Group (Faculty of Health and Medical Sciences) (ethics approval: H-2018-191) and Forensic Science SA Research and Development Committee (ethics approval: PATH 18-11).

2.3 Sample selection

The sample selected for this research consists of all motor vehicle occupants aged 18 years or older who died following a road traffic collision on South Australian roads between January 1981 and December 2020. As outlined in Chapter One, the inclusion and exclusion criteria as related to the TARS database were applied prior to data extraction from coronial autopsy reports.

2.4 Coronial autopsy reports

A search of coronial autopsy reports was conducted at Forensic Science SA (FSSA) for all occupant motor vehicle fatalities occurring between 1981 and 2020. Cases from 1981 to 2000 were in hardcopy format and cases from 2000 to 2020 were digitised. All occupant vehicle fatalities within the state of South Australia during the study time frame had been reported to the State Coroner in accordance with the current State *Coroner's Act* criteria and all had undergone full police, coronial and pathological investigations.

Prior to data extraction, individual cases needed to be verified as vehicle crashes within the TARS study parameters as outlined above. Casualty identifiers such as age, sex and date of crash were used to make this initial confirmation. As noted earlier, the focus was on vehicle occupants, and so fatally injured motorcyclists, cyclists and pedestrians were excluded. However, the fatal injuries and injury mechanisms that occur in collisions that are deliberate (e.g., suicide) were determined to be beneficial to injury pattern analysis and so were included in the data extraction from coronial autopsy reports, even though these cases are not included in TARS. Collisions that occurred on private or non-governmental roads or property remained excluded from data collection. This decision was made based on the probability that the majority of private roads and private properties would not conform to the safety standards of the government-controlled road transport system and the results could confound the injury pattern analyses based on the TARS cases.

Demographic, toxicological, and fatal injury data were extracted from coronial autopsy reports, de-identified and codified into a password protected Excel spreadsheet where they were combined with other variables from the corresponding, cross-matched TARS case. The spreadsheet did not include any individual casualty identifiers other than age, sex, and date of crash and/or death which are publicly available on Bureau of Infrastructure and Transport Economic (BITRE) and South Australian Police (SAPOL) databases.

2.4.1 Demographic factors

Demographic characteristics that were extracted from the coronial autopsy reports include age in years and biological sex. Height (metres) and weight (kilograms) were extracted to determine body mass index (BMI) (calculated by dividing weight in kilograms by height in metres squared). Date of death was also included as this may not always be the same as the crash date. Location of death was also recorded as either being at the scene, in transit to hospital or in hospital. Deaths at the scene may not all occur instantaneously but it implies that the injuries sustained were generally rapidly lethal. Death in transit to hospital suggests the patient was stable enough to be placed into the ambulance and death in hospital indicates the injuries were not immediately lethal, although the injury sequalae resulted in death.

2.4.2 Toxicological analysis

The *Road Safety Act 1961* in SA prohibits the operation of a motor vehicle whilst under the influence of alcohol or illicit drugs. The legal blood alcohol concentration (BAC) limit was previously 0.08g/100mL in SA but this was reduced to 0.05g/100mL in 1981. FSSA has been routinely screening post-mortem cases for alcohol. Drivers are also prohibited from driving in SA while testing positive for N-methyl-alpha-methylphenethylamine (methamphetamine), delta-9-tetrahydrocannabinol (THC) or 3,4-methylenedioxy-N-methamphetamine (MDMA).

Routine toxicological analyses performed on post-mortem samples at FSSA screen for alcohol and common drugs. The presence and amount of alcohol and the three prescribed drugs were only recorded for drivers using post-mortem blood samples. Only drivers with the active form of the drugs present at autopsy (i.e., excluding drug metabolites) were classified as drug positive. Levels of methamphetamine, THC or MDMA were not recorded; only the presence of the drugs was noted as post-mortem distribution may have affected the drug concentrations at the time the sample was taken.

2.4.3 Causes of death, fatal and associated injuries

Fatal injury data that were collected include cause of death in addition to the injuries caused by the collision. The cause of death was attributed by the examining pathologist to the injury or disease which produced sufficient physiological disruption to the body to result in death. To effectively collate and analyse causes of death over time, the causes were coded and grouped by the injured body region.

The codification of fatal and associated injuries at autopsy presented a complex problem. The majority of common injury coding systems base the injury severity scale on the injury's threat to life (i.e., an injury will be coded as more severe if the injury increases the risk of death). The injuries observed at autopsy are permanent and unchanging, with no potential to become more or less severe. Thus, this presents a challenge to quantifying the severity of injuries at death as it is difficult to comment on the potential outcome of certain injuries had the individual remained alive. Skeletal fractures can be recorded as present or absent, but it is more difficult to distinguish between minor and severe organ and soft tissue injuries once death has occurred. In an attempt to overcome this challenge, definitions of injury severity from an existing injury scoring system, the Abbreviated Injury Scale (AIS) (Association for the Advancement of Automotive Medicine, 2016), were adapted to establish a threshold of severity that the injury description at autopsy had to be of equivalent to, or

exceed, to be included in the dataset. The AIS, specifically AIS 2015, provides a 6-point ordinal scale that indicates injury severity with 1 being minor and 6 being maximal severity. An AIS of 3 indicates serious injury and was therefore chosen as the injury description threshold. The injury description at autopsy had to be equivalent to or exceed the AIS definition of a serious (AIS 3) injury. For example, for a liver laceration to be considered 'present' in the dataset, the definition of injury provide by the forensic pathologist must meet or exceed "intraparenchymal laceration >10cm or >3cm parenchymal depth or have major duct involvement".

Not all organ system injuries were included in the coding system. Lethal injury to the genitourinary and other major body regions such as the neck and skin are rare in vehicle collisions and so were not included as separate variables. If injuries were noted in these organ systems, they were usually in conjunction with other severe injuries which would then be captured in data collection. Injuries were extracted and tabulated in Excel spreadsheets.

2.4.4 Challenges of retrospective autopsy data collection

As outlined above, a significant challenge was in quantifying injuries. This process relied heavily on the reporting of injuries made by the pathologists which presented another barrier to standardising injuries, as the approach and technology used in post-mortems has changed substantially across the study time frame. For example, early autopsy reports lacked case comments or information that could assist in the cross-matching with TARS data.

There has been a move from full autopsies at FSSA to radiographic (CT) imaging and external reviews of cases. This has changed data capture for some injuries (e.g., radiographic imaging of the lungs cannot clearly differentiate small lacerations from pulmonary contusions unless major). This has only occurred in the last five years of this project and while it does not markedly impact the capture of significant injuries, it may influence the capture of smaller associated injuries.

As individual forensic pathologists may have their own nomenclature, style of autopsy report and emphasis on injury types or anatomical landmarks, all data extraction from coronial autopsies were reviewed by a Senior Forensic Pathologist.

2.4.5 Supplementing missing cases

Although the majority of cases underwent a medico-legal autopsy at FSSA, a small number of cases were not able to be located in the search of coronial autopsy files as they had been examined at other hospitals. Of the cases that still could not be located, several underwent pathology reviews, whereby the cause of death was determined by a physician and verified by a forensic pathologist with no autopsy examination.

2.4.6 Remaining limitations following data collection from coronial autopsy reports

As highlighted in Chapter One, there were several limitations and gaps in the TARS database, most significantly the lack of injury information, that the addition of data from coronial autopsy reports was aimed at addressing. Coronial autopsy reports provided the required injury information but other key factors relating to crash circumstances, known to modify injury severity, could not be obtained. These factors include speed of impact, airbag deployment, degree of vehicle intrusion, point of impact and direction of impact. Although a number of coronial autopsy reports commented on or described the crash type, degree of intrusion or airbag deployment, crash-related information contained within the reports were considered secondary accounts and not considered reliable for the purpose of this study.

Fatal injury information was also collected from Coroner's reports in a portion of cases and, although Coroner's reports do provide additional crash-related information, there were several reasons why Coroner's reports were not used to extract the missing factors. Primarily, the sample of Coroner's reports was incomplete across the study timeframe and therefore would not have provided a complete representation of data capture. Additionally, mechanical reports or images were not consistently included in Coroner's reports, particularly in earlier

reports. Furthermore, travelling speed and speed of impact are difficult to accurately determine post-crash, even with coronial investigation and crash reconstruction.

2.5 Final variables and statistical analysis

The final variables that were selected to be included in the analysis from the combined TARS and coronial autopsy reports are outlined in Table 2.1.

Table 2.1 Final variables from TARS and coronial autopsy reports

Occupant characteristics	Crash factors	Injury types
Age	Seat belt wearing	Cause of death
Sex	Year of vehicle manufacture	Closed head injury without skull fracture
BMI	Vehicle age^	Closed head injury with skull fracture
Height	Position in vehicle	Open head injury
Weight	Vehicle type	Location of skull fracture (where applicable)
Year of death		Brainstem injury
Location of death		Presence and location of facial fracture
Toxicology results (Drivers only)		Presence and location of spinal fracture including atlanto-occipital fracture and if the spinal cord was transected
		Presence of heart, lung, aortic or other cardiovascular injury with an AIS equivalent description above 3 including the diaphragm
		Presence of one or more rib fractures include sternal fracture
		Presence of liver, spleen, kidney, intestinal/mesenteric or abdominal vascular injury with an AIS equivalent description above 3
		Presence and location of pelvic fracture
		Presence and location of extremity fracture

^Vehicle age derived from year of vehicle manufacture and year of crash (TARS variable)

As discussed in Chapter 1.7, some categories within the TARS database are limited and may not represent the true crash circumstances (e.g., speed limit and crash type) and have therefore been omitted from the combined dataset. Additional descriptions of causes of death and types of injury are outlined in Appendix B Table 1 and 2. All statistical analyses included in this thesis (including publications) were performed using R studio statistical software

(version 1.4) (R Development Core Team). As each publication investigates a different research question (and therefore may have modified or additional information derived from the combined dataset), the details of each statistical analysis used are included within the publications. All other details regarding the statistical analysis are outlined in the results (Chapter Six).

2.6 Reference list

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2.7 Publication: Causes of fatalities in motor vehicle occupants: An overview

S. O'Donovan, C. Van Den Heuvel, M.R.J. Baldock, R.W. Byard

The cause of death in the majority of MVCs is blunt force trauma, either to an isolated area of the body or multiple body regions. However, there are other injury patterns and causes of death that result from MVCs. As this study involves the assessment of patterns of injury, it was pertinent that an overview of the various causes of death that might be encountered at autopsy as a result of a MVC was conducted.

Statement of Authorship

Title of Paper	Classification of deaths in motor vehicle occupant fatalities
Publication Status	☒ Published ☐ Accepted for Publication ☐ Submitted for Publication ☐ Unpublished and Unsubmitted work written in manuscript style
Publication Details	O'Donovan, S, Van Den Heuvel, C, Baldock, MRJ & Byard, RW 2022, 'Causes of fatalities in motor vehicle occupants: An overview', *Forensic Science, Medicine and Pathology,* advance online publication, DOI: 10.1007/s12024-022-00503-3.

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Name of Principal Author (Candidate)	Siobhan O'Donovan
Contribution to the Paper	Performed literature review, wrote manuscript
Overall percentage (%)	85%
Certification:	This paper reports on original research I conducted during the period of my Higher Degree by Research candidature and is not subject to any obligations or contractual agreements with a third party that would constrain its inclusion in this thesis. I am the primary author of this paper.
Signature	Date: 27/05/2022

Co-Author Contributions

By signing the Statement of Authorship, each author certifies that:

- the candidate's stated contribution to the publication is accurate (as detailed above);
- permission is granted for the candidate in include the publication in the thesis; and
- the sum of all co-author contributions is equal to 100% less the candidate's stated contribution.

Name of Co-Author	Corinna Van Den Heuvel
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Name of Co-Author	Roger Byard		
Contribution to the Paper	Co-supervisor of Siobhan, helped to evaluate and edit the manuscript, acted as corresponding author		
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REVIEW

Causes of fatalities in motor vehicle occupants: an overview

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Abstract
Injuries from motor vehicle collisions are frequently encountered in routine forensic practice. While the most common lethal events involve blunt force trauma with injuries to the head and neck, chest, abdomen, pelvis and limbs, review of the literature and case files shows that a wide variety of other fatal situations can occur that may involve sharp force and penetrating trauma, incineration, drowning, asphyxia, organic diseases and combinations of these. The following overview details potential factors that may contribute to death following vehicle crashes.

Keywords Vehicle crash · Blunt force trauma · Sharp force and penetrating trauma: incineration · Drowning · Asphyxia

Introduction

Motor vehicle collisions (MVCs) remain a leading cause of morbidity and mortality globally [1] and continue to occupy a considerable portion of medico-legal caseloads. Motor vehicle occupants comprise a substantial proportion of road fatalities [1]. Injuries most commonly sustained by motor vehicle occupants are usually caused by blunt force trauma although deaths may also involve penetrating and sharp force trauma, fire, drowning, asphyxia and organic diseases. The following overview outlines potential factors identified at autopsy that may contribute to death in occupants involved in MVCs.

Classification of deaths

Blunt force

Blunt force trauma is the leading cause of death for motor vehicle occupants with head injury being the most common lethal event [2, 3]. Head injuries may involve focal or diffuse trauma with primary impacts to the head causing skull fractures and cerebral haemorrhage, contusions and lacerations [4]. Depressed skull fractures or basilar fractures (including hinge and ring fractures) are often life-threatening given the potential for associated cerebral injury [5]. Basal-frontal skull fractures often have concomitant facial fractures which, although usually not life-threatening, may compromise airway integrity and/or cause significant haemorrhage. Axial rotational forces and angular acceleration, usually in combination with head impact, may cause shearing of axonal connections with diffuse axonal injury (DAI) [5, 6]. Mild forms of DAI may be difficult to recognise clinically and a neuropathological examination may be required for confirmation at autopsy. However, severe forms of DAI may produce visible lesions that involve the brainstem or corpus callosum [7].

Spinal cord injury is not uncommon in vehicle collisions and is often accompanied by vertebral fracture and/or dislocation. Upper cervical fractures are associated with the rapid hyperextension of the neck caused by extreme deceleration or chin contact with the instrument panel or steering wheel [8]. The atlanto-occipital joint is particularly vulnerable to fracture and dislocation during neck hyperextension which can cause rupture or tearing of the brainstem and/or spinal cord [9, 10]. Functional decapitation may also occur if the skin remains intact but the internal neck structures are disrupted below the occiput [11]. Decapitation may also be a result of vehicle-facilitated suicide by ligature strangulation whereby a ligature is tied

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to a fixed object, usually a tree, with the other end around the individual's neck and then the vehicle is driven away at a high speed [11–13].

Blunt chest trauma, often from impact with the vehicle interior, results in a cluster of thoracic injuries, some of which are immediately lethal. Fatal injuries to the heart include full thickness lacerations with penetration of the atrial or ventricular walls or, in extreme cases, complete avulsion. A phenomenon known as commotio cordis may also occur in cases of significant non-penetrating chest impact with no observable cardiac injury [14]. In this situation, impact may disrupt the electrophysiological rhythm of the heart and cause immediate cardiac arrest [15].

Lethal aortic injuries such as transection or avulsion cause rapid blood loss with death typically occurring at the scene of the collision [16]. Multiple rib fractures commonly occur following blunt chest trauma and are frequently associated with injuries to the lungs and intrathoracic vessels [17]. Three or more consecutive rib fractures at multiple locations in the same ribs can produce a flail chest which results in paradoxical movement of the flail segment during respiration [18]. While the paradoxical movement reduces pulmonary efficiency, associated lung contusions may also greatly reduce pulmonary perfusion and gas exchange [19]. Rib fractures from vehicle-related trauma must be distinguished from injuries sustained during attempted resuscitation [18, 20, 21].

Injuries to highly vascularised organs in the abdomen such as the liver, spleen and kidneys may result in significant blood loss, and in the setting of multiple trauma, abdominal injury may contribute to hypovolemic shock and exsanguination [22]. Significant torso injuries are frequently associated with pelvic fractures and destabilisation of the pelvic ring which can also damage major vessels, further contributing to blood loss [23].

Catastrophic blunt force trauma causing substantial body/organ disruption commonly results from collisions characterised by high impact speeds or impacts with objects larger than the vehicle itself, such as heavy vehicles or trains. Cases of whole body transection, typically occurring horizontally across the abdomen or pelvis, have also been reported and are often associated with severe damage to the vehicle cabin [24].

On occasion seat belts may be responsible for serious fractures, organ injuries and vessel transection, particularly in high speed collisions [25, 26].

Sharp force and penetrating injuries

Sharp force (SF) or penetrating trauma typically occurs concurrently with blunt force injuries [27, 28]. SF injuries may be caused by broken glass from the windscreen and windows [29] and penetrating injuries are caused by loose, airborne objects from inside the vehicle or external objects that enter the vehicle cabin during the collision [30]. Fatal penetrating injuries have also been reported from the malfunction and inappropriate deployment of airbags [31, 32]. Impalement is a severe form of penetrating injury characterised by an object penetrating the body [27]. Objects causing impalement are often from parts of the external road environment such as wooden guard rails or metal fences, or tree branches [27, 28, 33–35].

Incineration and inhalation of products of combustion

Incineration may occur as the primary cause of death or may follow lethal injury after a collision. As occupants are often either able to self-extricate from a vehicle or are removed by bystanders prior to the vehicle catching alight, incineration is not a common event [36]. A carboxyhemoglobin (COHb) saturation above 30% and/or the presence of soot in the airways at autopsy are in keeping with survival for some time after the collision [37]. Severe charring and thermal injuries may complicate pathological examination as differentiating traumatic from heat-related fractures may be difficult, especially when there is overlap [38, 39]. Incineration may also mask or destroy soft tissue and organ damage, making it difficult to ascertain the range of possible lethal blunt force injuries.

Drowning

Drowning when vehicles enter water is uncommon, as a driver who is not incapacitated can usually escape from the cabin. Driver incapacitation may, however, occur as a result of an acute medical episode, intoxication or due to loss of consciousness or severe injuries from the impact. In floods, drivers may either be trapped within a vehicle cabin or may be drowned in fast flowing currents once the vehicle has been exited. The possibility of a vehicle-assisted suicide should also be considered when a vehicle has apparently been deliberately driven into a body of water.

Asphyxia

There are several types of asphyxial events which may compromise tissue oxygenation that include thoracic compression [40], neck compression or an abnormal body position [41–43]. Crush asphyxia refers to mechanical compression of the thorax, common in roll-over crashes with complete or partial occupant ejection resulting in a vehicle landing on top of the occupant compressing the thorax [40]. Thoracic compression may result in fractures to the ribs or sternum,

lung and vascular injuries and other injuries associated with blunt force trauma [40]. Accidental asphyxia due to hanging is almost exclusively the result of a seat belt slipping over the thorax onto the neck and may have concomitant neck injuries such as ligature markings or fracture of the hyoid bone or thyroid cartilage [43]. "Submarining" under a seat belt may occur in children or people with smaller stature [44–46]. Suicidal hanging from seat belts has been reported [47]. Positional or postural asphyxia may occur if an occupant is placed in a position which impedes respiration through neck flexion, inversion or suspension from a seat belt [41]. Positional asphyxia may occur without injury; however, classic pathological features of asphyxia including facial petechial haemorrhage, particularly of the conjunctiva of lower eyelids, may be present [40, 42, 43].

Death from asphyxia may be associated with concomitant head injuries, other blunt force trauma and alcohol/drug intoxication as these may reduce the level of consciousness and interfere with attempts at self-extrication [41, 48, 49].

Toxicity

Death caused by carbon monoxide (CO) toxicity in vehicles is most often the result of fire or deliberate exposure, usually from car exhaust. Although rare, cases of accidental CO toxicity have been reported, often involving leaking exhaust systems [50–53]. In these cases, the odourless, tasteless and colourless nature of the CO meant that the leaks went undetected.

Byard et al. reported three cases of suspected gasoline toxicity following vehicle roll-overs with damage to fuel tanks. Volatile hydrocarbons were detected in post-mortem blood and corrosive external burns were documented associated with exposure to gasoline [54].

Natural disease

Certain underlying diseases may precipitate or cause a crash by impacting driving ability, perception and consciousness. Medical episodes include (but are not limited to) stroke, myocardial infarction/ischemic heart disease, pulmonary embolism, abdominal aortic aneurysm (AAA) rupture, seizures, hypoglycaemia, dementia and mental illness [55–59]. Several of these, such as myocardial infarction, stroke or rupture of an aortic aneurysm, may have warning signs enabling a driver to pull over or to reduce vehicle speed before losing consciousness [58]. However, seizure-related conditions such as epilepsy or syncopal episode may rapidly incapacitate a driver [60].

Organic underlying disease may also contribute to death following trauma in the elderly as the body's physiological response to trauma is reduced with age, often with the presence of other age-associated diseases [61].

Sequelae of injury

Improvements in pre-hospital care and emergency responses have greatly enhanced crash survivability but there are several injury sequelae that can contribute to mortality following a collision.

- Secondary injury mechanisms from TBI such as cerebral ischemia, hypoxia, oedema and raised intracranial pressure may be fatal despite surgical or pharmacological intervention [62, 63].
- Cerebral swelling and neurogenic pulmonary oedema from acute head trauma may occur very rapidly following head trauma [64].
- Fractures, particularly of long bones, may produce fat emboli.
- Vertebral or cerebral artery dissection may occur.
- Multi-organ failure, sepsis and hospital-acquired infections such as bronchopneumonia may develop [3].

Conclusion

This overview has demonstrated that a wide variety of immediate and delayed lethal mechanisms may complicate vehicle crashes. At autopsy, the possibility of the additive effects of several disparate lethal mechanisms should therefore be considered.

Key points

1. Motor vehicle crashes comprise a significant portion of medico-legal caseloads.
2. Blunt force trauma is the most common lethal event frequently with a combination of injuries to the head, chest, abdomen and limbs.
3. A variety of other fatal situations may occur that may involve sharp force and penetrating trauma, incineration, drowning, asphyxia, organic diseases or combinations of these.
4. Mechanism may be immediately lethal or death may occur from post-trauma sequelae.

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Chapter Three: Fatal crash risk

3.1 Introduction

Fatal MVCs are the result of complex interactions between various occupant, vehicle and road infrastructure factors that contribute to the likelihood of a crash occurring and/or to the severity and patterns of injury sustained by the occupant in the crash. These factors may be intrinsic occupant characteristics such as age or sex, external factors such as vehicle design and safety devices, crash circumstances such as speed of impact and crash type, or extrinsic elements within the road system such as emergency response times and rural locations that contribute to fatal outcomes. This thesis will focus on factors relating to the inherent occupant characteristics and selected aspects of crash circumstances and vehicle safety devices that mediate injury severity for occupants and which can be assessed with the combined TARS and fatal injury dataset.

The following three chapters will explore the literature on the various occupant characteristics and safety devices that contribute to risk of involvement in fatal crashes and modify patterns of injury. The results of several journal articles published within the scope of this thesis are integrated within this chapter to demonstrate the wide-ranging applications of fatal injury data collected from coronial autopsy reports. Publications referenced within each chapter can be found in full at the end of each chapter. This chapter will focus on driver factors which are known to increase the risk of crash involvement. Saliently, driver factors are not the only risk factors that contribute to fatal crashes, nor can all driver factors be assessed using the combined injury and crash dataset however, it was pertinent to acknowledge that these risk factors exists and some factors (such as driver intoxication or presence of medical conditions) can be investigated using a combined dataset.

3.2 Driver factors that increase fatal crash risk

The operation of a motor vehicle is a complex task involving the distribution of attention across a dynamic environment, and processing and reacting to multiple sources of information. Previous research has established various human factors related to driver performance or behaviour that increase the possibility of being involved in a fatal crash. The most common and well-known factors are referred to in Australia as the 'Fatal Five'. Broadly, the 'fatal five' factors are: speeding, driving while under the influence of alcohol or drugs, failure to wear a seatbelt, driver distraction and fatigue.

3.2.1 Alcohol

Alcohol intoxication is one of the most recognised risk factors associated with involvement in fatal crashes (Connor et al. 2004; Peck et al. 2008). Alcohol (specifically ethanol) affects the central nervous system: slowing responses, diminishing psychomotor skills, impairing cognition, and increasing sedation in a dose-dependent, biphasic fashion (Abrahao, Salinas & Lovinger 2017; Cromer et al. 2010). Numerous studies have described the effects of alcohol on driving performance, including impairments in division of attention, information processing, motor responses, complex reaction times, steering accuracy and maintenance of vehicle position in the lane (Breitmeier et al. 2007; Irwin et al. 2017; Laude & Fillmore 2015; Martin et al. 2013; Maylor et al. 1990, 1992; Rezaee-Zavareh et al. 2017). Crash risk has also been shown to increase with the level of a driver's blood alcohol concentration (BAC) (Martin et al. 2013; Phillips & Brewer 2011). The relative risk of a crash with increasing BAC follows a dose-response relationship beginning at BACs of 0.04-0.05 g/100ml and increasing exponentially at BACs of 0.10 and above (Blomberg et al. 2009). SA's BAC limit is 0.05g/100mL for fully licensed drivers and 0.00g/mL for drivers with a learner's permit or provisional licence. A recent South Australian study found that levels of alcohol involvement

in serious road crashes are trending downwards, which was attributed to declining levels of alcohol consumption among the young (Baldock & Lindsay 2020).

3.2.2 Illicit and prescription drugs

The detection of illicit substances in non-fatal and fatal crashes has been increasing over the past two decades (Baldock 2008; Baldock & Lindsay 2020; DiRago et al. 2021; Drummer, Olaf H & Yap 2016; O'Donovan et al. 2022; Schumann et al. 2021). While driving when impaired by drugs, illicit or prescription, is prohibited in Australia (*Australian Road Rules 1999*) only three illicit drugs: delta-9-tetrahydrocannabinol (THC), the active ingredient in various forms of cannabis, N-methyl-alpha-methylphenethylamine, or methamphetamine (MA) and 3,4-methylenedioxy-N-methamphetamine (MDMA) are the subject of roadside drug testing throughout Australia (cocaine is also tested for in New South Wales).

THC typically has a depressant effect on the central nervous system, affecting cognitive and psychomotor function including attention, concentration, coordination, reaction time, planning and decision making, and tracking, all of which impact driving ability (Asbridge, Hayden & Cartwright 2012; Baldock 2008; Bondallaz et al. 2016; Kurzthaler et al. 1999). The relative risk of a road crash associated with THC use alone, however, has only been found to be modestly elevated (Rogeberg & Elvik, 2016). THC is also frequently detected in combination with alcohol among crash-involved drivers (Baldock & Lindsay 2015; Gadegbeku, Amoros & Laumon 2011), a combination often found to be associated with a high relative crash risk (Hels et al. 2013).

Although THC was previously the most common drug detected in roadside drug testing, and in hospitalised crash-involved drivers and fatal crashes, MA detection has surpassed THC detection in both roadside testing and in hospitalised crash-involved motorists in the last decade, in SA and other Australian states (Bade et al. 2018; Baldock & Lindsay 2020; Mills et al. 2021; Schumann et al. 2021; Wundersitz & Konstad 2017). MA

increases risky driving behaviour (Bosanquet et al. 2013; Hayley et al. 2019) and significantly increases the odds of crash culpability, even without the presence of alcohol or other drugs (Drummer et al. 2020; Elvik 2013).

To determine if this upward trend in MA detection could also be identified in drivers involved in fatal MVCs, the following study was completed using autopsy reports collected as part of this thesis. The presence of MA and blood alcohol concentration (BAC) above the legal limit at autopsy was recorded between 2008 and 2018, and the trends for MA and alcohol detection were compared. Consistent with studies of hospitalised motorists, MA was increasingly detected in driver fatalities over the study period, although the apparent increase was not statistically significant. There was a significant decreasing trend in the detection of a BAC of greater than or equal to 0.05g/mL; however, this trend was not linear, suggesting that the trend may be plateauing or reversing. The full publication can be found at the end of this chapter (Chapter 3.5).

Other drugs can also be detected through forensic blood analysis following a crash. These include prescription medication such as opioids, benzodiazepines, anti-depressants, anti-psychotics, as well as illicit drugs not tested at the roadside in SA, such as cocaine. The crash risk and crash culpability of prescription drugs is related to the drug's capacity to affect the central nervous system (CNS), with the majority not associated with an increased crash risk or crash culpability (Drummer & Yap 2016; Engeland, Skurtveit & Mørland 2007). Benzodiazepines, however, have been shown to increase crash risk, particularly when combined with alcohol (Drummer & Yap 2016; Engeland, Skurtveit & Mørland 2007; Longo, Lokan & White 2001). While benzodiazepines are a prescription drug of concern, it remains unclear as to whether the effect of the CNS-depressant medications or the effect of the underlying disease for which the drug is administered increases the crash risk (Dischinger et al. 2011; Schumann et al. 2021). Given the complexity of separating the risks of

prescription drug effects from the risks associated with the medical conditions they treat, data collection for this thesis focused on alcohol and illicit drugs rather than prescription medication.

3.2.3 Other driver factors

There are other driver factors that contribute to fatal crashes including driver distraction and inattention, fatigue and speeding. Almost a third of fatal crashes in SA were attributed to driver inattention or distraction between 2014 and 2018 (Wundersitz 2019). Inattention can be defined as "insufficient, or no attention, to activities critical for safe driving" where a driver's attention may be restricted, misprioritised, neglected, cursory or diverted (Regan, Hallett & Gordon 2011, p. 1775; Regan & Strayer 2014). Distraction is considered a subtype of diverted inattention that may be visual, auditory, manual or cognitive in nature and that disrupts the cognitive and psychomotor process necessary for driving (Sundfør, Sagberg & Høye 2019). Vehicle technologies and personal technology such as smart watches and smart phones increase driver distraction, although Wundersitz et al. found that the majority of distractions were from cognitive states, such as emotional stress and internal thoughts (Beanland et al. 2013; Dingus et al. 2016; Wundersitz 2019). This is a major reason why crashes related to inattention and distraction are difficult to monitor and mitigate. The effect of alcohol is also amplified when the attention of the driver is divided between a driving and a non-driving task, reducing driving performance (Harrison & Fillmore 2011).

Fatigue remains one of the greatest challenges in road safety (Grigo 2011). Estimates suggest that sleepiness and fatigue contribute to 10-40% of crashes (Connor, Jennie et al. 2001; Fletcher et al. 2005; Grigo 2011; Moradi, Nazari & Rahmani 2019). Although drivers acknowledge the crash risk associated with driving while fatigued, many discount or underestimate the indictors of sleepiness (Armstrong et al. 2010; Fletcher et al. 2005). Other reasons such as closeness to home, acceptance of risk or pressure to arrive at the designated

location have been suggested to influence driver decisions to drive while fatigued (Armstrong et al. 2010; Fletcher et al. 2005). Drivers sometimes attempt to reduce fatigue or sleepiness by consuming caffeinated beverages or rolling down a window but there is little evidence to suggest that these strategies actually reduce sleepiness or lane drift (Armstrong et al. 2010; Reyner & Home 1998). Young drivers, shift workers, commercial drivers and those with a sleep disorder are at high risk of driving while sleepy as well as those who have worked long hours (Moradi, Nazari & Rahmani 2019). Fatigue can increase reaction times, affect cognitive processing, lead to lapses in concentration and affect decision making in response to road conditions or hazards (Grigo 2011). The effects of sleepiness on driving ability are comparable to that of alcohol intoxication (Grigo 2011). Additionally, alcohol in high doses can have a sedative effect, further exacerbating any existing driver fatigue. Sleepiness and fatigue remain difficult to identify in drivers involved in fatal crashes, as indicators of driver fatigue in the nature of the crash (I, running off the road) overlap with indicators of inattention, distraction, intoxication and deliberate collisions (Grigo 2011).

Speeding accounts for the majority of traffic violations and has been repeatedly demonstrated to increase crash risk and increase injury severity in the event of a crash (Cooper 1997; Doecke et al. 2020; Doecke et al. 2018; Kloeden et al. 1997; Moore, Dolinis & Woodward 1995; Walter & Studdert 2015; Williams, Kyrychenko & Retting 2006). A landmark study by Kloeden *et al.* found that, in a 60km/h speed limit area, the risk of involvement in a casualty crash doubles with each 5 km/h increase in travelling speed above 60km/h (Kloeden et al. 1997). It is likely that higher travelling speeds amplify multiple crash risk factors, such as increased reaction distance and braking distance, as well as increasing the risk of loss of vehicle control, thereby cumulatively increasing the risk of a casualty crash (Kloeden et al. 1997).

3.3 Medical Conditions

Pre-existing medical conditions and acute medical episodes have been found to be risk factors for road crashes (I, Lindsay & Ryan, 2011). Loss of consciousness and incapacitation of the driver due to a medical condition pose the greatest risk to crash involvement, as the driver may lose control of the vehicle. Various conditions associated with drivers losing consciousness include epilepsy or other seizure disorders, hypoglycaemia, sleep disorders, cardiovascular events such as abdominal aortic aneurysm (AAA) or myocardial infarction, and cerebrovascular diseases such as stroke (Drazkowski 2007; Ellen et al. 2006; Joshi et al. 2019; Krumholz 2009; Neal et al. 2018; Songer & Dorsey 2006; Tervo et al. 2008; Thiese et al. 2015; Youngquist et al. 2015). Other medical conditions related to cognitive function such as dementia, Parkinson's disease and other neurogenerative diseases can also negatively affect driving ability which may contribute to risk of crash involvement (Meuleners et al. 2016; Reger et al. 2004; Toups et al. 2022).

Cardiovascular disease, and specifically, ischaemic heart disease (IHD), is the leading cause of death in Australia (Australian Bureau of Statistics 2020). Furthermore, it is likely that the true incidence in the population is far greater than suggested by self-reported or medically diagnosed statistics. To investigate if severe coronary artery atherosclerosis (CAA) or cardiomegaly, as a marker of IHD, is playing a role in fatal crashes that has not been previously identified, a study was undertaken within the scope of this thesis using combined TARS and autopsy data. A subset of driver occupant fatalities aged 40 years or older from 2005 to 2017, with passengers selected as a control group, were analysed for the presence of severe CAA and cardiomegaly. This study also reported on crashes in which the cause of death was attributed to IHD or cardiomegaly. Severe CAA and cardiomegaly were not found to be higher in drivers compared to passengers and so it is unlikely that IHD is contributing to crashes to a degree greater than previously recognised. These findings also suggest that

trauma is not masking or obscuring the presence of IHD. However, the true incidence of IHD in the population remains unknown and so it was not possible to compare crash-related prevalence with any accurate comparison population data. Furthermore, the fatalities attributed to IHD, or other cardiac events only involved low impact severity collisions, suggesting that cardiac events precipitating a collision may exhibit early warning signs that alert the driver to slow down or pull over before losing consciousness. The full publication can be found at the end of this chapter (Chapter 3.6).

MVCs may be used as a method of suicide, with the occupant deliberately crashing to cause a fatal injury. Although deliberate MVCs are not included in the TARS database, they present an important and difficult challenge in road safety warranting further investigation within the scope of this thesis. An overview of the various methods of suicide that involve a motor vehicle was conducted, with cases extracted from FSSA. Deliberate MVCs are significantly underreported, with many being classified as accidental or an open finding. The rate of driver suicides in official reports is often below the rate reported in the literature, which ranges between 1.1% and 10% of vehicle crashes (Björnstig, Björnstig & Eriksson 2008; Henderson & Joseph 2012; Hernetkoski, Keskinen & Parkkari 2009; Ohberg, Penttila & Lonnqvist 1997; Pompili et al. 2012). The classification of an intentional vehicle crash is complex and complicated as a vehicle crash may be used to conceal the true intention. This presents a challenge in road safety to design road systems that can remain forgiving in circumstances in which the crash is intentional. A MVC may also be used as a method of murder-suicide as reported by Byard et al. (Byard et al. 2018). The full publication can be found at the end of this chapter (Chapter 3.7).

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3.5 Publication: Methamphetamine and alcohol detection in vehicle-driver fatalities in South Australia: A 10-year survey (2008-2018)

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The roadside detection of MA was not introduced until 2006 and, although MA may have been detected in toxicological samples in driver fatalities prior to 2006, this study time frame only includes driver fatalities after roadside drug testing was implemented. This study compares the trends in the detection of MA and a detected blood alcohol concentration above 0.05g/100mL in driver fatalities as a measure of safety countermeasure effectiveness.

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Certification:	This paper reports on original research I conducted during the period of my Higher Degree by Research candidature and is not subject to any obligations or contractual agreements with a third party that would constrain its inclusion in this thesis. I am the primary author of this paper.		
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By signing the Statement of Authorship, each author certifies that:

- the candidate's stated contribution to the publication is accurate (as detailed above);
- permission is granted for the candidate in include the publication in the thesis; and
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PAPER

Pathology/Biology

Methamphetamine and alcohol detection in vehicle-driver fatalities in South Australia: A 10-year survey (2008–2018)

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Abstract

Motor vehicle driver fatalities (≥18 years) from the files at Forensic Science South Australia were reviewed from January 2008 to December 2018 for cases in which either positive blood sample for methamphetamine (MA) or an illegal blood alcohol concentration (BAC) >0.05g/100 ml were found. Three hundred driver deaths were found with MA detected in 28 cases (age range 21–62 years; ave. 37.8 years; M:F 23:5). Hundred and fifteen cases with a BAC > 0.05 g/100 ml were identified (age range 18–67 years; ave 35.7 years; M:F 95:20). No change was found in numbers of MA cases, although alcohol cases showed a significant decline ($p < 0.001$). Drunk driving-related fatal crashes tended to occur in the evening (5 p.m. to 11 p.m.), while MA-related fatal crashes had a longer peak extending from late evening until late morning (11 p.m. to 8 a.m.). This study has demonstrated that while roadside breath testing, legislative changes, and increased monitoring have resulted in reduced levels of drunk driving, similar safety countermeasures have had negligible effects on MA use in drivers. Continued monitoring of MA use by drivers will, therefore, be necessary to assess the possible effects, or not, of new countermeasures.

KEYWORDS

Australia, autopsy, blood alcohol level, driver, forensic pathology, forensic toxicology, methamphetamine, road traffic accident

Highlights

- Between January 2008 and December 2018 in South Australia MA was found in 28/300 (9%) of driver deaths.
- Between January 2008 and December 2018 in South Australia blood alcohol >0.05 g/100 ml was found in 115/300 (38%) of driver deaths.
- Although numbers of alcohol positive cases declined significantly ($p < 0.001$), no change occurred in numbers of MA positive cases.
- While roadside breath testing, legislative changes, and increase monitoring have reduced drunk driving, this has not affected MA users.

1 | INTRODUCTION

Methamphetamine (MA), colloquially known as crystal meth, speed, or ice, is a highly addictive illegal drug that is a potent central nervous system psychostimulant that causes deficits in memory, executive functions, information processing, and motor skills (1). It also induces euphoria, aggression, and erratic behavior and has been found on toxicological testing at all ages (2–6). MA is associated with high dependency rates and its potential for abuse has been linked to overdoses, drug-related crime, and traumatic deaths including vehicle occupant fatalities (7–10). A study by González-Mariño et al. (11) analyzed wastewater samples for levels of amphetamine, 3,4-methylenedioxymethamphetamine (MDMA), and MA between 2011 and 2017. The study showed that Adelaide, South Australia had the highest levels of MA globally (507–659 mg/1000 people per day) with levels far above the next closest city, Seattle, United States (418 mg/1000 people per day). Cities were considered to have a high level of MA use if their averaged levels exceeded 150 mg/1000 people per day (11).

Driving under the influence of illicit drugs significantly increases the risk of motor vehicle collision and remains a major public health concern (12, 13). Baldock and Lindsay have shown an increase in recent years in illicit drug use among drivers and motorcycle operators admitted to hospital for testing in South Australia following crashes (12). This increasing trend has also been demonstrated in fatal collisions in Victoria, Australia (14). Primary prevention initiatives have involved the use of roadside drug testing of oral fluid for three illicit drugs specified in the South Australian *Road Traffic Act (1961)*: N-methyl-alpha-methylphenethylamine (MA), in addition to delta-9-tetrahydrocannabinol (THC—the psychoactive ingredient in forms of cannabis) and 3,4-methylenedioxy-N-MA (MDMA or ecstasy). The detection of MA on roadside testing has significantly increased within the past decade (15, 16), while there is evidence of declining rates of the detection of drunk-driving in random breath testing (RBT) operations (17). Given these changes in detection rates for impaired drivers, this study aimed to analyze the trends in MA concentrations in motor vehicle drivers involved in fatal collisions over the past decade and to compare this to the detection of blood alcohol concentrations (BAC) above the South Australian legal limit of 0.05 g/100 ml.

2 | MATERIALS AND METHODS

All fatal motor vehicle fatalities undergo major crash investigation, Coronial and full forensic investigation, including medicolegal autopsy and/or CT scan with full quantitative toxicological analysis. Coronial autopsy reports of all motor vehicle driver fatalities (≥18 years) at Forensic Science South Australia (FSSA) were reviewed over an 11-year period from January 2008 to December 2018 for cases in which MA and/or BAC above 0.05 g/100 ml were found.

These data were cross-matched with deidentified data from the Traffic Accident Reports System (the database for all police-reported crashes in South Australia) to provide further information about the crashes (e.g., the hour of collision). The decedents age, sex, cause of death, and toxicological results from postmortem blood sample analysis were also recorded. Antemortem sample taken shortly before death were used in cases where postmortem samples were not available. Cases where blood samples could not be obtained were excluded from the study.

The study period between 2008 and 2014, MA was screened and analyzed using an Applied Biosystems 4000Q-Trap with an Aglient 1200 LC system, involving liquid chromatography with tandem mass spectrometry detection (4). The screening method changed in 2009–2019 to involve an alkaline liquid-liquid extraction with analysis by liquid chromatography quadrole time-of-flight mass spectrometry. Chromatographic separation was conducted using an Aglient 1290 Infinity II liquid chromatography system (4). MA was detected through an Aglient 6545 QTOF system in conjunction with a Jetstream electrospray ionization (4). The detection limit remained 0.01 mg/L throughout the study period. BAC was measured by liquid-injection gas chromatography, performed by two Perkin-Elmer Autosystem gas chromatographs (Instruments A and B) (18). Samples are analyzed using both Instruments A and B with ethanol retention time at 0.9 and 0.6 min, respectively. Data were collected using TurboChrom Navigator software (18). The sample analysis is then repeated by a different analyst on a different day in line with road traffic toxicology procedure and the minimized potential instrument inferences and analyst errors.

Statistical analyses were performed using R 4.0.4; Pearson's chi-square was used to determine the relationship between sex and both MA and BAC detection/levels. Simple linear and polynomial regression was used to analyze the trend of the proportion of positive MA and BAC detections compared with negative cases over time. A Wilcoxon test was used to assess rates of detection of MA and BACs greater than 0.05 g/100 ml by age and hour of collision. All p values are two sided.

3 | RESULTS

3.1 | Methamphetamine

A total of 300 driver deaths due to motor vehicle collisions occurred between January 2008 and December 2018 with MA detected in 28 postmortem blood samples (Table 1). The age range of MA positive fatalities was between 21 and 62 years old (average 37.8 years) with 23 males and five females. A Pearson's chi-square test revealed no significant difference in the likelihood of MA detection by sex ($\chi^2 = 1.194$, $p = 0.275$). Multiple blunt force injuries were the primary cause of death in the majority of drivers (46.4%), followed by blunt head trauma with skull fracture (10.7%) and blunt chest trauma (10.7%). A simple regression was performed to determine the change in the number of driver fatalities detected with MA at autopsy. Although there appeared to be a small upward trend in MA-positive cases, it did not reach significance (Figure 1).

TABLE 1 Driver fatalities with positive blood sample for methamphetamine (MA) (mg/L) and other drug detected during toxicological analysis (n = 28)

Case number	Sample analyzed	MA concentration (mg/L)	Other drugs detected	Concentration of other drug
1	BLO[a]	0.34	NONE	
2	BLO	0.06	Carboxyhemoglobin	5%
3	BLO	0.15	Alcohol	0.157 g/L
			THC[c]	6 μg/L
4	BLO	0.13	MDMA[d]	1.3 mg/L
			MDA[e]	0.07 mg/L
5	BLO	9	NONE	
6	AM[b] BLO	0.14	Diazepam	0.4 mg/L
			Nordiazepam	0.1 mg/L
7	BLO (chest cavity)	0.16	Cocaine	0.03 mg/L
			Benzoylecgonine	0.8 gm/L
			THC	3 μg/L
			THC-COOH[f]	12 μg/L
8	BLO (cavity)	0.03	Ibuprofen	8 mg/L
9	BLO	0.26	Benzoylecgonine	0.03 mg/L
			7-aminoclonazepam	0.05 mg/L
			THC	3 μg/L
			THC-COOH	8μg/L
10	BLO	0.2	Alcohol	0.184 g/L
11	AM	0.14	Phenytoin	4 mg/L
			Lamotrigine	NA (low/therapeutic)
			THC	4 μg/L
			THC-COOH	12 μg/L
12	BLO	0.34	THC-COOH	8 μg/L
13	BLO	0.6	Alcohol	0.01 g/L
			Mirtazapine	0.02 mg/L
			THC	21 μg/L
			THC-COOH	49 μg/L
			Carboxyhemoglobin	54%
14	BLO	0.03	Morphine	0.21 mg/L
			Buprenorphine	0.9 mg/L
			Norbuprenorphine	3.3 mg/L
			Ketamine	0.8 mg/L
			Diazepam	0.2 mg/L
			Nordiazepam	0.2 mg/L
15	BLO	0.5	THC	24 μg/L
			THC-COOH	49 μg/L
16	BLO	0.02	Alcohol	0.101 g/L
17	BLO	1.4	O-desmethylvenlafaxine	0.6 mg/L
18	BLO	1.8	Tramadol	6.9 mg/L
			O-desmethyltramadol	1.1 mg/L
			THC	6 μg/L
			THC-COOH	39 μg/L
19	BLO	0.2	Diazepam	0.04 mg/L

(Continues)

TABLE 1 (Continued)

Case number	Sample analyzed	MA concentration (mg/L)	Other drugs detected	Concentration of other drug
			Alprazolam	0.01 mg/L
20	BLO	0.31	NONE	
21	BLO	0.57	Carboxyhemoglobin	7%
			Diazepam	0.59 mg/L
			Nordiazepam	0.22 mg/L
			Methadone	0.04 mg/L
			Cyanide	0.21 mg/L
			Morphine	0.02 mg/L
22	BLO	1.2	Alcohol	0.07 g/L
			Olanzapine	0.11 mg/L
			Paliperidone	0.011 mg/L
23	BLO	0.48	NONE	
24	BLO	0.012	Alcohol	0.13 g/L
25	BLO	2.4	THC	15 µg/L
			THC-COOH	40 µg/L
26	BLO	0.24	Alcohol	0.14 g/L
27	BLO	0.32	NONE	
28	BLO	0.03	NONE	

[a]Peripheral blood.
[b]Ante-mortem.
[c]Tetrahydrocannabinol.
[d]3,4-Methylenedioxymethamphetamine.
[e]3,4-Methylenedioxyamphetamine.
[f]11-nor-9-carboxy-Tetrahydrocannabinol.

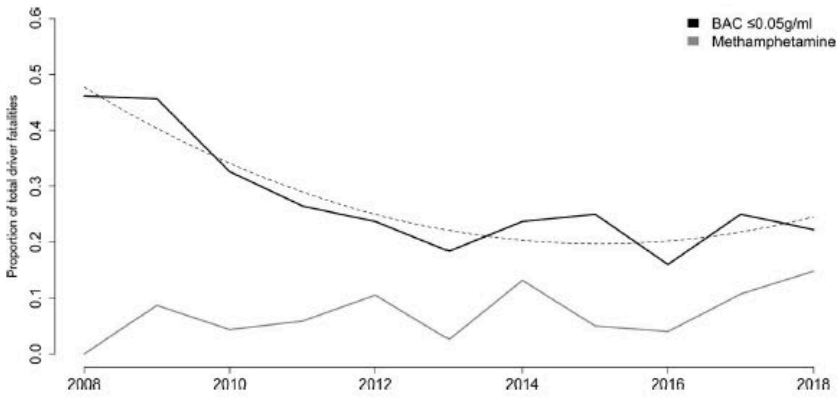


FIGURE 1 The proportion of driver fatalities with a BAC ≥ 0.05 g/100 ml compared with the detection of MA at postmortem by year (2008–2018). The dashed line represents the polynomial regression fit to BAC ≥ 0.05 g/100 ml

3.2 | Methamphetamine compared with BAC

A significant BAC above 0.05 g/100 ml was detected in 115 of the 300 decedents. The age range was 18 to 67 years old (average 35.7 years) with 95 male and 20 females. A Pearson's chi-square test showed a significant relationship between sex and illegal BACs ($\chi^2 = 9.522$, $p = 0.002$), with male drivers more likely to have illegal BAC. Multiple blunt force trauma was again the predominant cause of death in cases with an illegal BAC (45.2%), followed by blunt head trauma with skull fracture (19.1%) and blunt chest trauma (14.8%). A polynomial, second order regression identified a significant downward trend in the number of driver fatalities with illegal BACs over time ($p < 0.001$; Figure 1). As the trend was not linear, this suggests that the significant decrease may be plateauing or reversing, with the lowest level appearing around 2016. Figure 2 shows the proportion by sex and age of driver fatalities with MA and/or BAC greater than 0.05 g/100 ml detected at post mortem. Panels A and B describe the proportion in driver fatalities

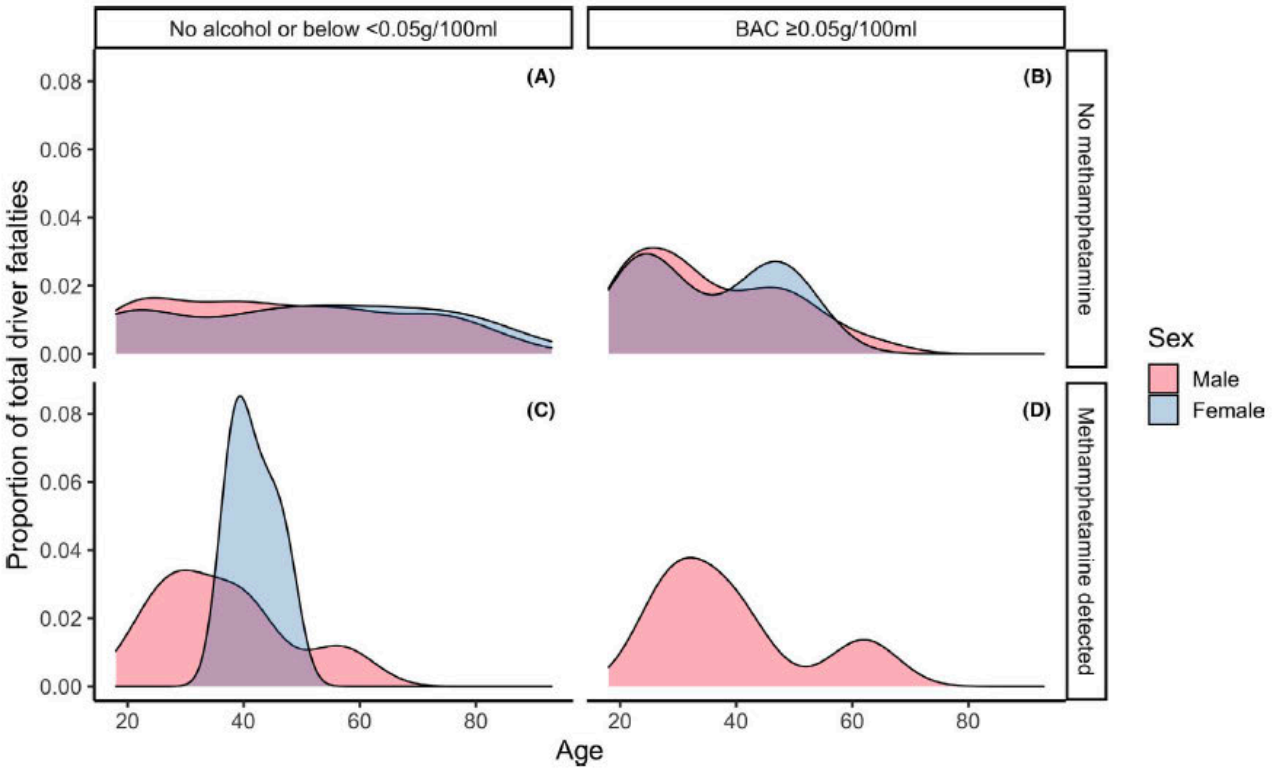
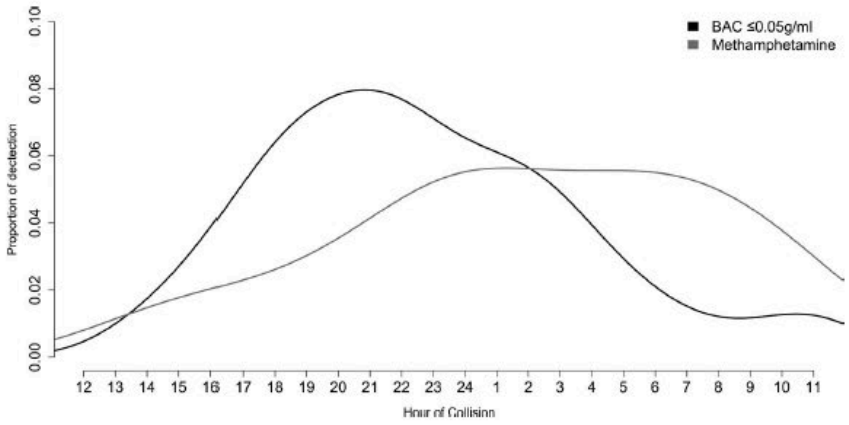


FIGURE 2 The proportion of methamphetamine (MA) and a BAC ≥0.05 g/100 ml detected at postmortem in driver fatalities by sex and age. Panel (A) shows decedents with a zero blood alcohol or level below 0.05 g/100 ml and no methamphetamine. Panel (B) shows decedents with BAC greater than 0.05 g/100 ml with no methamphetamine. Panel (C) shows decedents with positive MA detection and no alcohol or level detected below 0.05 g/100 ml. Panel (D) shows decedents with positive MA detection and BAC ≥0.05 g/100 ml

FIGURE 3 The proportion of driver fatalities with a BAC ≥ 0.05 g/100 ml compared with the detection of MA at postmortem by hour of crash



with no alcohol or BAC below 0.05 g/L (A) and driver fatalities with BAC above 0.05 g/L with no MA detected (B). Panel B shows a bimodal distribution of female driver fatalities with BAC above 0.05 g/L with no MA. Panels C and D describe the proportion of driver fatalities with MA detected. Panel C shows a marked increase in the proportion of female MA positive driver fatalities with no alcohol or BAC below 0.05 g/L. Panel D shows a bimodal distribution of male driver fatalities with both MA detected and BAC above 0.05 g/L.

A Wilcoxon test showed a significant difference in the proportion of MA detection compared with BACs above 0.05 g/100 ml by hour of fatal collision ($W = 1038.5$, $p < 0.01$). Figure 3 shows the proportion of driver fatalities positive for alcohol or MA starting at 12 p.m. in a 24-h timeframe. It appears that drunk-driving-related fatal crashes are more likely to occur in the evening (5 p.m. to 11 p.m.), while MA-related fatal crashes have a longer peak extending from late evening until early morning (11 p.m. to 8 a.m.).

4 | DISCUSSION

This study did not show a significant increase in the number of driver fatalities where MA was detected despite MA use being on the rise in South Australia shown by increases in waste water concentrations and drug-related deaths (7, 19, 20). As recent research by Baldock and Lindsay (12) has demonstrated an increasing proportion of MA being detected in nonfatal crashes resulting in hospitalization, it could be that the nonsignificant findings in this study are skewed by the relatively low numbers of fatalities. An increasing prevalence of MA in injured drivers and road fatalities has been shown in Victoria over a similar time period (14, 21).

There is a recognized association between increased risk taking, collision risk and MA (2, 22). Risk-taking behaviors can include nonrestraint compliance, speeding, and the performance of dangerous manoeuvrers (13, 23–25). For example, Stough et al. demonstrated an increase in erratic driving behavior and risk taking such as "car following and inappropriate braking" (26). However, determining the effect of MA on driving behavior and collision risk is difficult to quantify while still accounting for mediating factors including pattern of use (social vs habitual use), comedown effects, and combined use of other substances (22). Furthermore, significant ethical limitations, variation in the individual effects of MA and poor data quality remains a barrier for researcher (2, 12, 22).

Younger males are more likely to engage in risk-taking behavior while driving and are more likely to do so while intoxicated by drugs or alcohol (27–29). Interestingly, male positive MA fatalities were, on average older, being closer to 50 years of age compared with female MA positive decedents who were more often aged between 35 years and 45 years of age. This may correspond to the increase in age of individuals taking MA that has been detected at coronial autopsies in the state over the past 20 years (4). A significant difference in the crash circumstances associated with the use of MA and alcohol was that collisions with MA-positive drivers were more likely to occur in the early morning hours while drunken drivers were more likely to crash between early-mid evening with a peak around midnight.

It should be noted that determining the significance of MA in postmortem blood samples is problematic (1). Blood concentrations greater than 0.5 mg/L are typically seen in MA-related deaths but the effects of other drugs or pre-existing disorders can also contribute to death with levels as low as 0.05 mg/L (24). Pilgrim et al. analyzed 169 postmortem cases in which amphetamine-class drugs were detected, finding that MA concentrations ranged from 0.2 to 5.6 mg/L (30). All cases had a natural disease present and deaths were considered to be related to MA. Certainly, the increasing age of MA users has also been associated with an increasing incidence of ischemic heart disease in this group (4). Frequent use of MA can also lead to the development of tolerance, meaning that more of the drug is required to produce a desired effect (31). Therefore, blood concentrations alone within the living or the dead cannot determine what effect the stimulant drugs may have had on an individual (32).

The significant and predominantly downward trend in the number of driver fatalities with a BAC above 0.05 g/100 mL indicates that road safety countermeasures such as RBT, public education, and legislative deterrents have been effective in causing a shift in drunk-driving perceptions with reduced drunk-driving activity (33, 34). In addition, it may also reflect declining rates of alcohol consumption within Australian society, especially among the young (12).

The decrease in alcohol detection follows previous declines in South Australian alcohol-related fatalities and injury after the introduction RBT in October 1981 and the subsequent reduction in the legal limit from 0.08 to 0.05 g/L in July 1991 (34, 35). However, the curved nature of the trend suggests that this decline is starting to plateau, something which has been reflected Australia-wide (33). The sustained level of driver fatalities can be attributed to habitual high consumption drinkers with persistent behavioral patterns of alcohol use and driving, including alcohol dependence, and drunk-driving reoffending, with a significant proportion of these fatalities having quite high levels of blood alcohol detected (12, 36). Another useful countermeasure against drunk-driving recidivism is the alcohol interlock scheme (36), a vehicle-installed system that requires that the driver to provide an alcohol-free breath sample before the vehicle can be started. The earlier voluntary alcohol interlock program in South Australia, which commenced in 2001, was replaced in 2009 by a mandatory interlock program for serious drunk-driving offenders. Research has also shown that the social activity of drinking has moved from licensed venues to private homes and property, particularly in rural locations, and so the focus of RBTs and mobile testing may need to be on residential arterial roads (36). Similar strategies such as roadside drug testing, have been in place since 2008 to detect and reduce driving under the influence of drugs however recent increases hospital-admitted cases would suggest the current strategy is not effective (12).

Methamphetamine is a growing concern for road safety in South Australia with the increase in consumption of MA in South Australia and the apparent failure of current safety countermeasures (12). Specifically this study has shown that MA use in driver fatalities has not declined despite safety programs and legislation with increasing numbers of nonfatal crashes resulting in hospitalization involving the use of MA (12). Further analysis and continued monitoring of MA use by drivers will be necessary to assess whether new countermeasures such as rehabilitation programs are having the desired effect.

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ETHICAL APPROVAL

This research was approved by the University of Adelaide Low Risk Human Research Ethics Review Group (ethics approval: H-2018-191)

and FSSA Research and Development Committee (ethics approval: PATH 18-11).

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3.6 Publication: Cardiac disease and driver fatality

S. O'Donovan, M.A. Humphries, C. Van Den Heuvel, M.R.J. Baldock, R.W. Byard

The presence of coronary artery atherosclerosis (CAA) and cardiomegaly are markers of ischaemic heart disease (IHD) that can be detected at autopsy. The presence of these conditions increases the risk of a cardiac event which may contribute to a fatal MVC that was previously masked by trauma. However, the subsequent study did not find an increased incidence of CAA or cardiomegaly in driver fatalities when compared to passenger fatalities. This publication also identifies a subset of drivers who experienced a cardiac event and subsequently crashed the vehicle with minimal associated trauma.

Statement of Authorship

Title of Paper	Cardiac disease and driver fatality
Publication Status	☒ Published ☐ Accepted for Publication ☐ Unpublished and Unsubmitted work written in manuscript style ☐ Submitted for Publication
Publication Details	O'Donovan, S, Humphries, M, Van Den Heuvel, C, Baldock, MRJ & Byard, RW 2022, 'Cardiac disease and driver fatality', *Forensic Science, Medicine and Pathology*, advance online publication, DOI: 10.1007/s12024-022-00475-4.

Principal Author

Name of Principal Author (Candidate)	Siobhan O'Donovan		
Contribution to the Paper	Collected autopsy data, performed statistical analysis, wrote manuscript		
Overall percentage (%)	85%		
Certification:	This paper reports on original research I conducted during the period of my Higher Degree by Research candidature and is not subject to any obligations or contractual agreements with a third party that would constrain its inclusion in this thesis. I am the primary author of this paper.		
Signature		Date	27/05/2022

Co-Author Contributions

By signing the Statement of Authorship, each author certifies that:

- v. the candidate's stated contribution to the publication is accurate (as detailed above);
- v. permission is granted for the candidate in include the publication in the thesis; and
- v. the sum of all co-author contributions is equal to 100% less the candidate's stated contribution.

Name of Co-Author	Corinna Van Den Heuvel		
Contribution to the Paper	Primary supervisor of Siobhan, helped to evaluate and edit the manuscript		
Signature		Date	04/07/2022

Name of Co-Author	Matthew Baldock

Contribution to the Paper	Co-supervisor of Siobhan, provide access to Traffic Accident Reporting System through Centre for Automotive Safety Research, helped to evaluate and edit the manuscript		
Signature		Date	27/5/2022

Please cut and paste additional co-author panels here as required.

Name of Co-Author	Melissa Humphries		
Contribution to the Paper	Performed statistical analysis, assisted in data interpretation and visualisation, evaluation of manuscript		
Signature		Date	20/06/2022

Name of Co-Author	Roger Byard		
Contribution to the Paper	Co-supervisor of Siobhan, supervised collection of autopsy data, helped to evaluate and edit the manuscript, acted as corresponding author		
Signature		Date	08/07/2022

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ORIGINAL ARTICLE

Cardiac disease and driver fatality

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Abstract
To determine the role of cardiac disease in driver fatalities, a retrospective review of autopsy files at Forensic Science SA in Adelaide, Australia, was undertaken over a 13-year-period January 2005–December 2017 for individuals aged ≥ 40 years who had died while driving a motor vehicle. The incidence of significant coronary artery atherosclerosis (CAA) and cardiomegaly was evaluated with comparisons between drivers and a control group of passengers. Autopsy examinations were performed on 303 drivers and 72 passengers who died of trauma and on 63 drivers who died of a cardiac event while driving. The average age for drivers dying of trauma was 58.5 years (range 40–93 years) with 48 (15.8%) having CAA and 31 (10.2%) having cardiomegaly. This was not statistically different to passengers (aged 63.3 years; range 40–93 years; 20.8% having CAA; 11 (15.2%) cardiomegaly; ($p > 0.2$). Drivers with significant cardiac disease did not, therefore, have increased rates of death in crashes, although a distinct subgroup of drivers consisted of those who had died from cardiac events and not trauma, while driving. The latter may be increasing in number given the aging population.

Keywords Vehicle crash · Ischemic heart disease · Fatality · Forensic · Elder

Introduction

Acute medical episodes, with or without pre-existing conditions, occurring while driving, may markedly impair vehicle control. Recognized disorders include epilepsy and vascular conditions such as stroke [1, 2]. It has been proposed that the most important of these disorders is ischemic heart disease (IHD) [3]. In South Australia, medical events have been found to have directly caused 18.1% of motor vehicle collisions [2]. In response to the role of medical conditions in road crashes, certain conditions causing loss of consciousness require prior clearance or specific licensing provisions due to the increased risk of a crash [1, 4, 5]. These restrictions can be applied to individuals with physical impairments such as limited eye sight and following surgery such as coronary artery bypass grafting [6].

Although studies have shown an increased risk of non-fatal vehicle crashes correlating with cardiac disease [7], the possible role of cardiovascular disease, more specifically ischemic heart disease and cardiomegaly, has not been evaluated in detail in road fatalities. Thus, the following study was undertaken to determine if underlying cardiovascular disease may predispose drivers to involvement in fatal vehicle crashes. Given that an increased body mass index (BMI) has been associated with an increased risk of trauma in vehicle crashes and is associated with both ischemic heart disease and cardiomegaly [8, 9], this was also evaluated.

Materials and methods

Retrospective review of autopsy files at Forensic Science SA in Adelaide, Australia, was undertaken over a 13-year period between January 2005 and December 2017 for motor vehicle drivers aged ≥ 40 years who had died while driving a vehicle. All unexpected, unnatural, usual, or violent deaths are reported to the State Coroner and undergo either external autopsy with a CT scan or full autopsy [10]. Only cases with a full autopsy were included in this analysis. The deidentified

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autopsy files were reviewed, and the age, sex, BMI, and details of the cardiovascular status were recorded. Those whose deaths were due to either trauma or a cardiac event were extracted from the data. Passengers aged ≥ 40 years from the same autopsy database were used as a control group.

The presence of coronary artery atherosclerosis (CAA) was recorded as an indicator of ischemic heart disease. CAA was recorded when there was ≥ 75% stenosis of one or more of the major epicardial coronary arteries, with or without acute or chronic ischemic myocardial changes. Cardiomegaly was recorded when the heart weight was > 95% percentile for weight and sex [11]. Deaths were attributed to either trauma or natural diseases.

All statistical analyses were performed by using RStudio (Version 1.2.5001) [12]. Fisher's exact test was used to analyze the difference between the incidence of CAA and CM in drivers and passengers. This test was also used to analyze the difference in sex within the CAA and CM population in drivers. Odds ratios (OR) and confidence intervals (CI) were used to assess statistical significance at a p value less than 0.05. All p values are two-sided.

Table 1 Characteristics of 375 driver and passenger fatalities ≥ 40 years of age autopsied in South Australia (2005–2017) as a result of trauma from a motor vehicle collision

Characteristic	Driver, $N=303$	Passenger, $N=72$
Sex		
Male	218 (72%)	34 (47%)
Female	85 (28%)	38 (53%)
Age	58.5 (40–93)	63.3 (40–93)
Male	58.2 (40–89)	61.0 (40–87)
Female	59.4 (40–93)	65.32 (44–93)
BMI	28.9 (15.1–57.2)	28.2 (17.6–45.7)
Male	28.9 (17.4–48.8)	28.1 (17.6–38.4)
Female	28.8 (15.1–57.2)	28.3 (18.0–45.7)
Coronary artery atherosclerosis		
Absent	255 (84%)	57 (79%)
Present	48 (16%)	15 (21%)
Cardiomegaly		
Absent	270 (90%)	61 (85%)
Present	31 (10%)	11 (15%)

Results

Three hundred forty-seven driver fatalities due to vehicular trauma were recorded in the South Australian population for those aged 40 years and above from 2005 to 2017. Of the 347, 303 of the drivers underwent full medico-legal autopsies performed at FSSA (Table 1), and the remaining 44 cases were excluded from the study. The average age for drivers was 58.5 years (range 40–93 years) with 218 (72%) males having an average BMI of 28.9 (range 15.1–57.2). Of those traumatic fatalities, 48 (15.8%) had severe stenosis of one or more of the major epicardial coronary arteries and an average BMI of 28.4 (18.7–40). Cardiomegaly was noted in 31 cases (10.2%) with an average BMI of 28.9 (20.2–40). In two cases, no body weight was obtained due to incineration.

A total of 99 passenger fatalities (aged ≥ 40 years) with medico-legal autopsies performed on 72 were randomly selected from the same time period as controls. Of the 72 cases with autopsies performed, the average age was 63.3 years (range 40–93 years) with 34 (47%) males having an average BMI of 28.2 (range 17.6–45.7). CAA was found in 15 passengers (20.8%) with an average BMI of 28.08 (range 22.9–37.9), and cardiomegaly was found in 11 cases (15.2%), with an average BMI of 29.55 (range 21.9–37.9).

An additional 63 driver fatalities occurred over the study period due to a cardiac event while driving (Table 2). The average age of these drivers was 64.5 years (range 42–88 years) with 58 males (92.1%) having an average BMI of 30 (range 17–45). In 50 cases (79.4%), death was due to ischemic heart disease. The cause of death in seven cases was cardiomegaly, and four deaths were due to coronary artery thrombosis/myocardial infarction. Further two deaths were caused by hypertensive or valvular heart disease.

Coronary artery atherosclerosis (CAA) in traumatic MV fatalities

The number of driver fatalities due to trauma with CAA was compared to the number of passenger fatalities due to trauma

Table 2 Characteristics of 63 driver fatalities ≥ 40 years of age who died in vehicle crashes in South Australia (2005–2017) as a result of a cardiac event

Characteristic	Driver, $N=63$
Sex	
Male	58 (92%)
Female	5 (7.9%)
Age	65 (41–88)
Male	63.7 (41–88)
Female	73.8 (58–87)
BMI	30 (17–46)
Male	29.8 (17.0–45.8)
Female	33.1 (24.7–42.5)
Cause of death	
Ischemic heart disease	54 (85.7%)
Cardiomegaly	7 (11.1%)
Hypertension/valvular disease	2 (3.2%)

with CAA using Fisher's exact test. The results showed no significant difference between the numbers of drivers with CAA compared to passengers with CAA ($p=0.298$, OR 1.397, 95% CI 0.678–2.755).

Cardiomegaly in traumatic MV fatalities

The number of driver fatalities due to trauma where cardiomegaly was identified was compared to the number of passenger fatalities due to trauma with cardiomegaly, again using Fisher's exact test. There was also no significant difference in the incidence of cardiomegaly between drivers and passengers ($p=0.220$, OR 1.569, 95% CI 0.673–3.427). No significant difference in BMIs among the groups was found ($p>0.05$).

Discussion

Ischemic heart disease (IHD) results from critical narrowing of coronary arteries usually by atherosclerotic plaque (atheroma). Critical luminal stenosis is defined as 75% or greater stenosis of one or more of the major coronary arteries [13]. Significant stenoses may be clinically occult, and vessel occlusion by thromboembolism or plaque disruption can be an unpredictable event. Cardiovascular disease also included cardiomegaly in this study as a feature of various primary cardiomyopathies or a secondary response to hypertension, valvular disease, or obesity. Due to the occult nature of many cases, the true incidence of IHD and cardiomegaly within the Australian population remains unclear.

Age is strongly correlated with an increased risk of preexisting disease and acute medical episodes [14]. Older drivers have, however, a relatively low crash risk compared to drivers in younger age groups, which is attributed to less risk taking and to reduced amounts of driving [15]. Despite decreased exposure to vehicle-related trauma, the combination of a lower physiological reserve and the presence of established conditions does, however, reduce the capacity of older drivers to respond to trauma. This results in a higher fatality risk in the event of a crash [16].

Despite assertions that ischemic heart disease is the "most important disease" affecting driver ability and awareness," [3] cardiac events while driving appear uncommon, although there is an association with nonfatal crashes [7]. It may be that clinical symptoms alert drivers to an impending episode allowing them time to exit the roadway and bring their vehicle to a halt. However, certain cardiac events such as arrhythmias may incapacitate very quickly and so may not provide a sufficient lucid window to enable a driver to stop [17]. It has, however, been difficult to determine the relationship between IHD and motor vehicle collisions [13, 18]. Partly due to the possibility that major lethal trauma sustained in a crash may obscure the presence of underlying cardiac disease that may have initiated the event; that is, at autopsy, death may be attributed to blunt head or chest trauma despite a cardiac event having already caused death or serious incapacitation.

The current study has shown, however, no significant differences in the incidence of CAA as a marker of IHD or cardiomegaly between drivers and passengers as would be expected if cardiac disease was initiating crashes. Thus, it appears that trauma is not masking contributory cardiac illness. The trend for passengers to actually have a greater likelihood of CAA could be due to an older age of passengers, combined with possible poor health precluding driving [19].

In this study, a smaller number of cardiac deaths did occur while driving with a slightly higher average age (64.5 years) than that of drivers dying of trauma (58.5 years). This may be more of a problem in the future with Australia's aging population (> 65 years) projected to rise from 15% of the total population in 2017 to 20% by 2047, with 55% of those aged over 75 years [20]. This increase in numbers of the aged [14] with sprawling urban growth may also necessitate greater driving by older adults in the future.

In Australia the assessment of diminished driving capability is most often performed by general practitioners/family physicians who can refer individuals to specialists or specialized occupational therapists for further evaluation [21]. Sims et al. found that general practitioners more likely to undertake evaluations of fitness to drive when prompted by a specific road safety medical form, rather than during a routine health check or when concerns were specifically raised by the patient [21]. However, Langford et al. showed that population-wide age-based mandatory medical and driver safety assessments were insensitive and ineffective in determining fitness to drive [22, 23]. The Austroads publication, "Assessing Fitness to Drive," provides guidelines to healthcare professionals but is limited in coverage of the possible risks of IHD and cardiomegaly [6]. It is recommended that patients who have undergone valve replacement and coronary artery bypass grafting should not drive for up to 2 months following surgery, but routine screening for IHD risk factors is not required for licensing purposes [6].

A limitation of the study is that the presence of possible cardiac channelopathies was not assessed; however, the prime focus was on IHD and cardiomegaly. There were several other factors that may have contributed to cardiovascular function and driving ability such as alcohol and other substances that were not assessed in this study.

Although cardiac disease may cause rapid incapacitation, this study has not shown a higher rate of cardiac disease in drivers in fatal vehicle crashes compared to passengers. A concern that initiated this study was that subsequent trauma following vehicle impact may skew the determination of the cause of death away from heart disease, but this does not appear to be the case. A significant percentage of drivers

who have died of heart disease behind the wheel of a vehicle have, however, sustained either no or minimal trauma. Given the aging population, it may be that these types of deaths will become more common in the future.

Key points

1. The incidence of ischemic heart disease and cardiomegaly was evaluated in drivers and a control group of passengers.
2. The average age for drivers dying of trauma was 58.5 years (range 40–93 years) with 48 (15.8%) having IHD and 31 (10.2%) having cardiomegaly.
3. This was not statistically different to passengers aged 63.3 years; range 40–93 years; 20.8% having IHD; 11; 15.2% cardiomegaly ($p>0.2$).
4. Drivers with significant cardiac disease did not have increased rates of death in crashes.

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3.7 Publication: An overview of suicides related to motor vehicles

S. O'Donovan, C. Van Den Heuvel, M.R.J. Baldock, R.W. Byard

Suicides are an under-reported feature of fatal MVCs and are not included in the TARS database. However, vehicle-related suicides can be identified in coronial autopsy reports. A short literature review and case series was presented discussing the various suicide methods and complexities of assessing and determining vehicle-related suicides. This publication has been accepted by the journal, Medicine, Science and the Law and is in-press.

Statement of Authorship

Title of Paper	An overview of suicides related to motor vehicles
Publication Status	☒ Published ☐ Accepted for Publication ☐ Unpublished and Unsubmitted work written in manuscript style ☐ Submitted for Publication
Publication Details	O'Donovan, S, Van Den Heuvel, C, Baldock, MRJ & Byard, RW 2022, 'An overview of suicides related to motor vehicles', *Medicine, Science and the Law*, advance online publication, DOI: 10.1177/00258024221122187.

Principal Author

Name of Principal Author (Candidate)	Siobhan O'Donovan		
Contribution to the Paper	Performed literature review, wrote manuscript, acted as corresponding author		
Overall percentage (%)	85%		
Certification:	This paper reports on original research I conducted during the period of my Higher Degree by Research candidature and is not subject to any obligations or contractual agreements with a third party that would constrain its inclusion in this thesis. I am the primary author of this paper.		
Signature		Date	27/05/2022

Co-Author Contributions

By signing the Statement of Authorship, each author certifies that:

- the candidate's stated contribution to the publication is accurate (as detailed above);
- permission is granted for the candidate in include the publication in the thesis; and
- the sum of all co-author contributions is equal to 100% less the candidate's stated contribution.

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Review article

An overview of suicides related to motor vehicles

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Abstract

Although vehicles may be used in a wide variety of suicides, this has not been a focus in the forensic literature. Thus, an analysis of coronial autopsy reports at Forensic Science SA, Adelaide, South Australia over a 16-year period from January 2005 to December 2020 was undertaken to provide an overview of cases in which it was considered that a vehicle had been integral to the successful completion of a suicide. De-identified details were collected from all cases in which a vehicle had facilitated or been used as a method of suicide. A number of different types of vehicle-related suicide methods were identified, including cases where vehicles had been used as secure places for suicide or where the vehicle had been used to cause significant blunt force trauma or to enter a lethal environment such as water. Specific examples were taken from the following categories: (1) inhalation of gas, (2) drug toxicity, (3) hanging or ligature strangulation, (4) self-immolation, (5) drowning, (6) vehicle collision, (7) driving off a cliff, (8) jumping or lying in front of a vehicle and (9) the use of more than one method (i.e. complex). This report is not intended to provide an epidemiological analysis of car-related suicides. Instead, the details of selected cases have been used to illustrate the spectrum of methods that may be used in vehicle-assisted suicides.

Keywords

Suicide, motor vehicle, carbon monoxide, hanging, self-immolation, blunt force trauma, drowning, forensic pathology

Introduction

The most common methods of suicide vary depending on the location and/or community. For example, in the United States (US) firearms are the primary device utilized compared to Australia where hanging is the most popular method, or China where pesticides are often taken.[1] Carbon monoxide (CO) toxicity from vehicle exhaust is not an uncommon method used in Australia; however, vehicles may be used in a variety of other ways for self-termination. Given that the use of vehicles in suicides has not been a focus in the forensic literature the following study was undertaken.

Materials and methods

An analysis of coronial autopsy reports at Forensic Science SA, Adelaide, South Australia over a 16-year period from January 2005 to December 2020 was undertaken for cases in which it was considered that a vehicle had been integral to the successful completion of a suicide. Cases were classified as suicides after full police, coronial and forensic investigations had been completed. De-identified details were collected from all cases in which a vehicle had facilitated or been used as a method of suicide. Toxicological analyses had been performed on peripheral blood samples unless otherwise stated. Details were extracted from representative cases in each category of death to provide an overview of these types of fatalities.

Results

A number of different types of vehicle-related suicide methods were identified, including cases in which there had been (1) inhalation of gas, (2) drug toxicity,

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(3) hanging or ligature strangulation, (4) self-immolation, (5) drowning, (6) vehicle collision, (7) driving off a cliff, (8) jumping or lying in front of a vehicle and (9) the use of more than one method (i.e. complex). Case details are provided in Table 1.

Discussion

Vehicles may play a passive role in suicide, being used as a location for death as in hanging or self-immolation, or more actively in deliberate crashes or in pedestrian suicides. An individual's choice of suicide method is influenced by factors such as accessibility, perception of pain and likelihood of success and chance of intervention/prevention.[2,3]

The vehicle as a place of suicide

The vehicle itself can be used as a place of suicide as it may provide a secure, enclosed environment that can be driven to a site where discovery and resuscitation will be unlikely. It may also provide the means of suicide in the form of CO gas in exhaust emissions.

Inhalation of gas. The most common gas used in vehicle-related suicides is CO, a by-product of incomplete combustion of hydrocarbons in low oxygen environments that can be produced by car exhaust, petrol generators or gas heaters (as was seen in Cases 1–5). The colourless, tasteless and odourless nature of CO gas provides a painless and non-violent method of self-termination.[3–6] CO is an oxygen antagonist, readily binding to haemoglobin to form carboxyhaemoglobin (COHb) preventing oxygen exchange with tissues.[6–8]

Suicide by CO toxicity requires some level of premeditation and planning as occurred in Case 2 where the purchase of materials occurred the day before death with connection of a pipe to feed exhaust fumes into the vehicle cabin.[9] CO does not need to be directly piped into the cabin as the vehicle may be parked in an enclosed, confined space such as garage and allowed to run (as occurred in Case 1).

The introduction of catalytic converters saw a marked decline in CO toxicity deaths from vehicle exhausts[3,10–13] but these deaths have not been eliminated as average life of a catalytic converter is only 10 years.[14] External sources of CO such as petrol generators (Case 5) and coal barbeques (Cases 3 and 4) may also be used to generate CO inside a vehicle. The use of coal fires to generate CO has recently become a more common method in countries such as Hong Kong and Taiwan.[3,11,15–17] The use of other gases for suicide has been on the rise with reported increases in cases utilizing hydrogen sulphide (H_2S),[16] nitrogen (N_2), propane, natural gas[18] and helium (He).[1,3,15,16,19] In case 6 H_2S gas was generated by mixing hydrochloric acid and lime sulphur, chemicals found in common household products.[20]

Drug toxicity. Death by taking illicit or prescribed drugs is the second most common method of suicide in Australia.[21] As mentioned above, vehicles may provide a secure and isolated space for this act. Cases 7 and 8 illustrate a wife and husband suicide pact with the vehicle being found in an isolated rural location with suicide notes.

Hanging/ligature. Reported cases of hanging within a vehicle have involved an external ligature or the vehicle's seat belt, with victims being most often middle-aged males. To facilitate hanging, the ligature or seat belt may be fastened/tightened around other vehicle structures such as handles,[22,23] seats[24] or other fixtures.[25,26] Case 9 describes the use of a leather belt threaded and secured through the vehicle window by closing the window onto the belt. Pampin and Rodriguez described an almost identical case involving a 23-year-old male using a leather belt.[24] Cases of hanging in a vehicle are rare, likely due to restricted space within the vehicle cabin.[26]

Vehicle-facilitated ligature strangulation occurs when a ligature is tied to a fixed object, usually a tree, with the other end around the individual's neck and then the vehicle is driven away at a high speed.[27] Several cases of decapitation as a result of vehicle-facilitated ligature strangulation have been reported.[27–29] All were middle-aged males with the oldest being 59 years.

Case 10 describes a male found in driver's seat with a rope still fastened around his neck but without decapitation. A rear wheel had continued to spin, sparking a fire that had alerted bystanders. Barranco et al. reported a case with one end of the rope tied to a roadside post and the other end around the neck. The parking brake had been disengaged allowing the vehicle to roll downhill, gradually tightening the ligature.[22]

Self-immolation. Self-immolation is a rare form of suicide in most Western nations.[30–34] While the purpose of self-immolation may be a form of political statement or protest performed in a public arena in some countries, this does not generally apply to vehicle-related self-immolations.[31,33,35,36] Gauthier et al. showed only 1% of suicides were the result of self-immolation in Switzerland; five of which (10%) were performed in a vehicle.[33] The majority were middle-aged males with histories of previous suicide attempts and mental health disorders.[33] Death in self-immolation may involve incineration, CO toxicity and/or cyanide toxicity due to the by-products of combustion or a combination of mechanisms as in Case 11.

Table 1. Characteristics of various suicide methods involving a motor vehicle.

Case number	Age	Sex	Methodology	Cause of death	Previous history	Communication	Notes/Additional information
1	52	F	CO – exhaust in garage	CO toxicity	No	Indicated to relative in several messages that she had run out of options	Found in car with ignition on, in garage with sign 'Do not enter, call police'. A suicide note was also present.
2	64	M	CO – exhaust fed into vehicle cabin	CO toxicity	No	No	Found in vehicle located in an isolated rural location. A length of pipe had been attached to the vehicle exhaust with masking tape and run through a rear window. The ignition was on.
3	23	M	CO – heat beads in vehicle cabin	CO toxicity	No	No	Found in the driver's seat with two trays of heat beads.
4	41	M	CO – rotisserie barbeque in vehicle cabin	CO toxicity	History of suicide attempts	No	Found in driver's seat with rotisserie barbeque that had been burning.
5	85	M	CO – petrol generator fed into vehicle cabin	CO toxicity	No	Suicide note	Found in vehicle with a pipe running from a petrol generator through a passenger window. Suicide notes were located.
6	39	M	Hydrogen sulphide gas	H_2S inhalation	History of mental illness, detained under mental health act (3 months prior)	No	Found in a vehicle with a bucket containing hydrochloric acid and lime sulphur.
7	56	F	Overdose in vehicle cabin	Amitriptyline toxicity	No	Suicide note	Found in a vehicle with suicide notes and prescription medication.
8	62	M	Overdose in vehicle cabin	Amitriptyline toxicity	No	Suicide note	Found in a vehicle with suicide notes and prescription medication.
9	39	F	Hanging – leather belt wedged through driver's side window and tightened	Hanging	Mental health issues	Text messages	Found in a vehicle with a belt wrapped around the neck wedged through the driver's side window.
10	49	M	Ligature strangulation – ligature tied to tree and vehicle driven	Neck compression by ligature	Episodic depression and suicidal ideation	No	Found in a vehicle with a ligature around the neck. The vehicle rear tyres were spinning causing a fire.

(continued)

Table 1. Continued.

Case number	Age	Sex	Methodology	Cause of death	Previous history	Communication	Notes/Additional information
11	59	M	Incineration in a vehicle	Inhalation of products due to combustion and effects of heat	No	No	Found in a burnt out vehicle. Two 20 L fuel cans were in the vehicle.
12	28	M	Drowning in vehicle	Drowning	Possible previous psychotic episode	No	Found in a vehicle having driven off a boat ramp.
13	46	F	Vehicle collision	Multiple blunt force injuries	No	Text message indicating intent	Found in a burnt out vehicle having collided with two large trees. No soot was present the in airways and COHb saturation was 7%.
14	40	F	Vehicle collision	Multiple blunt force injuries	No	No	Found in a vehicle with a self-inflicted incised wound to the neck, four incised wounds to the forearm and three stab wounds to the breast
15	23	M	Vehicle collision	Multiple blunt force injuries	No	Phone calls indicating intent	Found in a vehicle following a head-on collision with a tree.
16	21	M	Vehicle collision	Multiple blunt force injuries	Depression and attempted suicide by overdose	Conversations indicating intent	Head-on collision with a prime mover causing the vehicle to catch fire, No soot was found in airways
17	36	F	Vehicle collision	Crush asphyxia	Mental health issues	No	While attempting to drive off a cliff the vehicle rolled after hitting a large rock causing the occupant to be partially ejected through the driver's side window and trapped underneath.
18	19	M	Pedestrian collision	Blunt force head injury	Depression and previous suicide attempts	Conversations indicating intent	Witnessed to jump directly in front of an oncoming vehicle
19	57	M	Pedestrian collision	Multiple blunt force injuries	Mental health issues, suicidal ideation, with unsuccessful suicide attempts in the weeks prior (detained under mental health act)	Suicide note in pocket reading 'Truck Suicide'	Witnessed to step in front of a truck
20	33	M	Complex – shooting in vehicle and	Gunshot wound to the head	No	No	Driven off a wharf into water following a gunshot wound to the head

(continued)

Table 1. Continued.

Case number	Age	Sex	Methodology	Cause of death	Previous history	Communication	Notes/Additional information
			driving into water				
21	46	M	Complex – driving off cliff into water	Drowning and multiple blunt force injuries	No	No	Driven off a cliff into water

CO: carbon monoxide; COHb; carboxyhaemoglobin; H_2S: hydrogen sulphide.

Vehicle-facilitated

Vehicle-facilitated suicide refers to cases where a vehicle may be used as a mechanical aid to cause or enhance a particular type of injury.

Drowning. Vehicle-facilitated drowning involves driving a vehicle into a body of water. It is unclear in Case 12 if the driver's window has been down prior to the vehicle entering the water; however, this may have increased the rate of sinking. This type of vehicle-related suicide is uncommon, with an incidence of between 2% and3.3% of all drowning suicides.[37,38] In Case 12, a boat ramp had provided access to the water.

Use of a vehicle to cause lethal injuries

The use of a vehicle to inflict lethal injuries either as a driver deliberately crashing a vehicle or as a pedestrian purposely placing themselves in front of a vehicle may be underreported or unrecognized, in part because of the potential for concealing intent.[2,39–41]

Driver suicide. Rates of driver suicide range between 1.1% and 10% of all vehicle collisions.[42–46] A number of factors and considerations may indicate suicide, which includes a single occupant,[47,48] a high speed before impact,[47–50] no use of restraints,[50] a lack of braking pattern (absence of skid marks and no impression on the brake pedal),[49,50] steering pattern,[48,50] location (i.e. straight road)[50] and good weather conditions.[46,47,51] While some studies[43,46,47] suggests alcohol intoxication as an indicator of deliberate crashes, either acute intoxication or as part of a substance use disorder, Connolly et al. found a low percentage of deliberate cases involved alcohol use.[50] The contradiction in the literature may be due to some studies classifying negligent fatal crashes as deliberate. Negligent crashes are an intermediary subtype between deliberate and unintentional crashes. Hernetkoski et al. outline the overlapping features of deliberate and negligent cases, namely the predominance of young male drivers with the majority of negligent crashes involving intoxicated drivers compared to only 22% of deliberate crashes.[51] Suicide may be more obvious when a note or verbal communication of intent has been found or made.[47] Cases 13 through to 16 describe four cases of driver suicide with communication of suicide intent or with evidence of self-harm.

Driver suicides involve predominantly males,[2,44,46,47,52] aged 25–44 years[2,44,46,53] and are more likely to involve a driver who had experienced a life stressor prior to the collision (e.g. relationship conflicts, legal and/or financial problems).[2,41,46–48] Other factors include mental health issues and histories of suicidal ideation with previous attempts,[2,46,48,52,53] poor impulse control[2,41,47,52] and risk taking and reckless driving behaviour.[47,54–57] Unlike other forms of vehicle suicides where planning is required, vehicle collisions may occur on impulse.

Suicides involving another vehicle, in particular heavy vehicles, are not uncommon,[2,44,46,47,58] and presumably occur in an attempt to maximize the lethality of the impact. While death is often due to multiple injuries, unusual cases, as in Case 17, may occur where other mechanisms such as crush asphyxia occur. On occasion, others may be injured or killed in such suicides. Pridmore et al. reported a case of a 36-year-old male who drove into an oncoming vehicle with a family inside, killing the driver and a child passenger, and injuring a female passenger.[59] Murder suicides can also occur involving occupants within the offending vehicle, with a case reported by Byard et al. of a familial murder suicide.[60] On occasion a vehicle may be driven off a cliff or an elevated area to cause death from impact with the ground as in Case 21.

Pedestrian suicide. Increasingly, pedestrian suicides involve heavy vehicles and are recognized as an occupational hazard for drivers.[61,62] Determining intent in pedestrian crashes is often unsuccessful unless prior communication has occurred. Cases 18 and 19 are examples of communication prior to the crash, and by a suicide note found on the person reading 'truck suicide' following the crash. In both cases, the truck drivers attempted to avoid collision. Eye witness accounts, road location traffic conditions as well as dash-cam footage can all aid in ascertaining the cause and circumstances of the collision.

Clarifying the manner of death in pedestrian suicide may not, however, be possible, particularly in cases of intoxication.[63,64] Intoxicated pedestrians have also been hit by vehicles while they have been lying or sleeping on a road surface. While a number of these cases may be unintentional there must also be a certain percentage that is suicides.

Complex suicide

Complex suicides involve the combination of two or more methods and may evolve as a planned process with fail-safe measures to ensure lethal injury, or as an unplanned event when a second method has to be used due to failure of the primary method.[65–68] Cases 20 and 21 involve complex suicides with a vehicle either as the primary (Case 21), or concurrent method (Case 20). Both Cases 20 and 21 use drowning as a secondary method, with evidence of salt water immersion in both. Murphy and Straka reported three cases of males with gunshot wounds to the head while driving, resulting in collisions of the vehicles.[52,66] Christin reported a 27-year-old male who had been driving erratically prior to a collision who was found with a knife in his chest.[69] Suicide was determined as he had made verbal communication of his intent.[69] In a review of complex suicides in Milan, Italy, Gentile et al. reported a single complex suicide by vehicle crash followed by drowning.[70] Other complex suicides include an overdose followed by CO toxicity[71] and an attempted self-immolation followed by vehicle collision.[34] A case series by Byard and James also reported an overdose combined with vehicle collision whereby the accelerator had been intentionally depressed using a broken broom handle to maintain vehicle movement post drug administration.[26]

In conclusion, this study has shown that vehicles may be used in a wide variety of ways to assist with, or cause, self-termination. The secure, sealed and isolated nature of a vehicle cabin may provide an ideal location to ensure privacy and reduce the risk of discovery before death has occurred. Deaths may result from a wide variety of mechanisms including gas inhalation, drug ingestion or self-immolation. Speed and mass may ensure death from significant blunt force trauma if a vehicle is driven into an object or if it impacts a pedestrian. The mobile nature of vehicles also enables them to be utilized in immersion deaths or in falls from heights.

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Ethical approval

This research was approved by the University of Adelaide Low Risk Human Research Ethics Review Group (ethics approval: H-2018-191) and Forensic Science South Australia Research and Development Committee (ethics approval: PATH 18-11).

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Chapter Four: The interaction between age and sex

4.1 Introduction

Age and sex are two important intrinsic occupant factors that affect the risk of crash involvement and risk of sustaining fatal injuries in a crash. Younger age adults are at higher risk of fatal crash involvement (Braitman, Chaudhary & McCartt 2014; Doherty, Andrey & MacGregor 1998; Ouimet et al. 2015; Rice, Peek-Asa & Kraus 2003; Shope & Bingham 2008; Walter & Studdert 2015; Williams 2003), whereas increasing age reduces the risk of crash involvement while increasing susceptibility to fatal injury in the event of a crash (Cicchino & McCartt 2014; Hanrahan et al. 2009; Rakotonirainy et al. 2012). Crash and injury severity trends also differ by sex. Males are more likely to be involved in a crash and therefore have a higher rate of fatalities while female occupants may be more vulnerable to injury (Al-Balbissi 2003; Amarasingha & Dissanayake 2014; Cullen et al. 2021). Sex and age, especially the extremes of age, interact to form distinct categories of occupants at a higher risk of involvement in a fatal crash; these are young male drivers and older female occupants. The purpose of this chapter is to outline the complexity of age and sex as individual, intrinsic factors and the interaction between them that contribute to occupant fatalities and patterns of injury. This section demonstrates these trends within the study data using the TARS sample from Chapter One.

4.2 The effect of age

Young drivers have the highest risk of MVCs across all levels of severity and are over-represented in fatal crashes (Bureau of Infrastructure and Transport Research Economics 2022; Shope & Bingham 2008; Williams 2003). Figure 4.1 describes the number of driver crashes by age and crash severity using TARS data between 1981 and 2020. Figure 4.1 clearly shows that drivers (aged 18-25 years) were involved in more crashes than drivers in any other age range.

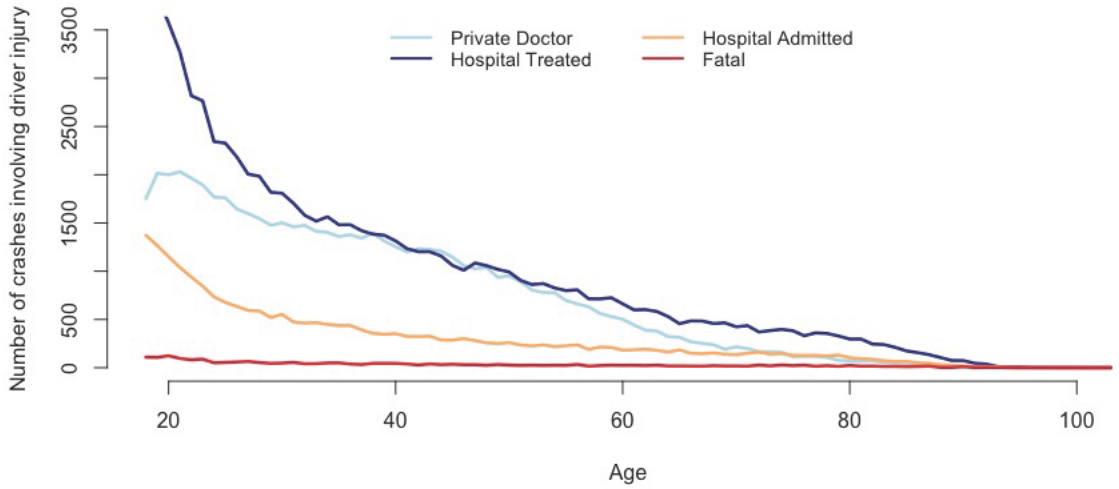

Figure 4.1 Number of crash-involved drivers by age and crash severity (1981-2020)

The increased risk of crash involvement for younger drivers is attributable to several factors including inexperience (McCartt et al. 2009; Shope & Bingham 2008) and higher likelihood of engaging in risky driving behaviours that also increase crash risk such as speeding (Simons-Morton et al. 2012) and driving while intoxicated (Shope & Bingham 2008; Williams 2003). An interesting feature of young driver crashes is that the presence of peer passengers in the vehicle with younger drivers has been found to increase fatal crash risk, a trend not seen across other age groups (Braitman, Chaudhary & McCartt 2014; Doherty, Andrey & MacGregor 1998; Ouimet et al. 2015; Rice, Peek-Asa & Kraus 2003; Walter & Studdert 2015; Williams 2003). The presence of younger passengers can increase younger driver distraction and inattention as well as promote more risky driving behaviour (Rice, Peek-Asa & Kraus 2003). This trend is reflected in Figure 4.2, also based on the TARS database, that shows the number of crash-involved passengers by age and crash severity between 1981 and 2020.

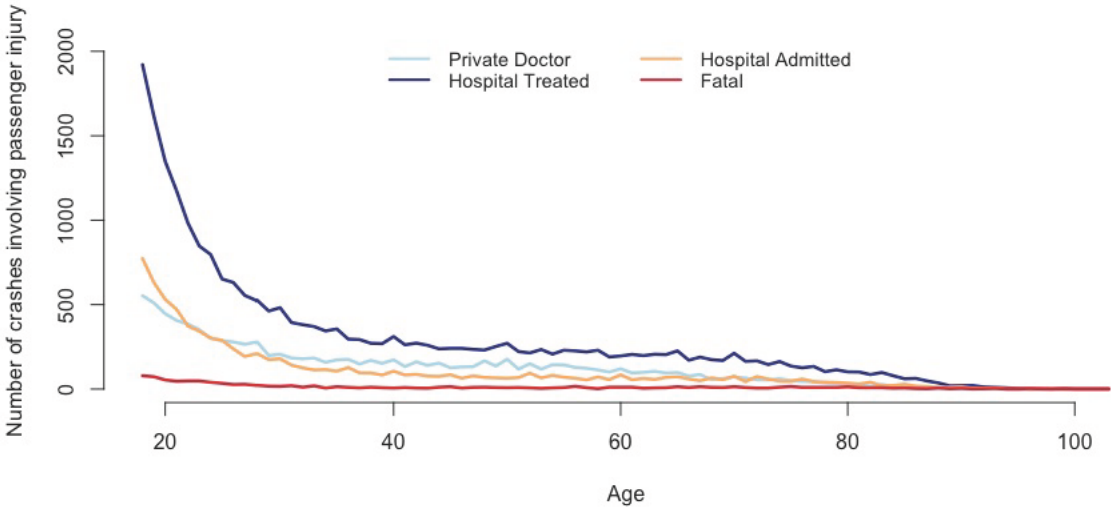

Figure 4.2 Number of crash-involved passengers by age and crash severity (1981-2020)

Figure 4.1 also demonstrates a gradual decline in driver crash involvement as age increases. However, the overwhelming representation of young drivers involved in all crash severities in Figure 4.1 masks the relationship between increasing age and risk of fatality. Figure 4.3 shows the proportion of crashes in which drivers were fatally injured as compared to all other crash severities by age group. It can be seen that the age group 65 years and above has the highest proportion of fatalities. Therefore, those aged 65 and above have a higher fatality *rate* given a crash, while drivers aged 18-25 and 26-45 comprise a greater *number* of fatalities.

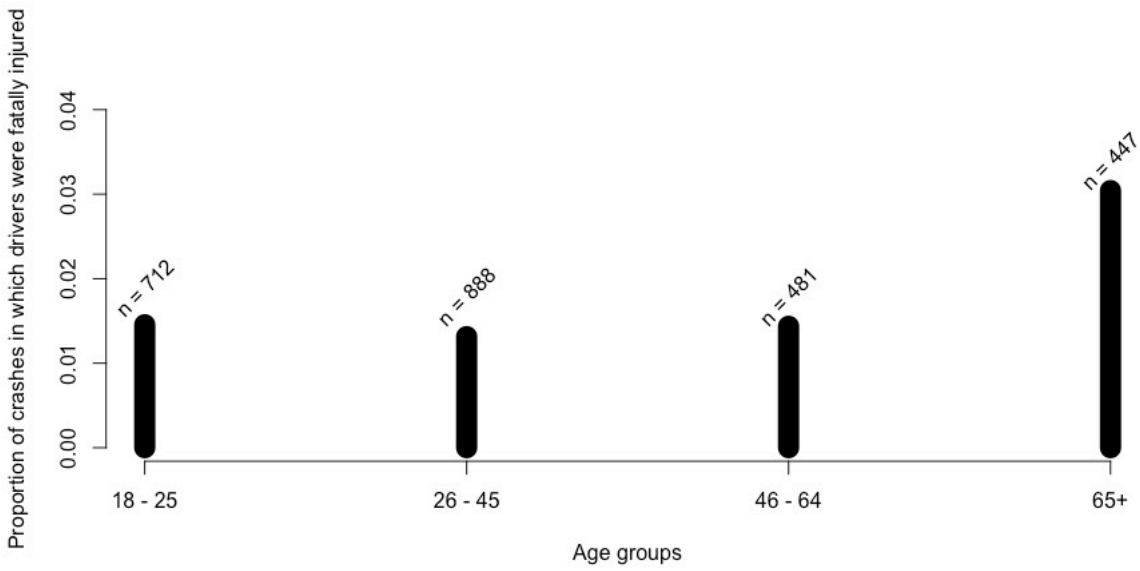

Figure 4.3 Proportion of crashes in which drivers were fatally injured as compared to all other crash severities by age group with number of fatalities in each group

These findings are consistent with previous research that has shown that older drivers are involved in fewer crashes (Baldock 2004; Thompson et al. 2013) but have a higher likelihood of serious or fatal injury in the event of a crash (Cicchino & McCartt 2014; Hanrahan et al. 2009; Rakotonirainy et al. 2012). The reduction in crash involvement for older drivers can be attributed, at least in part, to reduced exposure, as older adults drive less on average than those in younger age groups (Baldock 2004; Hanrahan et al. 2009; Thompson et al. 2013). This is demonstrated by the increased proportion of fatally injured older passengers as compared to all other crash injury severities by age group in Figure 4.4.

The greatest contributing factor to the increased vulnerability to fatal injury in older occupants is fragility. Fragility refers to the age-related increases in susceptibility to trauma (Li, Braver & Chen 2003). This is often due to age-related degeneration, particularly of the skeleton, with previous studies showing in increased risk of rib and cervical spinal fractures in older occupants (Kent et al. 2005; Kent, Richard & Patrie 2005; Stein et al. 2011). Thus, fragility increases risk of fatality by increasing the risk of injury, even in crashes that would otherwise be minor. The increased vulnerability of older occupants to injury is often

combined with frailty that leads to reduction in physiological reserve, which limits the body's capacity to survive or recover from the trauma (Hanrahan et al. 2009; Hashmi et al. 2014). Therefore, older occupants are at higher risk of being injured with a poorer physiological reserve and ability to compensate for trauma, resulting in a higher risk of fatality than younger occupants in a crash of equal severity. Essentially, in the view of the Safe System's core principles, age reduces the threshold of crash impacts that the human body can tolerate before sustaining serious or fatal injuries. It is for this reason that age will be included as a covariate in all statistical analysis for this thesis.

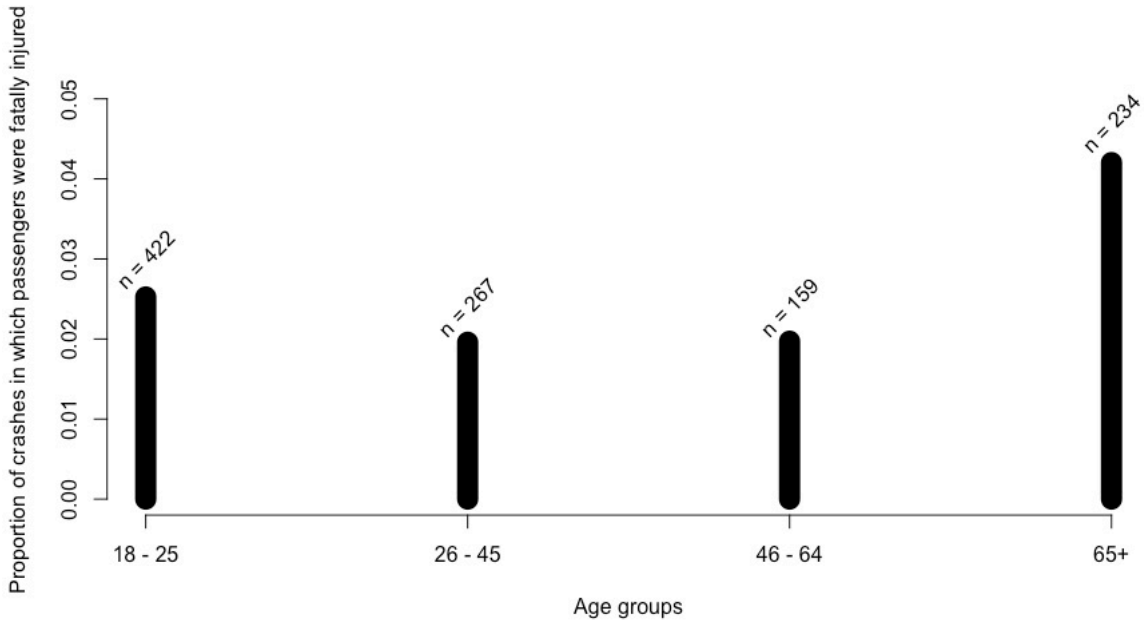

Figure 4.4 Proportion of passengers fatally injured in crashes, by age group with number of fatalities in each group

4.3 The effect of sex and its interaction with age

Sex is associated with an increased risk of crash involvement and increased risk of injury during a crash. Figure 4.5 decribes the proportion of minor and serious/fatal crashes involving male occupants compared to crashes involving female occupants. There are more minor injury crashes involving female occupants compared to males occupants but there are more male than female occupants involved in serious/fatal crashes.

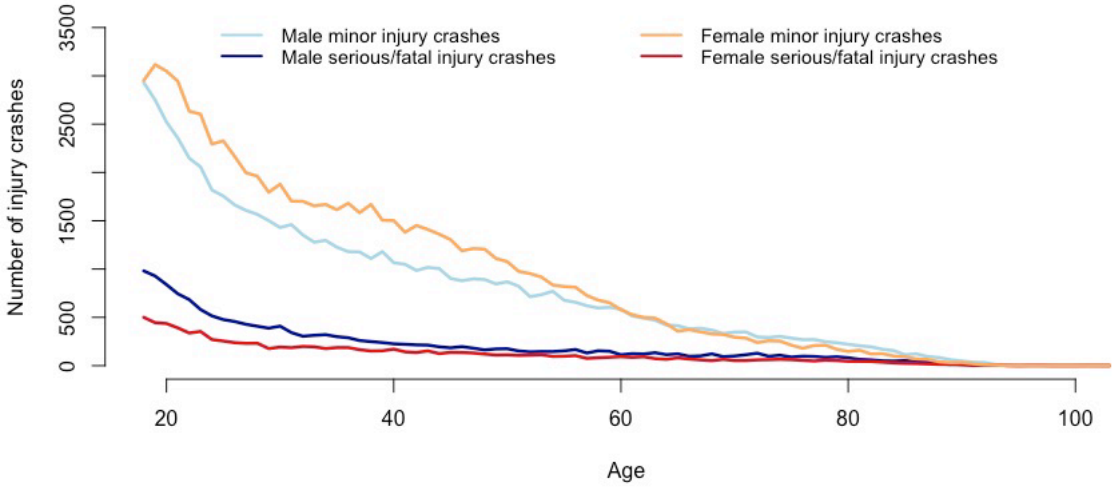

Figure 4.5 Frequency of minor and serious/fatal crashes by sex and age. Minor crashes are the combined total of private doctor treated and hospital treated crashes and serious/fatal crashes are the combined total of hospital admitted and fatal crashes from the TARS database from 1981-2020

This trend is well described in the literature, with males being more likely to be involved in serious and fatal crashes (Al-Balbissi 2003; Amarasingha & Dissanayake 2014; Cullen et al. 2021). This is also reflected in Figure 4.6, which shows all crashes in which occupants were fatally injured (drivers and passengers) by sex and age of the fatalities and demonstrates a higher incidence of fatalities involving males.

Male drivers are more likely to engage in risk taking behaviours such as speeding, driving under the influence of impairing substances and other driving violations (Al-Balbissi 2003; Cullen et al. 2021). The overlap of these traits between male drivers and young drivers produces a distinct group who have the highest crash risk of any driver group (Al-Balbissi 2003; Amarasingha & Dissanayake 2014; Jones 2017; Martin et al. 2013; Walter & Studdert 2015). The additional increase in fatality risk in younger male drivers is a combination of risky driving behaviour combined with driver inexperience (Alvarez et al. 2021; Everett et al. 2001; McEvoy, Stevenson & Woodward 2006; Peck et al. 2008; Shaaban, Gaweesh & Ahmed 2020; Stephens et al. 2017; Tsai, Anderson & Vaca 2010). Cullen et al. found young males scored the highest on indices of risk taking and sensation seeking behaviour and self-

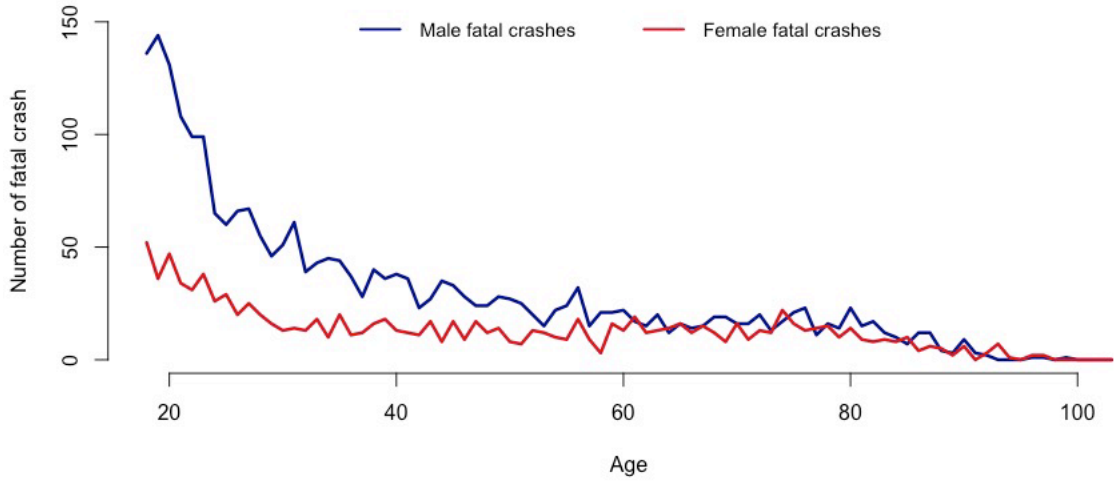

Figure 4.6 Number of fatal occupant crashes by age and sex

rated their driving ability higher than other drivers, a finding previously reported in the literature (Harré & Sibley 2007; Jiménez-Mejías et al. 2014). Furthermore, Cordellieri et al found that males' perception of risk is similar to that of females but that males were less concerned about the risk of a crash (Cordellieri et al. 2016). Research has suggested other psycho-social concepts such as masculinity and self-enhancement biases in males may contribute to risker driving behaviour and higher crash involvement (Harré & Sibley 2007; Schmid Mast et al. 2008; Simons-Morton et al. 2012).

In contrast, women are less likely to engage in risk-taking behaviour and drive less than males overall, contributing to a lower exposure to crash risk (Jiménez-Mejías et al. 2014). However, female drivers (particularly young female drivers) are becoming increasingly involved in fatal crashes that are associated with speed, improper manoeuvring or alcohol intoxication (Romano, Kelley-Baker & Voas 2008; Tsai, Anderson & Vaca 2010).

The risk of hospitalisation from a crash is higher in females than males (Bose, Segui-Gomez & Crandall 2011; Carter et al. 2014; Cullen et al. 2021). While an aspect of this increased risk may be related to female bias in the healthcare system (Cullen et al. 2021), several studies have demonstrated that women are at increased risk of injury, particularly in frontal and roll over crashes (Bose, Segui-Gomez & Crandall 2011; Brumbelow & Jermakian

2022; Carter et al. 2014; Kahane 2013; Wu et al. 2016). The anthropometric differences in males and females are suggested to contribute to this injury susceptibility, as females with a shorter stature are more at risk of lower limb injures (Bose, Segui-Gomez & Crandall 2011; Carter et al. 2014; Kahane 2013). Importantly, design of seat belts based on test models that were previously all anthropometric males may have contributed to the increased injury vulnerability in females, with restrained females having higher risk of spine and chest injuries than restrained males (Bose, Segui-Gomez & Crandall 2011).

Females are also at increased risk of pelvic fracture (Bansal et al. 2009; Schiff, Tencer & Mack 2008; Stein et al. 2006). The risk of pelvic fracture also increases with age, which has been attributed to loss of bone mineral density related to osteoporosis (Etheridge et al. 2005). The interaction between female sex and increasing age (Carter et al. 2014; Li, Braver & Chen 2003), combined with high prevalence of osteoporosis (Watts, Abimanyi-Ochom& Sanders 2013), characterises another highly vulnerable occupant category. For example, Li et al. found that female drivers aged 60 to 64 had nearly double the fatality rate per crash of female drivers aged 30 to 59 (Li, Braver & Chen 2003).

4.4 Summary

The interaction between age and sex creates categories of occupants who are at an elevated risk of being involved in a fatal crash. However, the underlying mechanisms of these increased risks differ across different high-risk groups. For young male drivers, the increased risk is due to the greater risk of crash involvement in general, while, for older occupants and particularly females, the increased fatal crash risk is due to an increased susceptibility to injury in the event of a crash.

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Chapter Five: Injury risk factors and modifiers

5.1 Introduction

Occupant characteristics and crash factors discussed in Chapters Three and Four are associated with both risk of fatal crash involvement and risk of fatal injury. This chapter discusses selected factors that affect the risk of the injury sustained in a crash, but which do not contribute to the risk of crash involvement. These factors and safety countermeasure can increase the risk or injury, or mitigate injury thereby changing the pattern of occupant injury. These include body mass index (BMI), seat belts, airbags, and vehicle age.

5.2 Body Mass Index

Body mass index (BMI) is a classification of body mass status calculated by the formula mass(kg)/height^2(m^2) (Samuelson 1995). Deviations from normality either side of an ideal BMI can be classified into several descriptive strata (e.g., underweight, overweight, obese, and morbidly obese). Australian National Health Survey data (2017-18) reported 76.9% and 72.1% of Australians aged 65-74 and ≥75years, respectively, are overweight or obese (Statistics 2019). Overweight and obese status is associated with increased mortality and co-morbidities including metabolic syndrome, diabetes mellitus, cardiovascular disease and cancer (Byard, 2012, 2015a). Obesity also correlates with increased mortality and injury risk following a MVC (Shi et al. 2015; Viano, Parenteau & Edwards 2008). The additional body mass has been demonstrated to increase the kinetic energy of the body during MVCs (Viano, Parenteau & Edwards 2008). Occupants with a higher BMI (>30kg/m^2) are reported to have higher fatality and injury risk compared with occupants with a “normal” BMI (Dubois et al. 2018; Hoebee et al. 2021; Joodaki et al. 2020; Viano, Parenteau & Edwards 2008). The reduced physiological response and associated complications with higher BMI post-injury may also be contributing to this increased risk (Viano, Parenteau & Edwards 2008). Literature also suggested that a low compliance with restraint use in occupants with higher

BMI, particularly in the morbidly obese (>40kg/m^2), may be increasing risk (Hoebee et al. 2021; Viano, Parenteau & Edwards 2008). However, in a study conducted as part of this thesis on seat belt marking, BMI was not a significant predictor of seat belt wearing, with 73.4% compliance overall in a sample of occupant fatalities from 2014 to 2018 (O'Donovan). Some studies also suggest that there is an interaction between sex and fatality risk in occupants with a higher BMI but whether males or females are at increased risk of fatality remains unclear. Zhu et al. found males are at greater risk, whereas Viano et al. and Dubois et al. found females are at greater fatality risk at higher than "normal" BMIs (Dubois et al. 2018; Viano, Parenteau & Edwards 2008; Zhu et al. 2006).

Excess body mass may also alter an individual's seating posture and restraint position, causing restraint system failure, greater energy dispersion across the pelvis and abdomen, and injurious airbag deployment (Kent, Forman & Bostrom 2010; Leung et al. 1982; Poplin et al. 2015; Shi et al. 2015). Greater body habitus and adipose tissue overlying the pelvis can delay the engagement of the pelvis, thus increasing seat belt webbing length while decreasing belt tension and introducing slack into the restraint system, causing the forward pelvic excursion (Carter et al. 2014; Forman et al. 2009; Hartka et al. 2018; Kent, Forman & Bostrom 2010; Leung et al. 1982; Reed, Ebert-Hamilton & Rupp 2012; Rupp et al. 2013; Shi et al. 2015). The position of the seat belt does not have to move above the pelvis to result in unwanted pelvic excursion, which may be the reason why some literature does not report an increased abdominal injury associated with increased BMI as the abdomen is not compressed. If, however, the abdominal portion of the belt does move above the anterior superior iliac spine, there is potential for the belt to inappropriately disperse force across the abdomen (Wang, Y et al. 2015). It is likely that in low impact crashes, this inappropriate compression is minimal and is offset by the additional abdominal tissue. In high impact crashes, the increased kinetic energy generated with higher BMI may lead to excess loading

of the abdomen if the belt slips above the anterior superior iliac spine that cannot be compensated for by additional abdominal adipose tissue. Disproportionate compression of the abdomen and submarining can further cause hyperflexion of the lumbosacral spine, potentially bursting the lumbosacral vertebrae in a fracture pattern known as a "Chance" fracture (Shanahan 2013; Shi et al. 2015).

Excess body mass can alter the occupant kinematics, increasing occupant velocity and the dispersion of impact energy through the knee-thigh-hip axis, thus increasing lower limb injury while reducing the impact to the upper torso and head (Arbabi et al. 2003; Boulanger et al. 1992; Hoebee et al. 2021; Hu et al. 2019; Kent, Forman & Bostrom 2010; Reed, Ebert-Hamilton & Rupp 2012; Rupp et al. 2013; Shi et al. 2015; Turkovich et al. 2013). The distorted restraint mechanism further alters chest deformation and spine translation, causing rib fractures and thoracic or lumbar fractures (Pipkorn et al. 2014; Shi et al. 2015; Tang, Zheng & Hu 2020; Wang, Y et al. 2015).

The extent of the protective nature of a high BMI for the abdominal and pelvic region in vehicle crashes remains contentious. The additional adipose tissue of the abdomen can mitigate impact force; more specifically, subcutaneous fat may 'cushion' an impact (Arbabi et al. 2003; Wang, SC et al. 2003). This is evident as underweight females are at greatest risk of pelvic fractures and a high BMI reduces pelvic fractures in lateral collisions (Bansal et al. 2009; Schiff, Tencer & Mack 2008; Stein et al. 2006). However, this absorptive action does not extend to skeletal extremities and is often dependent on collision force and orientation (Kent, Forman & Bostrom 2010; Zarzaur & Marshall 2008). The capacity for excess adipose tissue to absorb greater amounts of energy may be a protective factor (Arbabi et al. 2003; Boulanger et al. 1992; Fu et al. 2019; Turkovich et al. 2013; Wang, SC et al. 2003). This energy-absorbing potential may offset some of the additional force generated with a higher BMI, thereby

protecting the thoracic and intra-abdominal organs (Fu et al. 2019; Rupp et al. 2013; Wang, SC et al. 2003).

Low BMI or underweight individuals are considered to have a "chronic energy deficit" (Samuelson 1995) indicating a level of malnourishment often resulting in low skeletal fat and muscle mass. Although lower than "normal" BMIs are typically not associated with increased fatality risk, age amplifies the effect of BMI on fatality risk, with older drivers at the upper or lower parameters of the BMI spectrum having increased risk of fatality. Furthermore, Dubois et al. found in drivers aged 85 years and older that a higher than "normal" BMI may be protective, with a decreased likelihood of sustaining fatal injuries. In geriatric populations, low BMI is frequently associated with syndromes such as cachexia or frailty. Frailty syndrome describes age-relate cumulative decline of the body including deficits in physical ability and movement, physiological reserve and presence of comorbidities (Byard, 2015b). The 'frail obese' is an emerging concept describing a situation in which a level of frailty clinically exists (e.g., comorbidity or reduced physical ability), despite the individual's weight status traditionally omitting them. Many classification systems and scaling protocols now include obesity as a feature of frailty (Byard 2015b). Underweight and elderly individuals have a higher incidence of chest injuries related to the modified restraint energy dispersion across the thorax and reduced body mass causing multiple rib fractures (Carter et al. 2014).

5.3 Seat belts

Seat belts were mandatory in all vehicles registered at the beginning of 1967 in SA for drivers and front left passengers, and seat belts have been required in vehicles registered from 1971 for all occupant seating positions (Crinion, Foldvary & Lane 1975). Since seat belt wearing legislation came into effect in late 1971, several amendments have been added to include child restraints. Failure to correctly wear a seat belt in SA incurs a fine and demerit points.

Seat belt design has also evolved since the first version of the three-point belt was introduced by Volvo in 1959 (King & Yang 1995). Seat belts act by dispersing impact load through skeletal structures such as the rib cage and pelvis, while reducing decelerative forces through soft tissue and internal organs (D'Aulerio et al. 2019). Seat belts also minimise occupant movement during a collision, thus limiting impact with internal vehicle structures and preventing ejection. Seat belt restraint systems in most motor vehicles consists of three anchor points with belts positioned diagonally across the shoulder and chest, with the lap portion lying horizontally across the lower abdomen and pelvis. When positioned correctly, the abdominal portion lies across the upper anterior pelvis below the level of the anterior superior iliac spines (Greenston, Wood & Reinhart 2019; Jiang et al. 2019). Seat belts had an immediate and significant effect on the serious injury and fatality rate in MVCs, which was further improved with load limiters and pretensioners (Foret-Bruno et al. 2001; Trinca & Dooley 1975; Walz 2004). Undoubtedly, seat belt use significantly reduces the risk of fatal injury but there remain circumstances in which seat belt use has caused injury or in which seatbelt malfunction has been the mechanism of death.

One of those circumstances occurs when there is incorrect seat belt positioning prior to a crash. This may be due to increased abdominal circumference associated with higher BMI, pregnancy, small stature, or deliberate incorrect seat belt wearing. Occupants can also become out of position either due to movement during the crash or if the seat belt moves above the pelvis, inappropriately dispersing impact energy during a crash.

Seat belt action may leave cutaneous skin markings across the neck, thorax, or abdomen. Previous clinical studies demonstrated that seatbelt marking is an indicator of seat belt use and internal abdominal injury (Chandler, Lane & Waxman 1997; Doersch & Dozier 1968; Glover et al. 2018; Greenston, Wood & Reinhart 2019; Jiang et al. 2019; Shreffler et al. 2020). To investigate if this indicator is present in fatally injured vehicle occupants, a study

was undertaken within the scope of this thesis. The full publication can be found at the end of this chapter (Chapter 5.8). Seat belt marking was not associated with increased abdominal injury but was associated with increasing BMI and the presence of bilateral pelvic fracture. The findings from this study suggests that, while greater body mass may increase occupant kinetic energy causing greater restraint force across the abdomen, leading to cutaneous markings on the skin, the increased abdominal adipose tissue may absorb and disperse any excess energy that may be created, protecting the internal abdominal organs as discussed previously. The bilateral pelvic fractures suggest that there may be incorrect loading of the pelvis, likely due to abdominal lap belt movement above the anterior superior iliac spine. The increased incidence of bilateral pelvic fracture may also be due to the increased load through the knee-thigh-hip axis in occupants with higher BMIs. A separate study from the same sample showed an association between cutaneous seat belt marking and season of year, suggesting that the thicker or additional clothing worn during the winter months may be protecting against cutaneous marking. The full publication can be found at the end of this chapter (Chapter 5.9).

The movement of the occupant during a crash or the final position of the vehicle can lead to seat belt malfunction. While relatively rare, cases of lethal seat belt asphyxia have been reported in the literature, and a case report was published as part of this thesis describing the lethal asphyxia due to the seat belt compressing the occupant's neck (O'Donovan et al. 2021). The full publication can be found at the end of this chapter (Chapter 5.10). Several other studies report seat belts riding above the upper thorax to compress the neck either due to submarining or vehicle position after a crash (Byard, R. W. & Noblett 2004; Byard, Roger W., O'Donovan & Gilbert 2020; Hitosugi & Takatsu 2000; James & Byard 2001; Veenema 1994).

5.4 Airbags

Airbags were introduced as standard for Australian vehicles in 1990 predominantly for driver and front seat passengers, with side curtain airbags becoming standard from 1995. The first

generation of airbags was designed to be compatible with non-use of seat belts and so the airbags were more powerful when deploying to counteract a forward moving, unrestrained body (Cunningham et al. 2000). As the majority of unrestrained occupants would be out of position during deployment, there were a number of serious injuries resulting from airbags and several fatalities were caused by neck hyperextension and blunt chest trauma (Cunningham et al. 2000; Hoye 2010; Inamasu & Kato 2018; Wallis & Greaves 2002). Recent airbag models have also been recalled due to the risk of metal shrapnel being launched upon airbag deployment, which has led to serious and fatal injury. Airbags also pose the risk of thermal and chemical burns to occupants due to the heat generated at deployment which may be present at autopsy. Airbags have also been implicated in facial soft tissue and fracture, particularly due to lower speed collisions in which the airbag may have inappropriately deployed. A review of lethal airbag injuries and associated injuries was published as part of this thesis. The full publication can be found at the end of this chapter (Chapter 5.11). Second and third generation airbags have far less deployment power and are devised as a supplemental restraint to be used with seat belts (Segui-Gomez 2000). While there remains a slightly increased risk of serious injury for smaller occupants, such as women (Newgard & McConnell 2008) and children (Cunningham et al. 2000), and risk of upper limb injury (Cunningham et al. 2000) during deployment, the overall effect of airbags particularly during severe crashes remains conclusively protective (Duma et al. 2003).

5.5 Vehicle age

Vehicle age is an important factor that can mediate injury severity, as crash-involved drivers in older vehicles have higher injury severity than drivers of newer vehicles (Lécuyer & Chouinard 2006; Yasmin, Eluru & Pinjari 2015). This is related to improved crashworthiness (i.e., the vehicle's ability to withstand crash impacts) and improved vehicle safety features in newer vehicles (Anderson & Doecke 2010). Vehicle age is likely to be an important factor in

South Australian crashes as the South Australian passenger vehicle fleet is older than those in most other Australian jurisdictions (an average vehicle age in SA of 11.2 years compared to 9.9 years for Australia as a whole) (Anderson & Doecke 2010). Vehicle age may also interact with other factors that are related to crash and injury risk. For example, a study by Keall et al. found that younger drivers (18-25 years), who have the highest crash risk, drove older vehicles, while drivers aged between 26-59 years drove the newest vehicles (Keall & Newstead 2011).

5.6 Other injury modifying factors

There are several other factors that are known to influence injury severity and patterns of injury that were not obtainable for this research despite the combined data sources. These included speed of impact, change in velocity (known as delta-V), vehicle intrusion, and factors that relate to the crash sequence such as point and direction of impact. Principle impact direction and point of impact can affect the severity of a crash; for example, frontal and lateral impacts are associated with higher injury severity.

TARS does include information regarding the crash type, which provides a broad description of the crash sequence, but the lack of specific information regarding points and direction of impact may limit some of the conclusions that can be drawn from this research. This also limits analysis that investigates specific seating position and injury patterns particularly for rear seat occupants. The rear seating positions have therefore been combined into a single category for this study. However, previous studies have successfully drawn conclusions regarding the patterns of injury without the need to differentiate between the exact positioning of the rear seat (Bilston, Du & Brown 2010; Brown & Bilston 2014; Li et al. 2020; Shimamura, Yamazaki & Fujita 2005).

As discussed previously, speed is strongly associated with risk of crash involvement and fatal injury. Travelling speed prior to the crash is directly correlated to impact speed and

crash energy. Therefore, the greater the impact speed, the greater the associated injury severity (Doecke et al. 2020; Doecke et al. 2021; Kloeden et al. 1997). Speed of impact can also affect the degree of vehicle intrusion, which is also related to the extent of injury. Although this parameter is useful when determining injury severity, the current research focuses on fatal crashes, the endpoint of injury severity, and the majority of these crashes are assumed to involve high energy impacts.

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5.8 Publication: Seat belt injuries and external markings at autopsy in cases of lethal vehicle crashes

S. O'Donovan, C. Van Den Heuvel, M.R.J. Baldock, M.A. Humphries, R.W. Byard

Seat belt marking is a clinical indicator for potential underlying trauma of the chest or abdomen. To investigate if seat belt marking present at autopsy was associated with an increased incidence of underlying chest or abdomen injury, a 5 year review of a sample of vehicle occupants was undertaken. The seat belt marking time frame was restricted due to limitations in accessing digitised autopsy photographs prior to 2014.

Statement of Authorship

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Principal Author

Name of Principal Author (Candidate)	Siobhan O'Donovan		
Contribution to the Paper	Collected autopsy data, performed statistical analysis, wrote manuscript, acted as corresponding author		
Overall percentage (%)	85%		
Certification:	This paper reports on original research I conducted during the period of my Higher Degree by Research candidature and is not subject to any obligations or contractual agreements with a third party that would constrain its inclusion in this thesis. I am the primary author of this paper.		
Signature		Date	27/05/2022

Co-Author Contributions

By signing the Statement of Authorship, each author certifies that:

- the candidate's stated contribution to the publication is accurate (as detailed above);
- permission is granted for the candidate in include the publication in the thesis; and
- the sum of all co-author contributions is equal to 100% less the candidate's stated contribution.

Name of Co-Author	Corinna Van Den Heuvel		
Contribution to the Paper	Primary supervisor of Siobhan, helped to evaluate and edit the manuscript		
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Contribution to the Paper	Co-supervisor of Siobhan, provide access to Traffic Accident Reporting System through Centre for Automotive Safety Research, helped to evaluate and edit the manuscript		
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Name of Co-Author	Melissa Humphries		
Contribution to the Paper	Performed statistical analysis, assisted in data interpretation and visualisation, evaluation of manuscript		
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Seat belt injuries and external markings at autopsy in cases of lethal vehicle crashes

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Abstract

A study was undertaken to determine what injuries are associated with the wearing of seat belts and if the presence of cutaneous seat belt markings observed on victims of lethal vehicle crashes increased the likelihood of underlying injury. Autopsy reports from the files at Forensic Science South Australia were reviewed for all fatal motor vehicle crashes from January 2014 to December 2018. A total of 173 cases were included for analysis with 127 occupants wearing seat belts at the time of impact (73.4%) (age range = 18–93; mean = 45 M:F = 81:46). Of these, only 38 had external seat belt markings (29.9%) (age range = 19–83; mean = 49 M:F = 20:18). Logistic regression modelling showed that occupants who were wearing seat belts were more likely to experience closed head injury without skull fractures in addition to mesenteric and gastrointestinal injury. Increasing body mass index increased the incidence of seat belt markings ($p < 0.01$) and markings were more likely to be found in the presence of bilateral pelvic fractures. Thus, external seat belt markings were observed in only a minority of seatbelt wearers, and more often in individuals with higher BMIs and with bilateral pelvic fractures (possibly associated with greater momentum and impact force).

Keywords

Seatbelt, vehicle crash, cutaneous markings, internal injury, body mass index

Introduction

Seat belts in vehicles significantly reduce morbidity and mortality associated with crash impacts by dispersing impact load through skeletal structures such as the rib cage and pelvis, while reducing decelerative forces through soft tissue and internal organs.[1] Seat belts also minimise occupant movement during a collision thus limiting impact with internal vehicle structures and preventing ejection. Seat belt restraint systems in the majority of motor vehicles consist of three anchor points with belts positioned diagonally across the shoulder and chest, with the lap portion lying horizontally across the lower abdomen and pelvis. When positioned correctly, the abdominal portion lies across the upper anterior pelvis below the level of the anterior superior iliac spines.[2,3] Additional safety modifications have been integrated into restraint system design including pretensioners and load limiters which have significantly reduced excessive load being applied to an occupant's body on deceleration.

Seat belts can, however, cause occupant injury ranging from minor linear intra-dermal abrasions or contusions underneath the overlying belt to the fracturing of the sternum and ribs, injury to abdominal and thoracic organs, vascular transaction and fracturing of the lumbosacral spine (the so-called Chance fracture).[2–6] Previous research has shown an association between cutaneous seat belt markings and the presence of underlying internal, particularly abdominal injury.[2,3,7–10] The current study aims to investigate the type of injuries that are associated with the use of seat belts, and if the presence of cutaneous seat belt markings can be used as a marker for internal injury in occupant fatalities.

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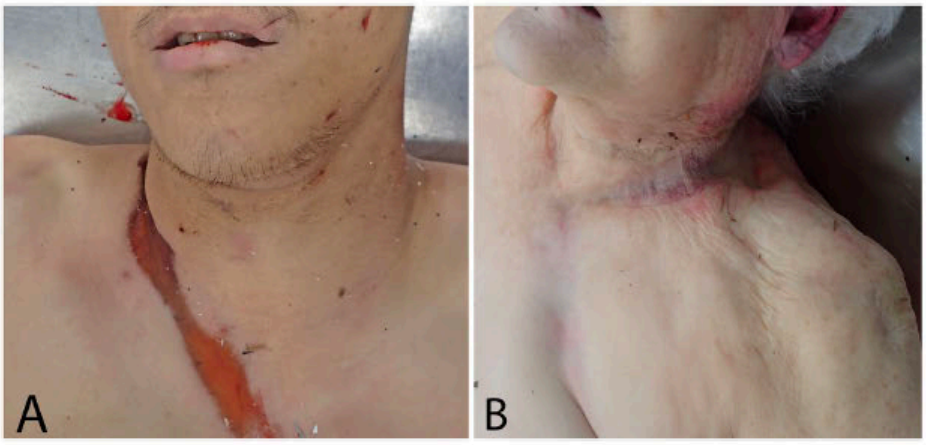

Figure 1. Markings on the neck and upper chest from seatbelts may vary from well-defined areas of linear parchmenting extending downwards and laterally (A), to more subtle areas of linear bruising limited to the base of the neck (B).

Materials and methods

Data source

This research was approved by the University of Adelaide Low-Risk Human Research Ethics Review Group and Forensic Science South Australia Research and Development Committee. A retrospective review of autopsy reports of adult (≥ 18 years old) vehicle occupants was undertaken at Forensic Science South Australia (FSSA) between January 2014 and December 2018. Individual cases were then cross-matched with corresponding collision data from the Traffic Accident Reporting System (TARS) provided through the Centre for Automotive Safety Research at The University of Adelaide. The TARS database includes deidentified information relating to the nature, cause, and time of all police-reported road crashes in South Australia involving one or more of the following conditions: one or more persons in the collision were injured, vehicular damage was $3000 AUD or greater, or one or more of the vehicles was towed. Crash location and restraint use are also recorded. Unfortunately, the force of collision impact and point of impact could not be accurately determined from the available records. As airbag deployment was inconsistently reported this was also not included in the analysis.

Case extraction

A seat belt marking was defined as a cutaneous abrasion, contusion and/or laceration corresponding to the position of the overlying seat belt. The presence of seat belt markings was identified in the external injury description in autopsy files and confirmed by examining autopsy photographs. Figures 1A, B to 4 show the types of cutaneous marks made by seat belts to the neck, chest and abdomen. Exclusion criteria included ante or perimortem incineration,

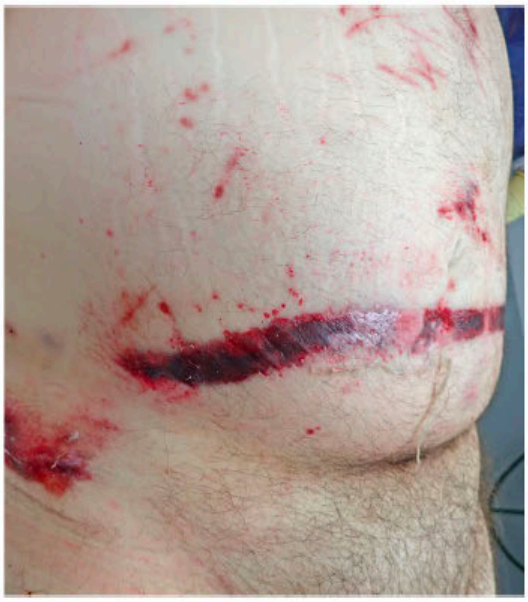

Figure 2. Horizontal parchmenting in the lower abdomen from the lap component of a seat belt.

chest and abdominal ecchymosis and bruising of uncertain origin, and unknown restraint use at the time of the collision (Figure 5).

The age and sex of de-identified decedents were extracted from autopsy files, along with other occupant characteristics such as body mass index (BMI) and associated injuries. The binary classification of injuries was applied, (1 = injury present, 0 = no injury present). Injuries to soft tissue and organs were classified as 1 = yes if the injury was greater than 3 or more (3 +) on the Abbreviated Injury Scale (AIS) which ranks injury severity on a scale from 1 to 6.[11] AIS 3+ is considered a serious-to-fatal injury.

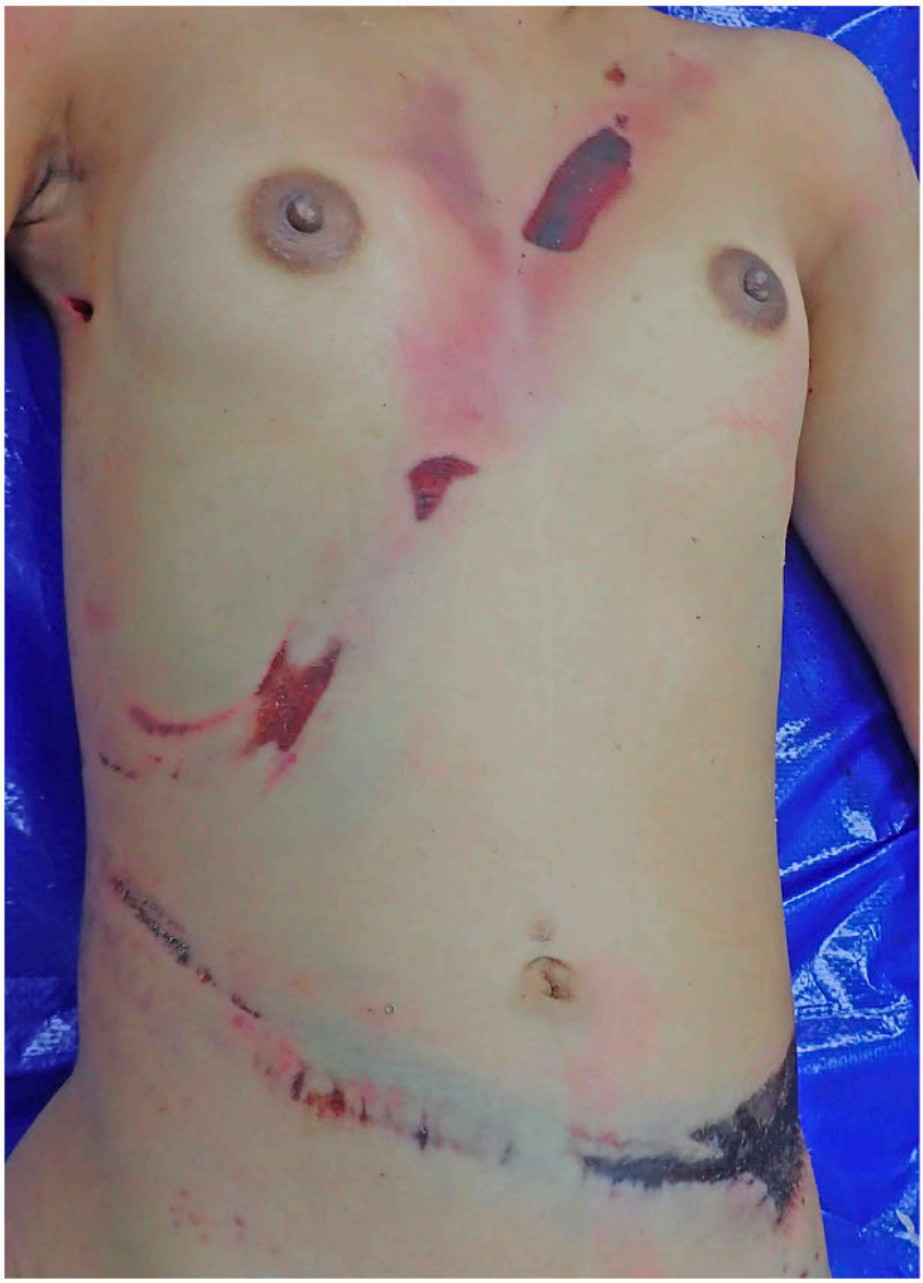

Figure 3. On occasion markings from both the sash and lap components of a seatbelt may be discernible.

Data analysis

The demographic features, crash characteristics and circumstances of fatal collisions were examined in relation to restraint use and the occurrence of seatbelt markings. Cases with missing data were omitted from the analysis (Figure 1). Statistical analyses were performed using R studio statistical software (version 1.4).[12] Model development ($\alpha = 0.05$) was separated into two stages and model selection was determined using Bayesian Information Criterion (BIC) scoring. Firstly, a stepwise logistic regression model predicting seat belt wearing status was performed to identify factors and injuries that may confound or mask interactions when modelling for the occurrence of seat belt marking. Injuries observed at autopsy were also included in the analysis and odds ratios (OR) and corresponding 95% confidence intervals (95% CI) were calculated for each independent variable. A second stepwise logistic regression was performed on the seat belt-wearing

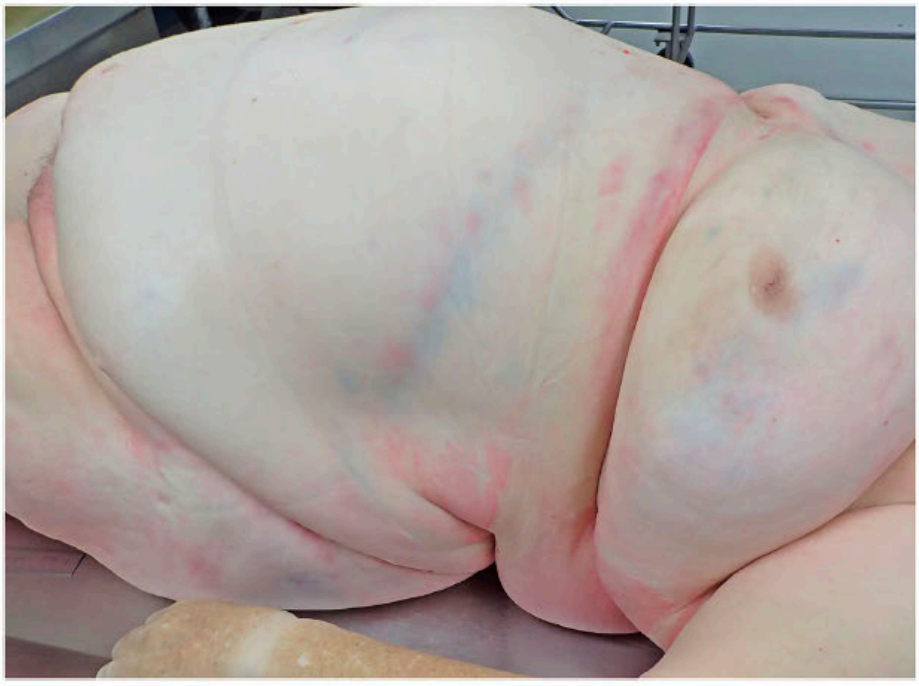

Figure 4. A transverse linear abdominal bruise demonstrating upward displacement of the lap component of the belt due to increased abdominal adipose tissue.

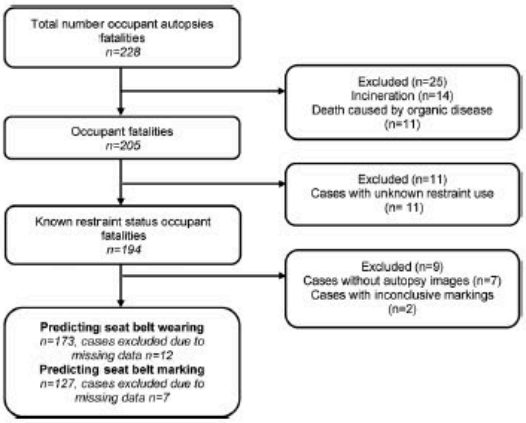

Figure 5. Flow diagram showing case inclusion and exclusion criteria.

cohort, predicting the occurrence of seat belt marking. This model included the same occupant characteristics, crash data and injury categories and OR and 95% CI were calculated for each independent variable.

Results

From 1 January 2014 to 31 December 2018, a total of 286 motor vehicle occupant fatalities were recorded on South Australian roads. Of these, 228 cases underwent coronial autopsy and, based on the above selection criteria, 173 were included in the analysis.

Seat belt wearing

Over half of the occupants were wearing seat belts (127 [73.4%]) (Table 1). Linear regression modelling found that occupants who drove more recent model vehicles were more likely to be wearing a seat belt ($p = 0.001$, OR = 1.11, 95% CI = 1.05–1.19) and that unbelted occupants were more likely to be ejected ($p < 0.001$, OR = 0.04, 95% CI = 0.01–0.12). Belted occupants were more likely to have closed blunt force head injuries without a skull fracture ($p = 0.026$, OR = 3.38, 95% CI = 1.22–10.80) and were more likely to have injuries to the mesentery and/or gastrointestinal tract ($p = 0.038$, OR = 8.57, 95% CI = 1.45–93.5) (Table 2). For full model output please see Online Appendix A.

Seat belt marking

Of the seat belt-wearing cohort, 38 (29.9%) had seat belt markings on either the chest, across the abdomen, or both (Table 3). Logistic regression modelling showed that the best predictors for the presence of seat belt markings were BMI ($p = 0.007$, OR = 1.10, 95% CI = 1.05–1.17) (Figure 6) and bilateral pelvic fractures ($p = 0.014$, OR = 3.44, 95% CI = 1.29–9.44) (Table 4). Abdominal

Table 1. Demographic characteristics of 173 seat belt wearing in occupant fatalities and unbelted occupant fatalities in South Australia (2014–2018).

Characteristic	Overall, N = 173	Unbelted, N = 46	Belted, N = 127	p-value[a]
Age	44 (18–93)	40 (18–79)	45 (18–93)	0.30
Sex				0.072
Male	117 (68%)	36 (78%)	81 (64%)	
Female	56 (32%)	10 (22%)	46 (36%)	
Body mass index (BMI)	28 (17–56)	27 (17–56)	28 (17–52)	0.28
Year of vehicle manufacture	2002 (1981–2016)	1998 (1981–2011)	2004 (1982–2016)	<0.001

[a]Wilcoxon rank sum test; Pearson's Chi-squared test; Fisher's exact test.

Table 2. Results of logistic regression designed to predict seat belt wearing in occupant fatalities in South Australia (2014–2018).

Characteristic	Odds ratio	95% CI	p-value
Ejection			
Not ejected	REF	REF	
Ejected	0.04	0.01, 0.12	<0.001
Year of vehicle manufacture	1.11	1.05, 1.19	0.001
Blunt force head injury without skull fracture			
No injury	REF	REF	
Injury present	3.38	1.22, 10.8	0.026
Mesenteric injury			
No injury	REF	REF	
Injury present	8.57	1.45, 93.5	0.038

CI: Confidence Interval; REF: reference category.

Table 3. Demographic characteristics of 127 seat belt wearing in occupant fatalities and unbelted occupant fatalities in South Australia (2014–2018).

Characteristic	No marking, N = 89	Seat belt marking, N = 38	p-value[a]
Age	44 (18–93)	49 (19–83)	0.28
Sex			0.088
Male	61 (69%)	20 (53%)	
Female	28 (31%)	18 (47%)	
Body mass index (BMI)	27 (17–45)	30 (19–52)	0.065
Year of vehicle manufacture	2003 (1982–2016)	2004 (1990–2016)	0.42

[a]Wilcoxon rank sum test; Pearson's Chi-squared test.

vasculature injury was also included in the final model based on the lowest BIC but was not significant. For full model output please see Online Appendix A.

Discussion

Increasing BMI and the presence of bilateral pelvic fractures were found to be the most significant features associated with seat belt markings in occupants involved in fatal MVC. High BMI which is often associated with large abdominal circumference can alter the fit of seat belts[13] as the lap portion of the belt shifts further upwards and above the anterior superior iliac spines (ASIS), increasing the webbing length of both the shoulder and lap portion of the belt.[13] The extended webbing length combined with greater body mass increases the counteractive restraint force applied to the occupant which could contribute to the increased incidence of seat belt markings.[13–15]

Despite a high BMI being associated with an increased injury risk, particularly involving cervicothoracic or intra-abdominal structures, modelling in the current study only demonstrated a significant association of seat belt markings with bilateral pelvic fractures.[3,7–9,16–21] Additional adipose tissue overlying bony structures can decrease belt tension and introduce slack into the restraint system delaying skeletal loading from the seat belt during impact.[13] The delayed engagement of skeletal structures may then increase the forward movement of the occupant, specifically forward excursion of the pelvis, that would have otherwise been restrained.[14,22–26] Excessive forward pelvic excursions can also lead to the seat belt moving above the ASIS (Figure 4), causing the occupant to "submarine" below the intended restraint position.[27] This has also been observed with children using booster seats.[28] Disproportionate compression of the abdomen and submarining can further cause hyperflexion of the lumbosacral spine potentially bursting the lumbosacral vertebrae in a fracture pattern known as a "Chance" fracture although this was not observed in the current study.[19,25] The altered occupant kinematics together with forward pelvic excursion may also increase occupant velocity and alter the distribution of impact energy through the knee-thigh-hip axis thus increasing lower limb injury while reducing the impact to the upper torso and head.[13,17,23–26,29,30] It may also be true that occupants

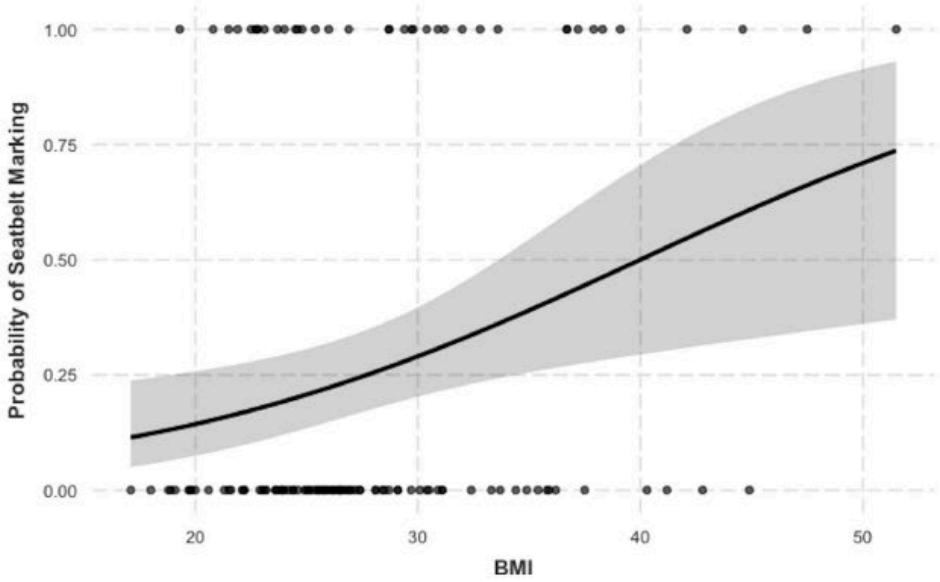

Figure 6. The probability of seatbelt markings by body mass index (BMI) in motor vehicle occupant fatalities with 95% CI (2014–2018).

Table 4. Results of logistic regression designed to predict seat belt marking in occupant fatalities in South Australia (2014–2018).

Characteristic	Odds ratio	95% CI	*p*-value
BMI	1.09	1.03, 1.17	0.007
Bilateral pelvic fracture			
No injury	REF	REF	
Injury present	3.44	1.29, 9.44	0.014

CI: confidence interval, REF: reference category; BMI: body mass index.

with seat belt markings and bilateral pelvic were subjected to more significant impact force resulting in the occurrence of both injuries.

The imposition and distortion of seat belt loading that occurs when a seat belt slips above the ASIS and disproportionately compresses the abdomen has the potential to cause intra-abdominal injury.[31,32] However, it appears that the capacity for excess adipose tissue to absorb greater amounts of energy may be a protective factor.[26,29,33–35] This energy-absorbing potential may offset some of the additional force generated with a higher BMI, thereby protecting the thoracic and intra-abdominal organs and contributing to the observed absence of abdominal injury in the final model.[24,34,35]

Occupants who did not wear seat belts were significantly more likely to be ejected.[36] Closed head injury without skull fracture and gastrointestinal/mesenteric injury were more frequently seen in belted occupant fatalities, findings consistent with previous literature.[16,37,38] Higher restraint compliance was observed in newer vehicles which may be attributable to improvements in vehicle technology such as seat sensors that prompt occupants to buckle seat belts before driving. Several studies have suggested that non-compliance with seat belt wearing may also increase with higher BMI due to difficulties with restraint fit.[17,29,39] However, BMI was not a significant predictor of seat belt wearing in the current study with 73.4% compliance overall. A review of crashes of varying severity in South Australia from June 2019 to June 2020 by Elsegood et al.[40] showed 97.9% driver compliance and 93.3% passenger compliance with seat belt laws.

There were several limitations to this study relating to gaps in data availability and collection. For example, several important vehicles and crash factors such as a change in vehicle velocity on impact and point and direction of the collision were unavailable for analysis. Fatal crashes are likely to be high-speed impacts and therefore there is an assumption that the majority of these collisions involved significant force. Airbag use can also protect occupants from various injuries but is not routinely recorded in TARS or forensic autopsy reports.

The present study demonstrated that an increase in BMI was associated with a greater likelihood of seat belt markings and bilateral pelvic fractures were observed significantly more often in the presence of external cutaneous belt markings. BMI should be considered a confounding factor for future research when assessing internal injury in the presence of cutaneous seat belt markings.

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Declaration of conflicting interests

The authors declared no potential conflicts of interest with respect to the research, authorship, and/or publication of this article.

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Supplemental material

Supplemental material for this article is available online.

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Supplementary Material to 5.8

Appendix A.

Table A1
Model output predicting seat belt wearing (1)

Deviance Residuals:

Min	1Q	Median	3Q	Max
-2.2216	-0.3698	0.3433	0.6249	1.9778

Coefficients:

	Estimate	Standard Error	z value	Pr(>|z|)
(Intercept)	-213.26691	65.00665	-3.281	0.00104 **
Ejected	-3.17493	0.58545	-5.423	5.86e-08 ***
Year of vehicle manufacture	0.10563	0.03247	3.253	0.00114 **
Blunt force head injury without skull fracture (1)	1.21869	0.54842	2.222	0.02627 *
Mesenteric injury (1)	2.12820	1.03648	2.073	0.03821 *

Signif. codes: 0 ‘***’ 0.001 ‘**’ 0.01 ‘*’ 0.05 ‘.’ 0.1 ‘ ’ 1

(Dispersion parameter for binomial family taken to be 1)

Null deviance: 195.88 on 168 degrees of freedom
Residual deviance: 127.30 on 164 degrees of freedom
AIC: 137.3
BIC: 152.9445

Number of Fisher Scoring iterations: 6

Table A1 is predicting the probability of seat belt wearing where an occupant wearing a seat belt was equal to 1.

Table A2
Model output predicting seat belt marking (1)

Deviance Residuals:

Min	1Q	Median	3Q	Max
-1.7752	-0.7495	-0.6524	0.9710	1.9398

Coefficients:

	Estimate	Standard Error	z value	Pr(>|z|)
(Intercept)	-3.57615	0.96995	-3.687	0.000227 ***
BMI	0.08942	0.03286	2.721	0.006503 **
Abdominal vascular (1)	-16.99417	1079.78222	-0.016	0.987443
Bilateral pelvis (1)	1.23593	0.50364	2.454	0.014127 *

Signif. codes: 0 ‘***’ 0.001 ‘**’ 0.01 ‘*’ 0.05 ‘.’ 0.1 ‘ ’ 1

(Dispersion parameter for binomial family taken to be 1)

Null deviance: 154.99 on 126 degrees of freedom
Residual deviance: 137.55 on 123 degrees of freedom
AIC: 145.55
BIC: 156.93

Number of Fisher Scoring iterations: 15

Table A2 is predicting the probability of the presence of seat belt marking. BIC was used for model selection with the model including abdominal vascular injury despite there being no significance.

5.9 Publication: Seasonal variation in cutaneous seat belt markings in fatal vehicle crashes

S. O'Donovan, C. Van Den Heuvel, M.R.J. Baldock, M.A. Humphries, R.W. Byard

Clothing imposition has been attributed to the absence of seat belt marking at autopsy but there is no evidence in the literature supporting this theory. This study of cases from 2014 to 2018 assessed if the incidence of seat belt marking was higher in the summer months when less clothing is presumably worn, and whether the incidence was lower during the winter months when more clothing is presumably worn.

Statement of Authorship

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Publication Details	**O'Donovan, S**, Van Den Heuvel, C, Baldock, MRJ, Humphries, MA & Byard, RW 2022, 'Seasonal variation in cutaneous seat belt markings in fatal vehicle crashes', *Australian Journal of Forensic Sciences,* advance online publication, DOI: 10.1080/00450618.2022.2117413.

Principal Author

Name of Principal Author (Candidate)	Siobhan O'Donovan		
Contribution to the Paper	Collected autopsy data, performed statistical analysis, wrote manuscript		
Overall percentage (%)	85%		
Certification:	This paper reports on original research I conducted during the period of my Higher Degree by Research candidature and is not subject to any obligations or contractual agreements with a third party that would constrain its inclusion in this thesis. I am the primary author of this paper.		
Signature		Date	27/05/2022

Co-Author Contributions

By signing the Statement of Authorship, each author certifies that:

- the candidate's stated contribution to the publication is accurate (as detailed above);
- permission is granted for the candidate in include the publication in the thesis; and
- the sum of all co-author contributions is equal to 100% less the candidate's stated contribution.

Name of Co-Author	Corinna Van Den Heuvel		
Contribution to the Paper	Primary supervisor of Siobhan, helped to evaluate and edit the manuscript		
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Name of Co-Author	Matthew Baldock

Contribution to the Paper	Co-supervisor of Siobhan, helped to evaluate and edit the manuscript		
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Please cut and paste additional co-author panels here as required.

Name of Co-Author	Melissa Humphries		
Contribution to the Paper	Performed statistical analysis, assisted in data interpretation and visualisation, evaluation of manuscript		
Signature		Date	20/06/2022

Name of Co-Author	Roger Byard		
Contribution to the Paper	Co-supervisor of Siobhan, helped to evaluate and edit the manuscript, acted as corresponding author		
Signature		Date	08/07/2022

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Seasonal variation in cutaneous seat belt markings in fatal vehicle crashes

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ABSTRACT

A retrospective review of autopsy reports of adult (≥18 years old) vehicle occupants was undertaken at Forensic Science SSA (FSSA) over a five-year-period from January 2014 to December 2018 to correlate seat belt wearing with cutaneous seat belt injuries and season of the year. Out of 185 cases included in the study 134 vehicle occupants were wearing seat belts (72.4%) and, of these, 44 (32.8%) had seat belt markings across either the chest, the abdomen, or both. No significant differences were found in the incidence of seat belt wearing over the seasons: summer $N = 37$ (28%), autumn $N = 28$ (21%), winter $N = 32$ (24%) and spring $N = 37$ (28%); however, there was a significantly lower number of cases with markings in the colder months: summer $N = 16$ (36%), autumn $N = 9$ (20%), winter $N = 4$ (9.1%) and spring $N = 15$ (34%) ($p = 0.027$). It is unclear why there are fewer cutaneous markings from seat belts in the colder months of the year; however, it is possible that this may be related to reduced impact forces due to lower vehicle speeds during inclement weather, or to the wearing of more clothing during colder months which coincidentally acts as padding.

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KEYWORDS
Seat belt markings; vehicle crash; clothing; season of year; temperature

Introduction

The compulsory wearing of seat belts in both commercial and domestic vehicles has been legislated in many countries in an attempt to reduce death and injury from crashes [1,2]. Seat belt webbing is designed to sit over bony prominences and thus disperse impact loads and reduce deceleration forces that may severely damage internal organs and soft tissues. In addition, properly worn seat belts prevent occupants impacting against the vehicle cabin or being ejected [3,4].

At autopsy, one of the undertakings in the assessment of crashes is to ascertain whether seat belts had been worn by checking for cutaneous markings. This can assist in the analysis of injuries and also in helping to position the deceased to one side of the vehicle or the other [4]; such positioning may be of particular importance if criminal charges are to be laid.

To determine how common cutaneous seat belt markings are, and whether they may occur less frequently at particular times of the year, the following study was undertaken.

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Materials and methods

A retrospective review of autopsy reports of adult (≥18 years old) vehicle occupants was undertaken at Forensic Science SA (FSSA) over a five-year-period from January 2014 to December 2018. The age and sex of the deceased were tabulated. Seat belt wearing or not was noted from the police report and Report of Death to the Coroner and the autopsy reports, and post-mortem photographs were reviewed by the first and senior authors to record all cases with cutaneous marks from seat belts. These consisted of linear markings (usually parchmented abrasions or bruises) on the chest or abdomen in positions corresponding to the sash and lap components of seat belts. As airbag deployment was inconsistently reported this was not included in the analysis. Cases were also excluded if there had been incineration, chest and abdominal ecchymosis and bruising of uncertain origin, and unknown restraint use at the time of the collision. The seasons of the vehicle crashes were noted: summer (December–February), autumn (March–May), winter (June–August) and spring (September–November).

Statistical analyses were performed using a Fisher's exact test.

Results

Over the five years of the study, 286 motor vehicle occupant fatalities were recorded on South Australian roads. Of these cases, 228 presented for coronial autopsy and 185 were suitable for analysis. Over half of the occupants were wearing a seat belt (134 [72.4%]) and of these 44 (32.8%) had seat belt markings across either the chest, the abdomen, or both. No significant differences were found in the incidence of seat belt wearing over the seasons: summer $N = 37$ (28%), autumn $N = 28$ (21%), winter $N = 32$ (24%) and spring $N = 37$ (28%). There was, however, considerable variation in the occurrence of cutaneous seat belt markings over the seasons: summer $N = 16$ (36%), autumn $N = 9$ (20%), winter $N = 4$ (9.1%) and spring $N = 15$ (34%) (Figure 1). Fisher's exact test confirmed a significant difference ($p = 0.027$).

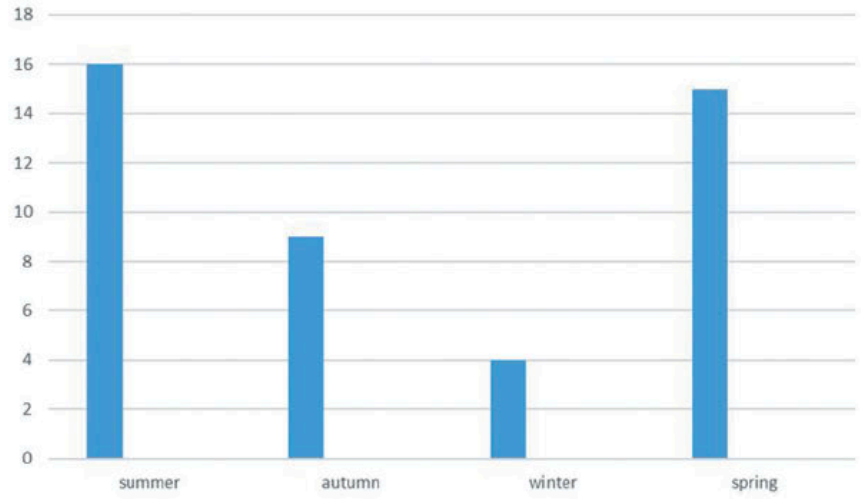

Figure 1. Number of cases with cutaneous seat belt markings per season in a cohort of 44 occupants killed in vehicle crashes in South Australia from January 2014 to December 2018 (summer: $N = 16$; 36%; autumn: $N = 9$, 20%; winter: $N = 4$; 9.1%; spring: $N = 15$; 34%).

Discussion

Seat belt injuries per se may range from superficial abrasions and bruises to fractures and life-threatening disruption of internal organs [1,5–7]. The finding of cutaneous markings from seat belts has been termed the 'seat belt sign' [8] and consists of linear bruises and abrasions corresponding to the seat belt position at the time of impact. After death, linear brown parchmenting may be seen as the abraded skin desiccates.

In this study, only a minority of those wearing seat belts had cutaneous markings (44/134 = 32.8%) that were present across either the chest, the abdomen, or both. Analysis of the seasonal variation in the occurrence of markings showed that only 9.1% of those wearing seat belts in winter had marks, compared with 36% in summer ($p = 0.027$). The reasons for this are unclear; however, the absence of markings from seat belts at autopsy is often attributed by forensic pathologists to the effects of padding by clothing, but data are lacking.

The average seasonal temperatures for Adelaide, South Australia are 16.7–28.6°C in summer (December–February), 12.7–22.7°C in autumn (March–May), 8–16°C in winter (June–August) and 11.8–22°C in spring (September–November) [9]. In 2020–2021 the hottest day occurred at Moomba Airport (46.6°C) in summer and the coldest day at Gluepot Reserve in winter (–6.3°C) [10]. Given that thicker and warmer clothing is likely to be worn in colder months it might be anticipated that seat belt markings would be less common at this time (i.e. winter) if the interposition of clothing is a significant factor in preventing cutaneous seat belt trauma.

An additional factor that may be operating in the colder months may be reduction in the speed of vehicles during inclement weather, resulting in less force of impact in the event of a crash, as higher speed crashes are associated with increased seat belt trauma [11].

In conclusion, this study has shown that significantly fewer cutaneous marks from seat belts occur in winter months. This may be associated with either slower speed of impact or greater amounts of clothing being worn. As seat belt markings may be crucial in some cases at autopsy to clarify the deceased's positioning in the vehicle to assist with police investigations and to clarify possible medico-legal issues, further studies will be required to clarify the aetiology of this seasonal trend and could incorporate other injuries sustained in these crashes, which might strengthen such conclusions. For example, injuries from the steering wheel, the main pillars of the vehicle and the windows.

Disclosure statement

No potential conflict of interest was reported by the authors.

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5.10 Publication: Lethal mechanisms in cases of inverted suspension from lap component of seat belts

S. O'Donovan, N.E.I Langlois, C. Van Den Heuvel, R.W. Byard

Seat belts are designed to restrain the occupant during a collision, reducing risk of ejection and occupant movement within the vehicle during impact. However, a number of cases were identified during data capture of vehicle roll-overs in which the driver was suspended upside down by the lap component of the seat belt and the driver's neck was angled down toward the chest, restricting the upper airway. A case report of the five cases identified with similar crash circumstances was published to highlight these unusual seat belt-associated fatalities.

Statement of Authorship

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Publication Details	O'Donovan, S, Langlois, NEI, Van Den Heuvel, C & Byard, RW 2021, 'Lethal mechanisms in cases of inverted suspension from the lap component of seat belts', *Medicine, Science and the Law*, vol. 61, no. 3, pp. 227-231 DOI: 10.1177/0025802421993990.

Principal Author

Name of Principal Author (Candidate)	Siobhan O'Donovan		
Contribution to the Paper	Wrote manuscript		
Overall percentage (%)	85%		
Certification:	This paper reports on original research I conducted during the period of my Higher Degree by Research candidature and is not subject to any obligations or contractual agreements with a third party that would constrain its inclusion in this thesis. I am the primary author of this paper.		
Signature		Date	27/05/2022

Co-Author Contributions

By signing the Statement of Authorship, each author certifies that:

i. the candidate's stated contribution to the publication is accurate (as detailed above);

ii. permission is granted for the candidate in include the publication in the thesis; and

iii. the sum of all co-author contributions is equal to 100% less the candidate's stated contribution.

Name of Co-Author	Neil Langlois		
Contribution to the Paper	Contributed case material; helped to evaluate and edit the manuscript;		
Signature		Date	22 June 2022

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Contribution to the Paper	Primary supervisor of Siobhan, helped to evaluate and edit the manuscript;		
Signature		Date	04/07/2022

Name of Co-Author	Roger Byard		
Contribution to the Paper	Co-supervisor of Siobhan, helped to evaluate and edit the manuscript, acted as corresponding author		
Signature		Date	08/07/2022

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Lethal mechanisms in cases of inverted suspension from the lap component of seat belts

Siobhan O'Donovan[1,2], Neil El Langlois[1,2], Corinna van den Heuvel[1] and Roger W Byard[1,2]

Abstract

A retrospective review of autopsy files at Forensic Science South Australia in Adelaide, Australia, was undertaken over a five-year period from January 2014 to December 2018 for all motor vehicle crashes with rollovers ending with the vehicle inverted and the occupants suspended by the lap component of their seat belts. There were five cases, all male drivers (aged 18–67 years; $M_{age} = 32$ years). Acute neck flexion or head wedging was noted in four cases, with facial petechiae in four and facial congestion in one. Deaths were due to positional asphyxia in four cases, with the combined effects of positional asphyxia and head trauma accounting for the remaining case. Although all drivers had evidence of head impact which may have caused incapacitation, in only one case was this considered severe enough to have contributed to death. A blood alcohol level above the legal limit for driving was detected in two cases, but no other drugs were detected. This series demonstrates another subset of cases of seat belt–associated deaths where suspension upside down by the lap component of a seat belt had occurred after vehicle rollovers. Predisposing factors include incapacitation of the victim and delay in rescue. The postulated lethal mechanism involved respiratory compromise from the weight of abdominal viscera on the diaphragm, as well as upper airway compromise due to kinking of the neck and wedging of the head.

Keywords

Lap seat belt, vehicle rollover, suspension, positional asphyxia, death

Introduction

Seat belts are designed to produce a more gradual deceleration of vehicle occupants and to distribute collision forces more evenly in addition to preventing ejection from a vehicle and impact with the steering wheel or dashboard.[1,2] Their use has clearly reduced both morbidity and mortality from vehicle crashes by an estimated 25%–53%, with more than 50,000 lives saved in the UK alone since mandatory wearing was legislated.[3,4] Occasional situations occur, however, where seat belts have resulted in either death or injuries involving vascular, abdominal, thoracic and spinal trauma.[5,6] This is most likely to occur if seat belts have not been properly positioned, resulting in a 'compromised occupant–seat belt relationship'.[1]

More rarely, seat belts may be associated with airway compromise, most often involving neck compression due to hanging from shoulder seat belts.[7,8] The following series of cases demonstrate a unique situation where deaths occurred following vehicle rollovers with abdominal suspension of occupants from the lap component of their seat belts. The types of possible mechanisms contributing to death are analysed.

Methods

A retrospective review of autopsy files at Forensic Science South Australia in Adelaide, Australia, was undertaken over a five-year period from January 2014 to December 2018 for all motor vehicle crashes

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where vehicle occupants had been found suspended from the lap component of their seat belts following vehicle rollovers. The de-identified files were reviewed, and the age, sex, body mass index (BMI), injuries, findings at autopsy and details of the crash were recorded.

Case reports

Five cases were found out of a total of 231 vehicle crash autopsy cases (2.2%).

Case 1. An 18-year-old male driver and sole vehicle occupant failed to negotiate a bend on a highway, resulting in his vehicle leaving the road, overturning and impacting against a large tree, finally coming to rest on its roof. He was found inside the vehicle suspended by the lap component of his seat belt in an inverted position with his neck acutely flexed to the side (Figure 1). At autopsy, there was evidence of non-lethal blunt cranio-cerebral trauma with haemorrhage into the mid-corpus callosum and subdural haemorrhage over the cervical spine with haemorrhage into the posterior spinal ligament. Florid facial petechiae were present. The BMI was 24 kg/m^2, and toxicological testing for alcohol and common drugs was negative. Death was due to positional asphyxia.

Case 2. A 20-year-old male driver and sole vehicle occupant lost control of his vehicle on a gravel highway verge, resulting in the vehicle overturning. It came to rest on its roof. He was found inside the vehicle suspended by the lap component of his seat belt in an inverted position with his head wedged between the roof and road surface (Figure 2). At autopsy, the major findings were severe blunt cranio-cerebral trauma with an open complex fracture of the skull with subarachnoid and intraventricular haemorrhage. The face showed marked congestion with florid petechiae. The BMI was 25 kg/m^2, and toxicological testing for alcohol showed a blood alcohol level of 0.18%, with no common drugs detected. Death was due to blunt cranio-cerebral trauma complicated by positional asphyxia.

Case 3. A 67-year-old male driver and sole vehicle occupant lost control of his vehicle, causing it to leave the road, overturn and impact against a large tree. The vehicle came to rest on its roof. He was found inside the compressed cabin suspended by the lap component of his seat belt in an inverted position with his neck acutely flexed to the side (Figure 3). At autopsy, there was bruising of the right side of the scalp, with no significant cranio-cerebral injuries. The face was congested but with no petechiae. The BMI was 29 kg/m^2, and toxicological testing revealed a blood alcohol level of 0.079%, with no common drugs detected. Given the position of the body and the absence of significant injuries, death was attributed to positional asphyxia.

Case 4. A 35-year-old male driver and sole vehicle occupant lost control of his vehicle, resulting in his vehicle leaving the road and overturning. It came to rest on its roof. He was found inside the cabin suspended by the lap component of his seat belt in an inverted position with his head wedged between the door frame and ground. At autopsy, there was minor

Figure 1. An 18-year-old male driver and sole vehicle occupant found inside his overturned vehicle suspended by his seat belt in an inverted position with his neck acutely flexed to the side (arrow).

Figure 2. A 20-year-old male driver and sole vehicle occupant found inside his overturned vehicle suspended by his seat belt in an inverted position with his head wedged between the roof and road surface. (Unfortunately, due to the angle of the photograph and the position of the body, it is not *possible* to visualise the head.)

Figure 3. A 67-year-old male driver and sole vehicle occupant found inside his overturned vehicle suspended by his seat belt in an inverted position with his neck acutely flexed to the side (arrow).

bruising of the face and scalp, with no significant cranio-cerebral injuries. The chest, neck and face were congested with numerous petechial haemorrhages. The BMI was 26 kg/m^2, and toxicological testing revealed a non-toxic concentration of diazepam and tetrahydrocannabinol. Alcohol and other common drugs were not detected. Death was due to positional asphyxia.

Case 5. A 21-year-old male driver lost control of his vehicle. It left the road, collided with trees and overturned, coming to rest on its roof. He was found trapped inside the cabin of the vehicle suspended by the lap component of his seat belt in an inverted position. At autopsy, there was bruising of midline scalp with a minor left subdural haemorrhage, but there were no other significant cranio-cerebral injuries. Petechial haemorrhages were noted on the eyelids and conjunctivae. The BMI was 34 kg/m^2 (obese), and toxicological testing revealed no alcohol or common drugs. Death was due to positional asphyxia.

All these events occurred on country roads. Four of the decedents were dead by the time ambulance personnel arrived; the fifth was entrapped and required some time to extricate. None of the decedents had underlying organic conditions that could have caused or contributed to death.

Discussion

Life-threatening injuries caused by seat belts may involve the abdomen, with intestinal perforation, solid organ disruption or aortic laceration/transection (particularly if there is atherosclerotic calcification), or the thorax, with vascular or pulmonary injuries.[2] This is particularly so in children who may not have seat belts or booster seats properly fitted.[9,10] Linear external bruising in the pattern of a seat belt (the 'seat belt sign') may be a marker of significant internal injuries to the thorax (the so-called seat belt syndrome).[3] Neck trauma from seat belts may lead to lethal cervical fractures or vascular injury, particularly if there has been submarining under the belt,[11] or to rapid death from carotid compression.[10,12]

Asphyxiation from vehicle crashes may result from a variety of mechanisms. In rollover crashes, occupants may be pinned underneath vehicles and die of crush asphyxia.[13] This sometimes may involve occlusion of the airways if the face is pushed or held into soft soil or sand.[14] Compression asphyxia may also occur within the cabin of a vehicle if unconscious, intoxicated or injured occupants are lying on top of each other.[15] Alternatively, unconscious or incapacitated individuals have died following neck compression from shoulder seat belts around the neck.[11,16]

In the present series, incapacitated individuals were suspended upside down from the lap component of seat belts in vehicles resting on their roofs in a characteristic position demonstrated in Figures 1–3. All victims were male drivers aged between 18 and 67 years ($M_{age} = 32$ years). Acute neck flexion or head wedging was noted in four cases, with facial petechiae in four and facial congestion in the remainder. Deaths were due to positional asphyxia in four cases, with the combined effects of positional asphyxia and head trauma accounting for the remaining case. All of the crashes occurred on country roads that would have resulted in delays in the arrival of ambulance personnel.

Death has been well described in an inverted position, although the mechanisms are not necessarily clear. It is likely that chest excursion and pulmonary expansion are compromised by the weight of the viscera on the diaphragm, possibly associated with respiratory muscle fatigue, particularly in the obese.[17] In the current study, however, only one of the five cases had an elevated BMI (34 kg/m^2). Cardiovascular changes also occur with alterations in blood pressure and possible myocardial hypoxia. It has also been suggested that increased venous pressure in the head may also result in reduction of cerebral blood flow.[18–21]

Cases of death due to suspension from inversion following vehicle rollovers are very rare[15] and comprised a small proportion (2.2%) of motor vehicle crash deaths at our centre. Requirements for such a death include a vehicle rollover with the vehicle coming to rest on its roof. A further feature is use of a seat belt so that the lap component performs its restraining function, causing the occupant to be suspended in an inverted position in their seat while incapacitated. Delayed rescue provides time for death to result from asphyxia while in the inverted position (Table 1). Although the major

Table 1. Requirements for lethal inverted suspension from the lap component of a seat belt.

1. Vehicle rollover resulting in the vehicle coming to rest on its roof
2. Wearing and preservation of a fitted lap seat belt harness
3. Suspension of the victim upside down by the lap component of the seat belt
4. Incapacitation by drugs, alcohol and/or injury
5. Delay in, or failure of, rescue

Table 2. Lethal mechanisms in cases of inverted lap seat belt suspension.

1. Underlying injury
2. Suspension and positional asphyxia involving
a. visceral compression
b. airway obstruction
3. Drug/alcohol effects
4. Combinations of factors

emphasis has been on the effects of suspension on cardiorespiratory control, the current cases also clearly show that sharp angulation of the neck or wedging of the head are additional factors that should be considered contributory to asphyxia.[22] Thus, deaths in these circumstances may be due to underlying injuries, positional asphyxia with visceral compression and/or airway obstruction, drug/alcohol effects or a combination of these factors (Table 2).

In conclusion, this series illustrates an unusual situation where suspension upside down by the lap component of seat belts resulted in deaths following vehicle rollovers. Predisposing factors included incapacitation of the victim and delay in rescue, with lethal mechanisms involving lung compression by abdominal viscera, as well as upper airway compromise due to kinking of the neck and wedging of the head. These cases form yet another subset of cases of seat belt–associated death. The scene photographs in the reported cases provide an excellent demonstration of exactly how this position can compromise respiration.

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5.11 Publication: Injuries, death and vehicle airbag deployment

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TARS does not record if a vehicle had an airbag fitted or if an airbag was deployed. Airbags, in combination with seat belts, unequivocally decrease the severity of injury and reduce mortality in vehicle crashes. There are, however, reported cases of airbags resulting in fatal injury, usually from airbag malfunction. Although airbags were not able to be assessed in this thesis, a short review of lethal airbag injury was conducted as part of this thesis.

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Principal Author

Name of Principal Author (Candidate)	Siobhan O'Donovan		
Contribution to the Paper	Performed literature review, wrote manuscript		
Overall percentage (%)	85%		
Certification:	This paper reports on original research I conducted during the period of my Higher Degree by Research candidature and is not subject to any obligations or contractual agreements with a third party that would constrain its inclusion in this thesis. I am the primary author of this paper.		
Signature		Date	27/05/2022

Co-Author Contributions

By signing the Statement of Authorship, each author certifies that:

- the candidate's stated contribution to the publication is accurate (as detailed above);
- permission is granted for the candidate in include the publication in the thesis; and
- the sum of all co-author contributions is equal to 100% less the candidate's stated contribution.

Name of Co-Author	Corinna Van Den Heuvel		
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Signature		Date	27/5/2022

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Name of Co-Author	Roger Byard		
Contribution to the Paper	Co-supervisor of Siobhan, helped to evaluate and edit the manuscript, acted as corresponding author		
Signature		Date	08/07/2022

Viewpoint

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Abstract

Airbags are impact-activated safety devices which deploy from the interior of vehicles to protect occupants from trauma during crashes. Although airbags effectively reduce the risk of death and injury, this it is not without issues. For example, high-impact unbelted rigid-barrier testing in the USA led to the adoption of powerful, large airbags that were associated with numerous airbag-related deaths and injuries. In contrast, European designs were tested and certified in conjunction with the use of three-point restraint systems, meaning that the airbags could be smaller with reduced 'punch-out' power. An overview is provided of the mechanism of action of airbags and the associated non-lethal and lethal injuries that may be sustained by vehicle occupants.

Keywords

Airbags, injury, death, mechanism

Introduction

Airbags work by cushioning impact and controlling the force of impact of occupants against internal vehicle structures. They deploy within 10 ms of impact, utilising an exothermic chemical reaction that produces hot gas that rapidly inflates the bag.[1] This creates a cushioned impact point, as well as absorbing energy and decelerating the occupant. The area protected from injury by airbags ranges from just the torso to both the head and torso. Head-and-torso side airbags have reduced the risk of death by almost 50%,[2] and so airbags are now mandatory in all new vehicles in Europe, the USA and Australia.[3]

Critically, the time between crash and occupant impact with internal structures is brief, and prior to airbag implementation, occupants would collide with extreme force into the dashboard, windshield and steering wheel, often resulting in severe head and chest injuries.[4] Airbags have decreased the severity of head injury during frontal collisions.[5] Side airbags are more commonly used in combination with front airbags, and they deploy from either the side pillar or the outer occupant seat.[2] Side airbags are, however, constrained during deployment, with limited expansion space given the narrowness of the space between the seat, occupant and side car door.

Mechanisms of injury

Although the trauma reduction associated with airbags has been well established, airbags can cause hyperextension of the neck, resulting in atlanto-occipital fractures, thoracic injuries or aortic dissection, all of which have proven fatal.[3,6,7] Injuries such as these occur in the second phase of airbag deployment when the inflating airbag has pushed body parts away from the sternum.[8] These injuries have been associated with spontaneous, unwarranted, mistimed or excessive force during deployment where the airbag action has caused greater injury than the impact otherwise would have.[5,7]

A higher risk of injury is also associated with particular positions and manners of occupant seating,[8] for example in unrestrained or improperly restrained occupants, especially during low-speed collisions

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where the airbag punch-out force is disproportionate to the collision force.[3,5] Additional compounding factors include driving smaller vehicles and sustaining greater impacts.[3] A difficulty in attempting to determine the overall success of airbags previously was in determining how many vehicles were in fact fitted with them.[2,5]

Children and occupants of shorter stature are more often seriously injured by airbags due to their relative position to the airbag trajectory.[7] Airbags are designed to deploy outward from the steering wheel with the direct force focused on the sternum. The improper application of force in deployment due to the difference in position has led to more atlanto-occipital, cervical and thoracic spinal fractures which is further complicated by increasing age with its associated physical vulnerabilities.[4,7] Hoye also suggested that women are at higher risk given that they are generally of shorter stature and thus sit closer to the steering wheel and dashboard.[3]

Defective airbags

Cases of defective airbags have also been reported. These have exploded on deployment, launching metal projectiles towards the occupant's head and neck. In one such case, fragments penetrated an occupant's neck, causing lacerations and transection of major neck structures, including the trachea, right carotid artery and part of the cervical vertebrae. The crash itself was not considered to have been capable of causing serious injury, and so the fatality was exclusively the result of airbag malfunction.[4] This case has been inquested and is part of a global recall of Takata airbags.[9,10]

Fatalities

The highest number of fatalities caused by airbags have been in the USA, mainly due to first- and second-generation airbags being substantially larger (70 L) and more powerful compared to western European 30 L models.[3,7] By January 2009, 296 deaths in the USA were contributed to by front airbags, which included 191 children, 92 drivers and 13 adult passengers.[11] Initial testing methods for airbags in the USA that utilised an unbelted rigid-barrier test with high-impact velocity led to first-generation airbags being extremely large and powerful.[3] In 1997, crash test conditions were changed to use a 25 mph sled test,[3] which resulted in successive decreases in fatality numbers, although this was also influenced by an increase in restraint wearing and an overall improvement in road-safety awareness. The original significantly more powerful US airbags that had been designed for a higher collision performance meant that belted occupants involved in low-speed collisions were being exposed to high-energy inflations, resulting in numerous unnecessary injuries and fatalities. Whilst the European design was modelled around belted low-speed collisions, these countries are not without airbag fatalities, with the first recorded case occurring in the UK in 2000.[7]

The balance between collision force and airbag-deployment force remains contentious. For example, while smaller lower-powered airbags reduce airbag-related injury in low-speed collisions, they may offer little protection in high-impact collisions. Conversely, high-powered airbags may cause injuries that otherwise would not have occurred during collisions at lower velocity.[3] The newest generation of airbags include collision force and occupant weight sensors to mediate the deployment power.

Non-lethal injuries

Non-lethal injuries may also be associated with airbag deployment and inflation. These injuries often occur in the head and neck region as well as to the upper limbs, and include facial fractures, lacerations, contusions, otologic injury such as tympanic membrane rupture and ocular injuries such as a ruptured globes and retinal haemorrhage.[4,7] The pattern of injuries has changed with each generation of airbag, with occupants being less likely to sustain facial or cranio-cerebral trauma from third-generation devices.[3,12] Injuries can also be caused by chemical burns from corrosive substances and by thermal burns from contact with airbag exhaust vents.[1] It is also recognised that seat belts may contribute to other injuries, including vascular transection, airway compromise and fractures.[13–15]

Conclusion

Airbags are an integral part of the safety system within modern vehicles and, when operating correctly, can not only save lives but also reduce injury. However, as with any safety device, misuse or incorrect performance may instead lead to injury. Furthermore, when significant forces are involved in a crash, the forces may surpass the operating ability of the safety device.[3] Thus, the autopsy assessment of vehicle crash victims should be conducted with an appreciation and understanding of the mechanism of action of vehicle airbags and an awareness of the range of associated lethal and non-lethal injuries that may occur.

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Chapter Six: Data analysis and results

6.1 Introduction

The following chapter presents broader analysis exploring fatal injury patterns using the combined TARS and fatal injury dataset. Previous chapters have outlined the research methodology used to establish the combined dataset (Chapter 2), discussed relevant literature on occupant and crash factors known to contribute to fatal outcomes (Chapters 3-5) and presented several publications that use information from the combined dataset (Chapters 3-5). This chapter explores several other research questions that are specifically related to the patterns of fatal injury and explores the relationships that may exist between injury patterns, occupant characteristics and crash factors. This chapter also includes an overview of the overall data capture from coronial autopsy reports and descriptive summaries of causes of death and types of injury. Two publications are included that have been derived from the larger dataset are included in this chapter.

6.2 Statistical Analysis

All statistical analyses were performed using R studio statistical software (version 1.4) (R Development Core Team) as mentioned in Chapter 2.5. Various statistical modelling approaches were used within this chapter such as linear regression, logistic regression, and decision trees ($\alpha = 0.05$). For some of the statistical analyses, a training and testing dataset were portioned from the original dataset to provide the opportunity for model validation and accuracy testing. All training datasets are 60% of sample data and testing datasets are 40% (as outlined in Chapter One). Individual cases were omitted during each statistical analysis if there were missing data within the factors included in the analysis. The sample of data used in each statistical analysis, including the predictor variables selected for analysis, number of cases omitted from the dataset and final sample size, is reported in Appendix C. A suite of packages were used in R, the details of which are provided in Appendix C.

6.3 Data capture and cross-match

A total of 3641 occupant fatalities were identified in TARS from 1981 to 2020 in South Australia. The black line in Figure 1 shows the total number of occupant fatalities by year. There was an overall downward trend in occupant fatalities during the study period. Following a search of FSSA and Coroner's files, 2800 occupant fatalities were identified and cross matched to cases in TARS. The data capture rate across the 40 years was 76%. The grey line in Figure 1 describes the total number of occupant fatalities that underwent autopsy by year from 1981 to 2020.

The rate of data capture fluctuates throughout the time frame, with periods of high data capture particularly between 1985 and 1995 and between 2006 and 2010. Unfortunately, due to the retrospective nature of data collection, deidentification of the TARS database and, in some cases, no autopsy being performed, complete data capture was highly unlikely. There are, however, distinct periods in which data capture was lower than expected. The first period between 1981 and 1984 (denoted by the dot dash line in Figure 1) had less than 50% data capture (see Appendix C, Table C.1). This was largely due to missing information in the autopsy reports regarding mechanism of death. Cases were not cross-matched (and therefore considered unidentified in autopsy reports) unless the autopsy report provided clear indication of a vehicle crash or supplementary mortuary records (not available before 1985) indicated that the mechanism of death was a vehicle crash. Although there were cases that appeared to match for age and sex (crash date and autopsy date were usually two to three days apart), the reported cause of death was not always an indicator of a vehicle crash, as 'blunt force trauma' can occur as the result of other high energy mechanisms (e.g., pedestrian crash, aeroplane crash etc). Furthermore, other causes of death such as drowning, incineration or complications following a crash were even more difficult to determine for a potential TARS occupant from earlier autopsy reports.

The period between 2000 and 2002, indicated by the dotted lines in Figure 1, had a noticeable decline in autopsies captured. During this period, FSSA was not the primary facility for medico-legal autopsies, and it is likely that a large portion of the decline in capture was due to autopsy reports being located at other facilities.

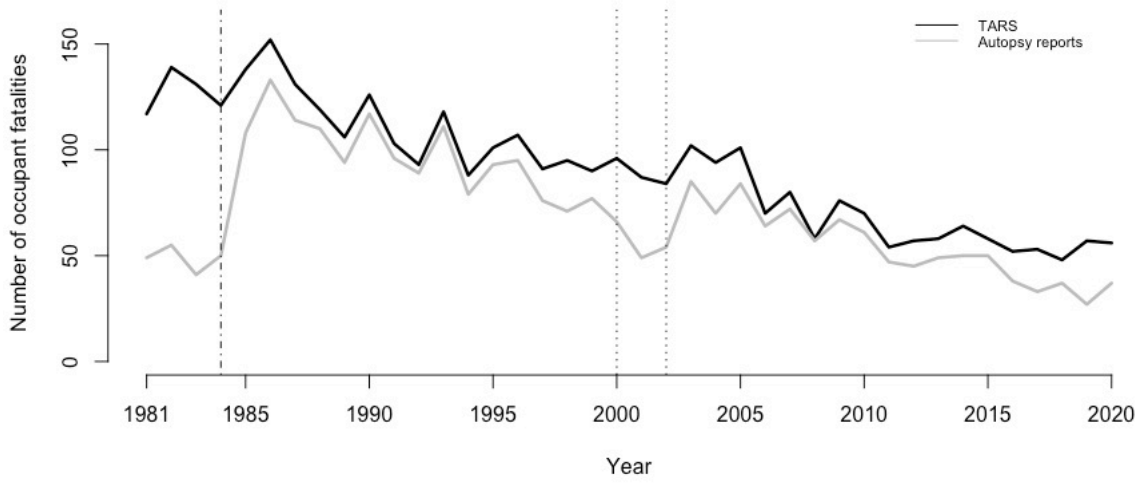

Figure 6.1 Number of occupant fatalities between 1981 and 2020 in South Australia as reported by TARS and identified in coronial autopsy reports at Forensic Science SA

There are three categories of autopsy with varying degrees of internal examination, all undertaken with the aim of documenting injuries and determining the cause of death. These three categories are full forensic autopsy, computer topography (CT) scans or external only examination. Full forensic autopsy involves a stepwise internal examination of the body by a forensic pathologist with documentation of all injuries, distinguishing features, evaluation of organs and detection of underlying factors or disease, as well as ante-mortem and post-mortem artefacts. More recently, CT scans have been used in conjunction with, or in preference to, a full forensic autopsy. External only examination is rare in victims of MVCs; however, several cases were identified in the data capture. Often the cause of death in these cases is obvious or a clinical diagnosis is made which is then supported by the pathologist and comments on the external injuries are made.

Despite the increase in minimally invasive methods of examination, the overall capture rate of data is still declining. Part of this decline may be related to the increased use of pathology review. Pathology review occurs usually when an occupant has survived to hospital, received a clinical diagnosis, and subsequently passed away. As the cause of death is attributable to the clinical diagnosis, the pathologist reviews the clinical finding without undertaking any form of forensic examination. While this process reduces medicolegal workload, particularly in cases where the cause of death is apparent, it does limit the amount of information available for future research.

6.4 Summary of occupant age and sex

Of the 2,800 occupant fatalities identified from 1981 to 2020, there were significantly more male occupant fatalities (68.3%, n = 1913) than females (31.7%, n = 887). The proportion of male occupant fatalities compared to female fatalities has remained relatively consistent across the study time frame aside from the year 1999 when the proportions of male and female occupant fatalities were equal (Figure 6.2).

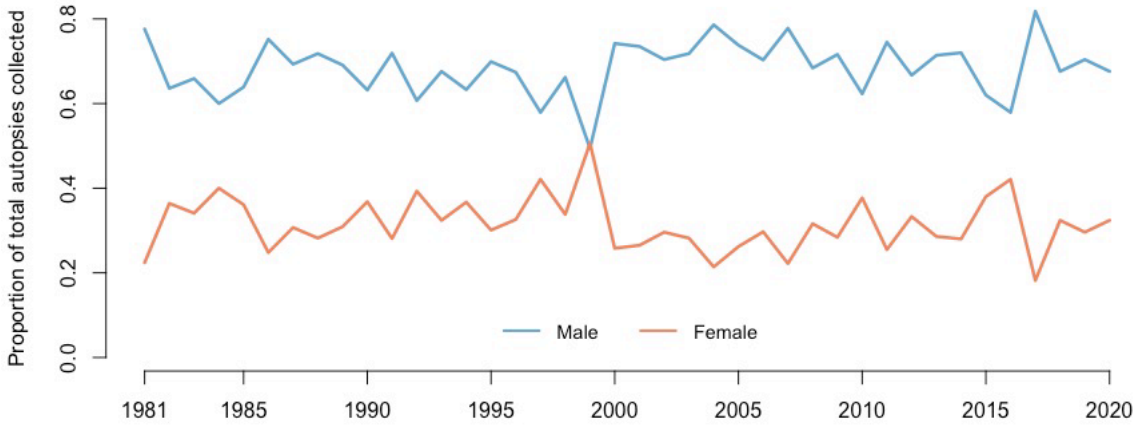

Figure 6.2 Occupant fatalities by proportion of male to female fatalities

The distribution and frequency of fatalities according to age group did change across the study time period. Figure 6.3 shows the frequency of fatalities by age group from 1981 to 2020. The 18-24 year old and 25-44 year old age categories are distinctly higher in the earlier

years compared to the 45-64 year old and 65+ year age groups but begin to trend downwards entering the 2000s. Interestingly, the 45-64 year old and 65+ year old age categories consistently recorded similar numbers of fatalities across the study time frame. This could indicate that road safety countermeasures introduced across the study period, and which targeted fatal crashes may have been more effective in preventing fatal crashes in younger occupants than older occupants. Younger occupants are more likely to be involved in crashes (as highlighted in Chapter Four) due to their propensity to engage in risky driving behaviours, particularly young male drivers. Many countermeasures for fatal crashes target

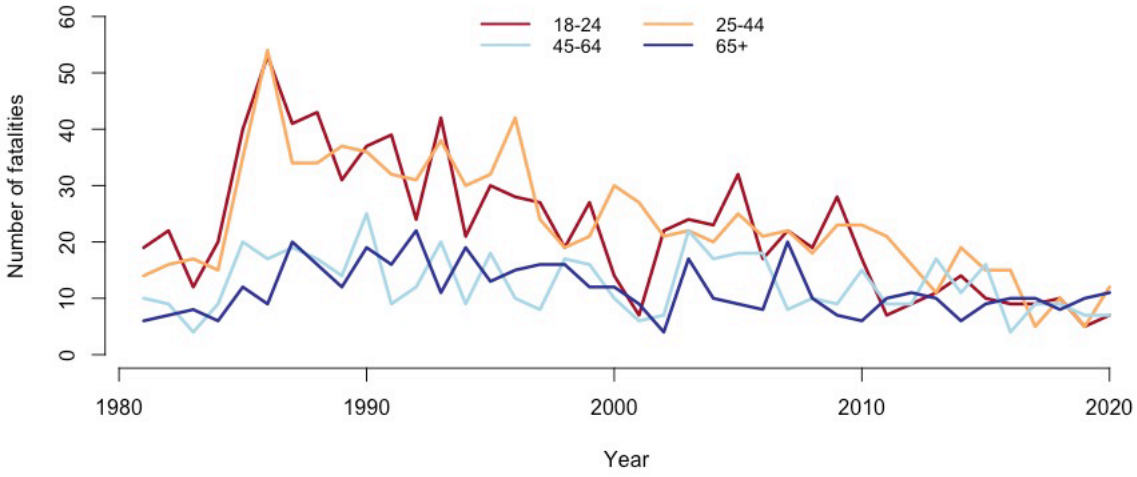

Figure 6.3 Number of occupant fatality by age group

risk taking behaviour factors (the fatal five, as discussed in Chapter Three) and so may effectively target young occupants. Another contributing factor to the reduction in the proportion of younger occupant fatalities is the aging population, as the proportion of licensed drivers in the older age categories increases, so too would the proportion of older driver fatalities (relative to younger drivers) (Baldock et al. 2016).

As discussed in Chapter Four, there is an interaction between age and sex which affects the likelihood of fatality, with higher risks for younger male drivers and older female occupants. To explore the relationship between age and sex in this cohort, a logistic regression was performed. Sex was a binary independent variable with 0 = female and 1 =

male. Odds ratios (OR) and corresponding 95% confidence intervals (95% CI) were calculated. The overall regression was statistically significant ($p < 0.001$, OR= 0.98, 95% CI = 0.98, 0.99). Figure 6.4 describes the relationship between age and sex, with the probability of an occupant fatality being male being greater in younger years. As age increases, there is an increasing likelihood of fatalities being female. For full model output, please see Appendix C.2.

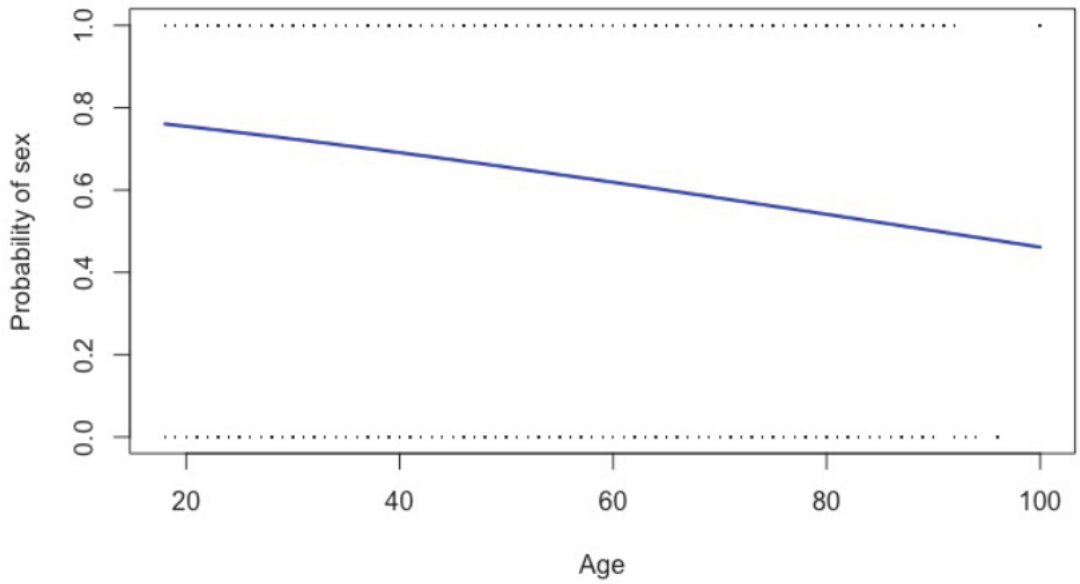

Figure 6.4 Fitted logistic regression line (blue) for the relationship between occupant age and sex, where 0 = Female and 1 = Male

6.5 Causes of death over time

To address the over-arching research question relating to the patterns of fatal injury over time, an analysis of the causes of death and how these have changed with time was undertaken. The most common cause of death between 1981 and 2020 was multiple blunt force injury (39.7% of fatalities), followed by closed head injury with skull fracture (20.3%) and blunt chest injury (16.9%) (Table 6.1).

Table 6.1 Frequency of occupant causes of death

Cause of death	Number of fatalities *n, (%)*
Multiple blunt force	1108 (39.7)
Closed head injury (skull fracture)	568 (20.3)
Blunt chest injury	475 (16.9)
Spinal injury	144 (5.1)
Closed head injury (no skull fracture)	123 (4.4)
Incineration	120 (4.3)
Other	72 (2.6)
Abdominal injury	71 (2.5)
Open head injury	59 (2.1)
Complication	39 (1.4)
Exsanguination	15 (0.5)
Pelvis/limb injury	6 (0.2)

Figure 6.5 describes the three most common causes of death and how these categories have changed over time. Multiple blunt force injury became the dominant cause of death from the early 2000s, while closed head injury with skull fracture and blunt chest injury were consistently attributed between 1981 and 2000 before an apparent downward trend thereafter. This apparent downward trend in these causes of death being coincident with the marked increase in deaths attributed to multiple blunt force injury suggests that closed head injuries with skull fractures and blunt chest injury are no longer occurring in isolation but, rather, are occurring with other blunt force trauma of equal lethality.

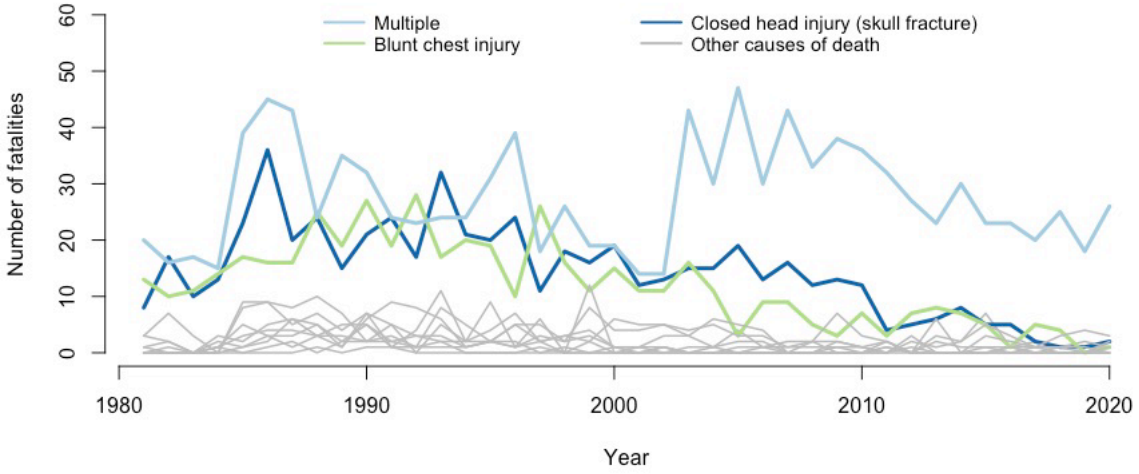

Figure 6.5 Number of occupant fatalities by cause of death from 1981 to 2020

Changes in reported causes of death may also reflect a shift in the nomenclature or categorisation of injuries by pathologists. The trends identified post-2000 in Figure 6.5 may be occurring because pathologists are making less distinction between the isolated injuries as a primary cause and considering death in the presence of other injuries as a cumulative result of multiple injuries.

The rise in deaths attributable to multiple trauma may also be an unintended result of improved vehicle safety. The introduction of seat belts and airbags into vehicles markedly decreased the injury severity and mortality associated with vehicle crashes, especially head injuries and chest injuries. The decline in isolated head and chest trauma may be due to this improved safety, and, as motor vehicle become safer, the level of crash impact severity that must occur to produce fatal injuries increases. Advances in emergency response and trauma medicine may also be inadvertently contributing to the increased incidence of multiple trauma among fatalities due to a decreased risk of fatality from isolated injuries, particularly head injuries. As emergency care has improved, the interval between time of crash and medical assistance has decreased, reducing the mortality associated with some injuries, particularly those injuries with the potential for significant blood loss. Advances in medical care, especially in the treatment of traumatic brain injuries, may mitigate the lethal sequalae

of trauma particularly when the occupant has sustained an isolated life-threatening injury. Although this possible explanation for the results shown in Figure 6.5 cannot be investigated using only fatal crashes, it provides a possible avenue for future research to combine fatal injury information with minor and serious injury crash data to determine if fatal crashes now typically involve high impact severity resulting in multiple injuries more than crashes resulting in serious injury.

Causes of death such as spinal injury, closed head injury without skull fracture or abdominal injury did not change dramatically over the study time frame. Additionally, other causes of death such as pelvic/limb injury and complications occurred so infrequently that there is no clear trend over time. Several causes of death that are the result of exceptional circumstances or external factors that distinguish them from other causes of death include incineration, types of asphyxia, complications, drowning, decapitation, and crush injury. As these causes of death are unique, the following sections provide a summary of cases involving these causes of death. These cases will be omitted during further statistical analyses as the injury patterns or mechanisms of injury differ from the majority of blunt force trauma injuries and could therefore confound statistical modelling.

6.5.2 Incineration

Occupant fatalities involving incineration are relatively uncommon, with 4.3% of fatalities between 1981 and 2020 involving occupant incineration post-collision. Figure 6.6 shows the number of fatal incineration cases over the study time frame. Although there appears to be fewer deaths attributed to incineration in the earlier years, this could be a feature of incomplete data capture, as some earlier autopsy reports did not include information that

would indicate a vehicle crash and, therefore, fire-related causes of death were not obviously related to a vehicle crash.

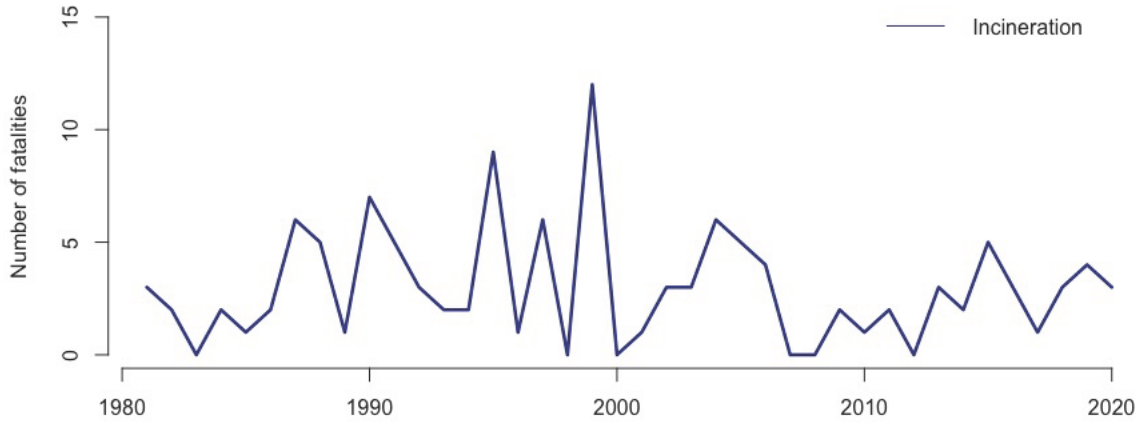

Figure 6.6 Number of fatalities involving incineration between 1981 and 2020

A total of 120 occupant fatalities involved incineration; 89 of these were drivers and 31 were passengers. The average age was 40 years old (range = 18-90 years) and were predominantly males (M:F, 86:34). Only 25 (20.8%) occupants were known to be wearing a seat belt and 9 (7.5%) were known to not be wearing a seat belt, with the seat belt wearing status of the remaining 86 (71.7%) occupants unknown or not recorded in TARS. In many cases, it is likely that the fire destroyed relevant evidence regarding seatbelt use. The majority of occupants died at scene (n = 84), with 2 occupants surviving to hospital and succumbing to complications. The location of death for the remaining 34 occupants was unclear. Alcohol was detected in 22 drivers, 60 without and 32 had unknown BAC. The average year of vehicle manufacture was 1988 (ranging from 1965 to 2020).

Although all fatalities recorded in this category reported thermal injuries following a post-collision fire, the primary cause of death was not always specific to the thermal injuries sustained by the occupant. Primary cause of death in these crashes may be related to the blunt force trauma experienced by the occupant, particularly with head injuries or cardiovascular trauma that would lead to almost instantaneous death. Cause of death may also be related to

inhalation of carbon monoxide produced by the fire. This highlights another complexity of incineration fatalities: the occupant may be incapacitated by blunt force trauma or trapped by the post-crash vehicle position, resulting in the occupant being unable to escape before the vehicle is engulfed in fire. Blunt force injuries sustained by the occupant may sometimes result in fatality prior to the subsequent thermal trauma but the cause of death may not be clear. For these reasons, deaths involving incineration were grouped into a single category. There are also challenges in determining the origin of some injuries, as it may be difficult to distinguish between thermal fracture or blunt force trauma fracture. Thermal injury may also mask traumatic injury to bones or soft tissue. Deaths involving incineration will therefore be removed from further analysis within this thesis as thermal injury may be incorrectly attributed to traumatic injury, or traumatic injury may be mistaken for, or masked by, thermal injury.

6.5.3 Asphyxial deaths

Deaths relating to asphyxia were another infrequent and distinct cause of fatalities. Asphyxial deaths typically do not involve the extensive blunt force injury seen in other crashes and the mechanism of death is often different to other MVC victims. These will therefore not be included in subsequent statistical modelling. Asphyxial deaths were categorised into four main groups and summarised in Table 6.2.

Table 6.2 Summary of asphyxia-related deaths

		Asphyxia, n=32	Alcohol intoxication, n=5	Crush asphyxia, n=13	Postural asphyxia, n=4
		mean, (range)	*mean, (range)*	*mean, (range)*	*mean, (range)*
Age		37 (18 – 68)	28 (18 – 35)	32 (18 – 57)	36 (18 – 67)
BMI		27.1 (17.6 – 36.0)	29.5 (26.3 – 34.4)	27.7 (19.8-45.7)	29.0 (23.6-34.4)
	Unknown	7	2	4	0
		n, (%)	*n, (%)*	*n, (%)*	*n, (%)*
Sex					
	Male	27 (84)	3 (60)	10 (77)	4 (100)
	Female	5 (16)	2 (40)	3 (23)	0 (0)
Seat belt wearing					
	No belt	7 (22)	1 (20)	7 (53.8)	0
	Belt	6 (19)	0	1 (7.7)	3 (75)
	Unknown	2 (6)	0	0	1 (25)
	Not recorded	17 (53)	4 (80)	5 (38.5)	0
Seating position					
	Driver	18 (56)	2 (40)	13 (100)	4 (100)
	Front passenger	8 (25)	0	0	0
	Rear passenger	6 (19)	1 (20)	0	0
	Unknown	0	2 (40)	0	0
Ejection					
	Ejected	2 (6.25)	0	4 (31)	0
	Not Ejected	30 (93.25)	5 (100)	9 (69)	4 (100)

As the risk of asphyxia can be increased by traumatic head injuries that incapacitate the vehicle occupant, the following is a summary of the injuries sustained within each group of asphyxia-related deaths. There were seven deaths (21.9%) attributed to asphyxia which involved skull fractures and one (3.1%) involving an open head injury. Cervical fractures were reported in three cases (9.4%) and thoracic spinal fracture in one (3.1%). Lung contusions and lacerations were reported in three (9.4%) and two (6.3%) cases, respectively, with one or more rib fractures reported in 15 cases (46.9%).

The average blood alcohol concentration for asphyxia related to alcohol intoxication was 0.23 g/100mL. Of these five cases, there was one reported fracture of the right arm and one case with one or more ribs fractured.

Closed head injury without skull fracture was reported in two cases (15.4%) of crush asphyxia, and closed head injury with skull fracture was reported in one case (7.7%). Cervical spinal fracture and lung contusion were reported in one case each (7.7% each) with five cases (38.5%) involving one or more ribs fractured.

There was a single case of postural asphyxia with a closed head injury without skull fracture.

6.5.4 Complications

Deaths due to complications were uncommon (1.4% of cases) but encompassed several specific types, including hypoxic ischaemic encephalopathy, pulmonary oedema, bronchopneumonia, respiratory failure, multi-organ failure, congestive heart failure, myocardial infarction, pericarditis with pulmonary oedema, sepsis and adult respiratory distress syndrome. As outlined in Chapter One, a fatality within 30 days of a motor vehicle is included in TARS, with several of the cases reported as dying of complications having survived for several days post-collision and having received medical intervention. Detailed information regarding the initial injuries the occupant sustained and medical procedures the occupant received are not included in the autopsy report and, therefore, the possibility that some injuries may have been repaired surgically could not be ruled out. Thus, deaths due to complications were excluded from subsequent statistical modelling. The following summaries stratify complications by the specific underlying cause of death.

A 79-year-old male driver experienced a myocardial infarction en route to hospital and was pronounced dead on arrival. He had a BMI of 26.2, had been wearing a belt and was

not intoxicated. It is likely that the rib fractures reported in the autopsy report relate to resuscitation attempts.

Four occupants died of hypoxic ischaemic encephalopathy with an equal number of male and female fatalities (M:F, 2:2). There was a single driver, two front seat passengers and the seating position of one occupant was unknown. The average age was 32 years (range 18-66). The average BMI was 26.4 (range 19.1 – 33.6). Seat belt wearing was reported in two cases, while it was unknown if the other two cases wore a seat belt. Closed head injury without skull fracture, cervical spinal fracture and thoracic spinal fracture were all reported in two cases each. Atlanto-occipital injury was also reported in two cases, with one case of lung contusion and one case of lung laceration. A single case was reported to have had one or more ribs fractured, which may be related to resuscitation attempts.

Five occupants died of pulmonary oedema. Four were drivers, one was a front seat passenger, and they were predominantly male (M:F, 4:1). The average age was 27 years (range (20 - 51) and average BMI was 26.3 (range = 25.6 -27.8). It was unknown if any of the occupants was wearing a seat belt at the time of the crash. Closed head injury without skull fracture was reported in two cases; one other case had closed head injury with skull fracture. Single fractures to the left arm and left leg were reported and two cases had one or more ribs fractured that may be attributable to resuscitation attempts.

Eleven occupants died of bronchopneumonia with a similar number of male and female occupants reported (M:F, 5:6). Eight were drivers, two were front seat passengers and there was a single rear seat passenger. The average age was 61 years (range = 20 – 86) and the average BMI was 21.4 (median = 20.5, range = 28.7). It was unknown if a seatbelt was worn in the majority of cases (n=8), with one occupant not wearing a seat belt and two occupants being belted. There were six cases with closed head injury without skull fracture and one case with skull fracture. Three cases of cervical spinal fracture were recorded, with

three cases of left arm fracture, and one case each of right arm fracture and right leg fracture. A single case of pelvic fracture was reported, with two cases of lung lacerations and seven cases of one or more rib fractures.

Four occupants died from respiratory failure, with equal numbers of male and female occupants (M:F, 2:2). There were three drivers and a single front seat passenger. The average age was 50 years (range 33 – 82) and the average BMI was 38.1 (range = 21.7 – 62.4). One occupant was wearing a seat belt, one was not wearing a seat belt and belt use was unknown for the remaining two occupants. A single case of closed head injury without skull fracture was reported, with three cases involving cervical spinal fracture. There was one case of kidney injury, one case of pelvic fracture, and a single case of one or more fractured ribs, likely due to resuscitation attempts.

Five cases of multi-organ failure were reported. There were three drivers, one front seat passenger and one rear seat passenger. There were three males and two females. The average age was 73 years (range = 41 – 86) and the average BMI was 28.3 (range = 25.3 – 31.2). A single case of closed head injury without skull fracture was reported and there was a single case with skull fracture. There were single reports of thoracic spinal fracture, pelvic fracture and lung contusion. One or more rib fractures was reported in two cases, potentially a result of resuscitation attempts.

An 84 year old male front seat passenger died of congestive heart failure. He had a BMI of 37.9, with one or more rib fractures, likely due to resuscitation attempts.

A 94 year old female driver with a BMI of 23.4 died of pericarditis with pulmonary oedema. She had been wearing a seat belt and had a closed head injury without skull fracture, cervical spinal injury, bilateral lower leg fractures and a lung laceration.

Sepsis was reported at the cause of death in four cases. There was an equal number of male and female fatalities (M:F, 2:2), with three drivers and one front seat passenger. The

average age was 73 years (range = 18 – 73) and the average BMI was 29.1 (range = 23.2 – 33.7). Seat belt wearing was reported in three cases, while it was unknown if the remaining occupant was wearing a seat belt. Closed head injury without skull fracture was reported in one case, thoracic spinal fracture was also reported in one case, pelvic fracture was reported in two cases and one or more ribs were fractured in two cases.

Three occupants died from adult respiratory distress syndrome. Two were male and one was female. There was one driver and two front seat passengers. One had been wearing a seat belt, one was unbelted, and one had unknown seat belt wearing status. The occupants were aged 18, 19 and 73 with BMI of 30.7, 23.2 and 33.7, respectively. Reported fractures included one left arm, two left legs, two right legs and a single case of one or more fractured ribs.

6.5.5 Other causes of death

There are other causes of death which are distinctly different from causes of death relating to blunt force trauma. These other causes reported in the current study include crush injury, decapitation and drowning. As the mechanism of death in crush injury and drowning deaths are different, and decapitations are extremely rare in MVCs (0.2%), cases in which the cause of death was attributable to drowning, crush injury or decapitation will not be included in subsequent statistical modelling.

A 20-year-old male rear passenger with a BMI of 34.1 died of crush injuries. He had not been wearing a seat belt at time of the crash and had a positive blood alcohol concentration of 0.084g/100mL.

Six cases of decapitation were reported with more female than male occupants (M:F, 2:4). There were three drivers, one front seat passenger and two rear seat passengers, with an average age of 28 years (range = 19-40) and an average BMI of 26.4 (range = 23.3 – 34.5). Seat belt wearing was reported in three cases, while it was unknown if the remaining three

cases were wearing a seat belt at time of crash. There were significant injuries reported, notably three cases of lacerations to the heart and two cases of pelvic fracture. This is consistent with a high degree of crash impact severity to result in decapitation.

There were 11 cases of drowning reported, the majority being male (M:F, 8:3). There were seven drivers, two front seat passengers and two rear seat passengers. The average age was 43 (range = 20 -77) and the average BMI was 26.1 (range = 21.4 – 30.5). Seat belt wearing was reported in two cases and the remaining cases had unknown seat belt wearing status. There were minimal associated injures reported, with a single case of closed head injury without skull fracture and one case of thoracic spinal injury.

6.6 Correlation between occupant and crash characteristics

Several continuous occupant and crash characteristics were tested for correlation. A linear regression was performed to determine if there was any correlation between the factors of interest. All model outputs and goodness of fit tests are included in Appendix C.

To determine whether vehicle age changed across the study time frame, a linear regression was performed. Vehicle age was derived from the year of crash and year of vehicle manufacture. Figure 6.7 shows the vehicle age increased, on average, by 0.28 each year from 1981 to 2020. The overall regression was statistically significant ($R^2 = 0.039$, $F(1,2548) = 103.2$, $p < 0.001$) suggesting that more recent fatal crashes tend involve older cars more than earlier fatal crashes did. However, the low R^2 indicates that there is a considerable amount of unexplained variance within the data (i.e., year of crash only accounts for a small amount of the variation in vehicle age). This shows the increase in vehicle age is not large enough to be a leading contributing factor to fatal vehicle crashes. Therefore, the age of the vehicle will not be included in any subsequent analysis. For full model output, please see Appendix C.3.

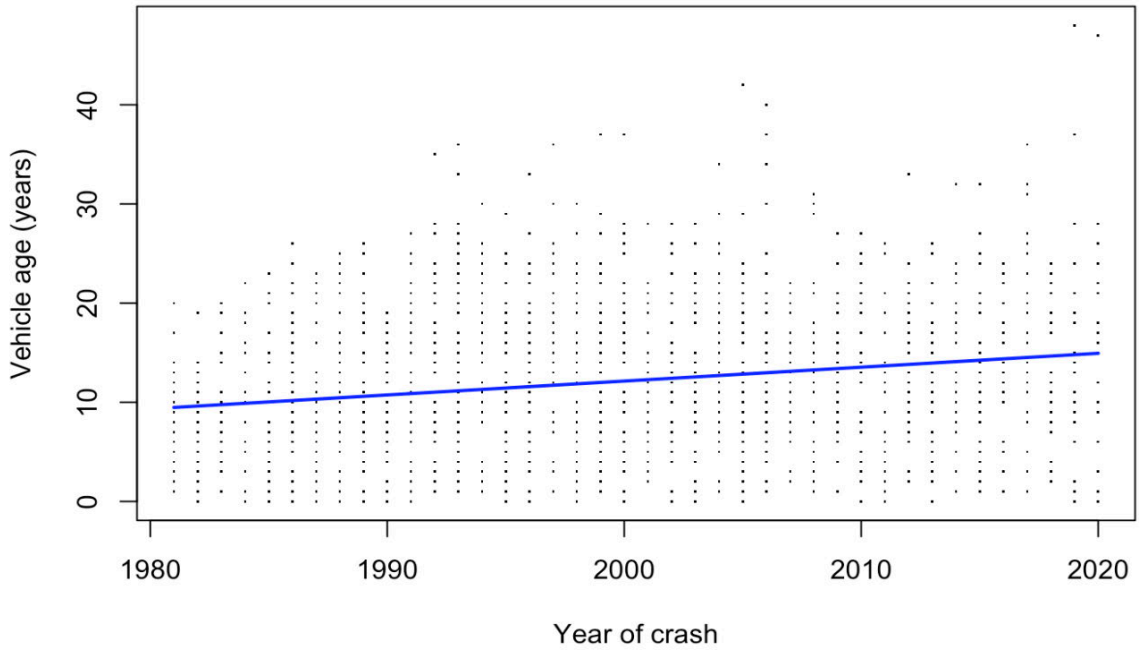

Figure 6.7 Fitted linear regression line (blue) for the relationship between vehicle age and year of crash

A linear regression was performed to assess if vehicle age changed with the age of the driver. This was performed on driver age and not occupant age, consistent with the approach of Keall & Newstead (2011) and Anderson & Doecke (2010). Analysis of residuals for the initial linear regression suggested that a polynomial regression was more appropriate to describe the relationship between driver age and vehicle age. The relationship between vehicle age and driver age in fatal crashes followed a U-shaped curve, suggesting younger and older drivers drove older vehicles, see Figure 6.8 (Adjusted $R^2 = 0.015$, $F(2,1780) = 14.45$, $p < 0.001$). As with the above analysis relating the age of the vehicle with the year of the crash, although the relationship between driver age and vehicle age was statistically significant, the low R^2 indicates that driver age only accounted for a small amount of the variation in vehicle age. This supports the previous finding that the age of the vehicle will not be necessary for subsequent analyses. For full model output, please see Appendix C.4.

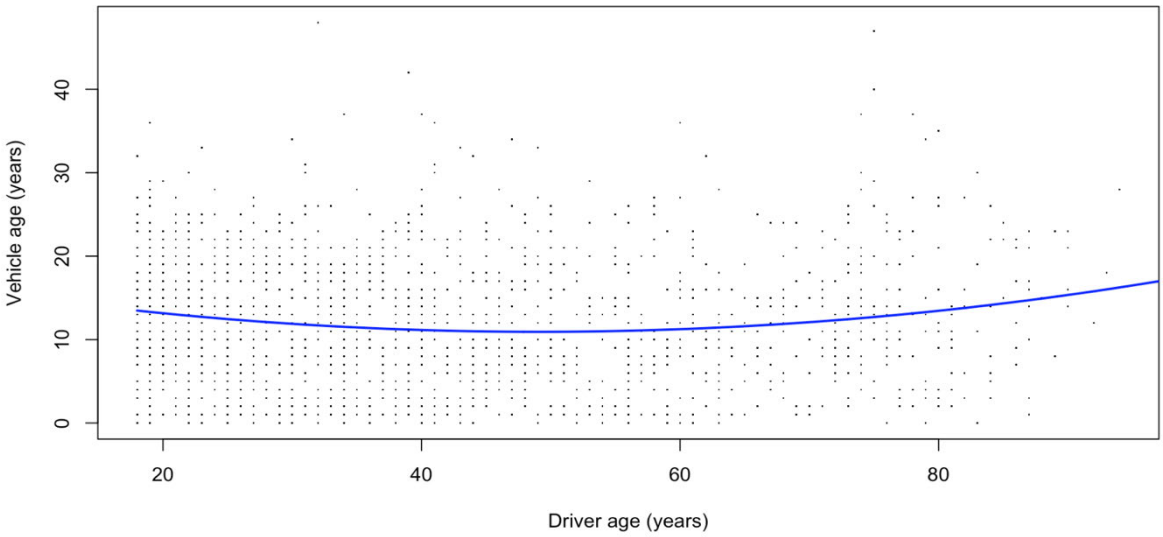

Figure 6.8. Fitted quadratic regression line (blue) for the relationship between vehicle age and driver age

As mentioned in Chapters Four and Five, both occupant age and BMI are known to interact to modulate injury severity. Furthermore, sex appears to interact with age, BMI and injury severity. To determine if a relationship exists between age and BMI among fatally injured vehicle occupants, a linear regression was performed. Sex was then also added to the model. The analysis of residuals suggested that a polynomial regression was more appropriate to describe the relationship between occupant age and BMI (Appendix C.5). The quadratic relationship between occupant age and BMI in fatal crashes, as shown in Figure 6.9, suggests that the average BMI in younger occupants gradually increased until a peak in late middle age before gradually decreasing in old age. The final model including sex showed that, on average, males had a BMI 0.5 lower than females, with both males and females following a quadratic relationship (adjusted $R^2 = 0.089$, $F(3,2200) = 73.2$, $p < 0.001$). There was a statistically significant relationship between age and BMI but the low R^2 indicates that occupant age only accounted for a small amount of the variation in BMI. Occupant age

cannot be reliably used as a predictor of BMI and therefore an interaction effect between the two variables will not be used in any subsequent analysis.

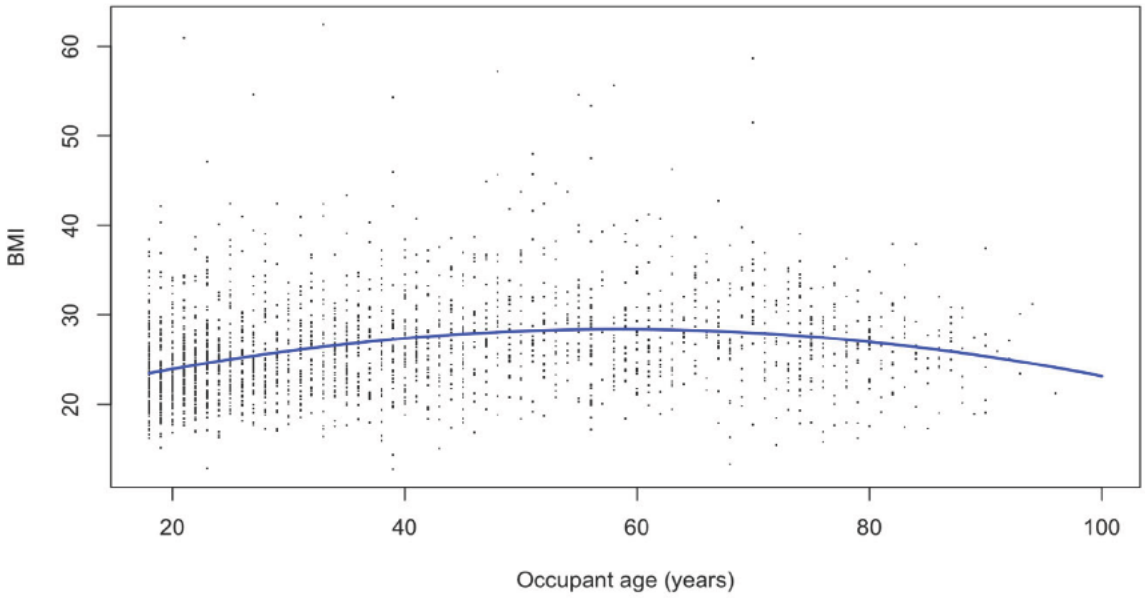

Figure 6.9 Fitted quadratic regression line (blue) for the relationship between BMI and occupant age

6.7 Location of death

The high energy impacts associated with fatal vehicle crashes often result in a rapid fatality at the scene of the crash. However, there is a subset of occupant fatalities who survive to hospital and a further subset of occupant fatalities who survive in hospital for a period of time. This points to the question of what factors determine location of death. Identification of death location was made through analysis of autopsy reports with clear indicators of the location of death (e.g., pathologist commenting on location of death, hospital equipment in situ etc). A number of cases (n=1704) for which location of death could not be determined or for which other case information was unavailable were therefore omitted from analysis of location of death. For this analysis, complications as a cause of death were included. To explore this question, a conditional inference decision tree was performed on a training dataset (a 60% randomised sample derived from the data pool) (Hothorn, Hornik & Zeileis 2006). The accuracy and validity of the model was then assessed using a testing dataset (the

remaining randomised 40% sample derived from the data pool). The final decision tree (Figure 6.10) contained two interior nodes that split the data into three categories. The first node splits the data by presence of an aortic injury (1) or no aortic injury (0). If an aortic injury ($p < 0.01$) is present, the majority of fatalities occurred at scene. If there was no aortic injury, a secondary node splits the data by age ($p < 0.01$). Occupants aged 73 years of age or younger were more likely to die at scene than on arrival to hospital or in hospital. Occupants aged over 73 years of age were slightly more likely to survive for a period of time in hospital than to die at scene.

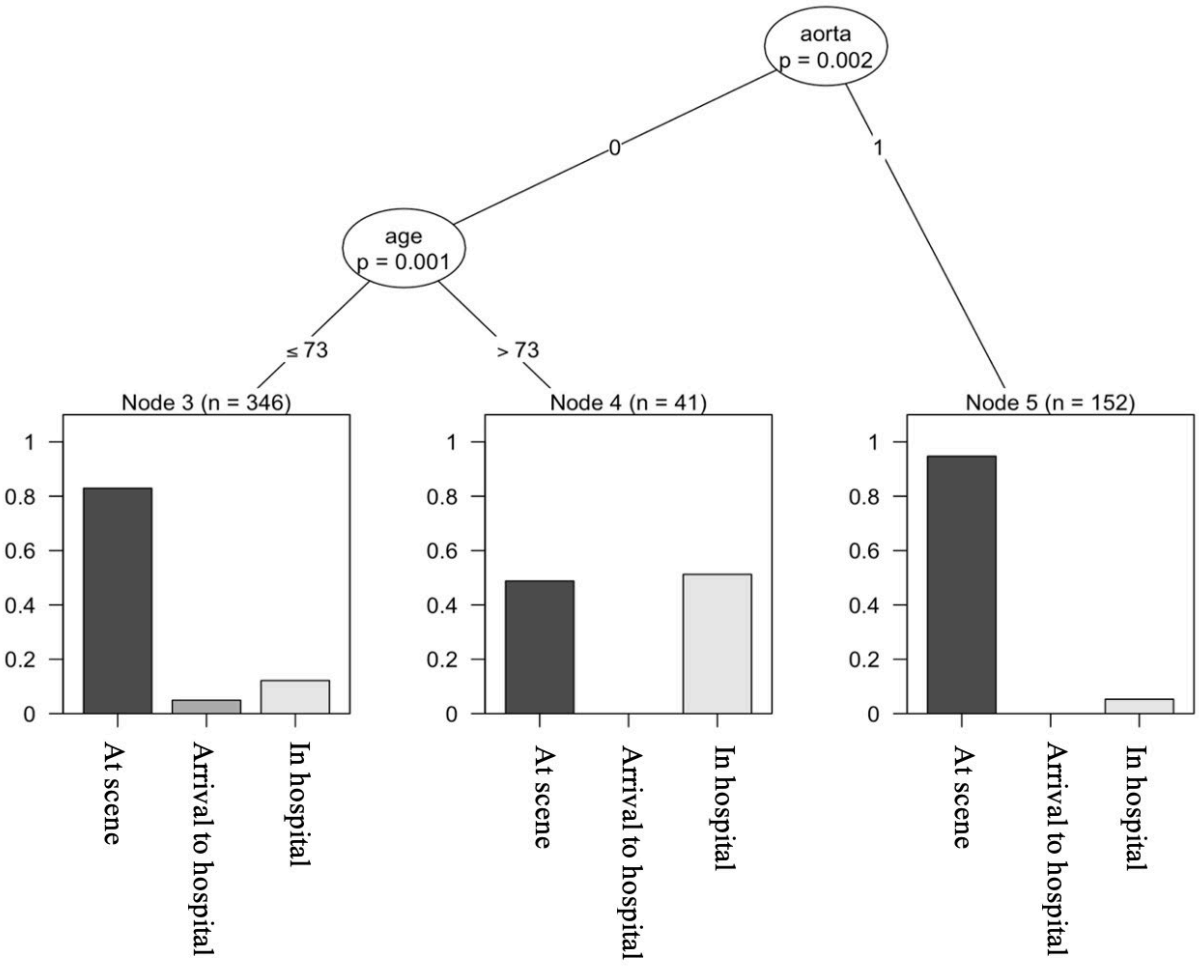

Figure 6.10 The results of a conditional inference tree predicting location of death trained on 60% training dataset

The results of a confusion matrix using the training dataset to test the fit of the decision tree found that the current model had a predictive accuracy level of 80.0% (95% CL 75.5-84.0) (Appendix C.6). The 20% of the cases that were incorrectly classified all involved prediction of death at the scene when they did in fact die enroute to hospital or in hospital. It is likely that the model accuracy is affected by the majority of the sample dying at scene. This may also explain why the confusion matrix did not predict any deaths on arrival to hospital as it was rarely observed in the dataset.

The initial decision tree was trained while including cases in which the occupants were unbelted and/or ejected from the vehicle, which is known to increase injury severity and therefore make death at scene more probable. A second decision tree was developed following the above protocol (with a training and testing dataset), but which only included restraint occupants who were not ejected. The final decision tree (Figure 6.11) contained a single interior node that split the data into two categories. The single node splits the data at the age of 73 ($p < 0.001$). Occupants 73 years of age and younger were more likely to die at scene than on arrival to hospital or in hospital. Occupants aged over 73 years of age were also more likely to die at scene but the proportion of occupants who survived for a period of time in hospital was higher than for occupants aged 73 years or younger. The results of a confusion matrix using the training dataset found that the current model had a predictive accuracy level of 82.0% (95% CL 76.5-87.0) (Appendix C.7). All cases successfully predicted by the confusion matrix were dead at scene, which may have been influenced by the high proportion of deaths at scene in the overall dataset. For full model and confusion matrix output, please see Appendix C.6 and C.7.

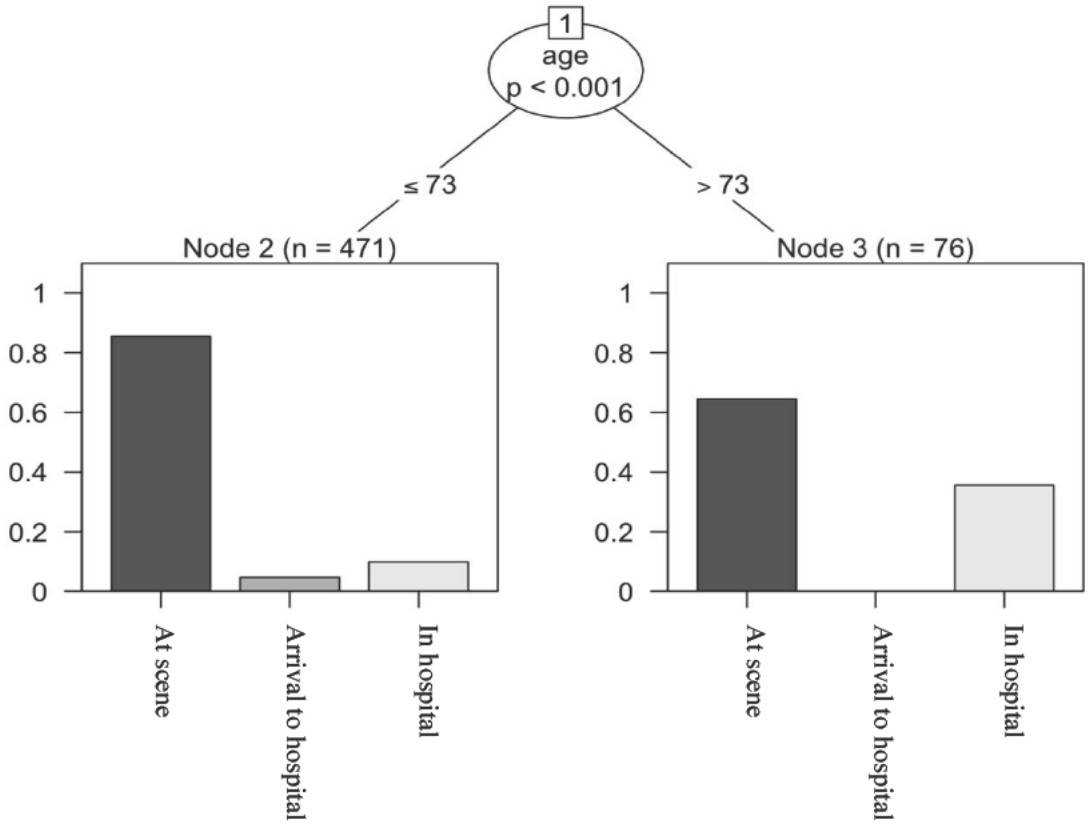

Figure 6.11 The results of a conditional inference tree predicting location of death in belted, non-ejected occupants, trained on 60% training dataset

6.8 Head injury

Head injury is one of the most common causes of death in MVCs, second only to multiple trauma, which usually includes head injury (approximately 73% of multiple trauma cases in the present dataset). There are several types of head injury that can be broadly categorised by the presence or absence of a skull fracture and whether the dura mater (the outmost layer of the meninges) has been breached (open head injury). Table 6.3 provides a summary of occupant and crash characteristics by type of head injury.

Table 6.3 Summary of occupant and crash characteristics by type of head injury				
	No head injury, n = 751	**Closed head injury without skull fracture, n = 545**	**Closed head injury with skull fracture, n = 1143**	**Open head injury, n = 129**
	mean, (range)	*mean, (range)*	*mean, (range)*	*mean, (range)*
Age	47 (18 – 100)	43 (18 – 96)	35 (18 – 90)	38 (18 – 81)
BMI	26.6 (12.7 - 57.2)	26.4 (14.4 – 58.7)	25.9 (16.2 – 60.9)	26.3 (17.9 – 48.0)
	n (%)	*n (%)*	*n (%)*	*n (%)*
Sex				
Male	483 (64)	344 (63)	827 (72)	96 (74)
Female	268 (36)	201 (37)	316 (28)	33 (26)
Seat belt wearing				
Worn	262 (35)	216 (40)	371 (33)	47 (36)
Not worn	121 (16)	90 (16)	195 (17)	19 (15)
Unknown	368 (49)	239 (44)	577 (50)	63 (49)
Seating Position				
Driver	490 (65)	378 (69)	825 (72.2)	94 (73)
Front seat passenger	201 (27)	130 (24)	243 (21.3)	27 (21)
Rear seat passenger	54 (7)	32 (6)	67 (5.8)	8 (6)
Unknown	6 (1)	5 (1)	8 (0.7)	0
Ejection				
Ejected	59 (8)	52 (10)	111 (10)	18 (14)
Not Ejected	692 (92)	493 (90)	1032 (90)	111 (86)

Similar patterns across the types of head injury were evident for BMI, sex, seat belt wearing and ejection.

6.8.1 Predicting head injury

An initial logistic regression including relevant occupant characteristics and crash factors was performed to determine which factors are most strongly associated with head injury. For this analysis, a binary response of no head injury or head injury (including closed and open head injury, with or without skull fracture) was used as the dependent variable. Model selection was determined using Bayesian Information Criterion (BIC) scoring. Injuries observed at autopsy were also included in the analysis and odds ratios (OR) and corresponding 95%

confidence intervals (95% CI) were calculated for each independent variable. The following variables were included in the model: Increasing age decreased the likelihood of head injury ($p < 0.001$, OR= 0.97, 95% CI = 0.96, 0.98), the presence of mandible fracture increased the likelihood of head injury ($p < 0.001$, OR = 7.59, 95% CI = 3.52 – 29.0), upper left limb fracture increased likelihood ($p < 0.001$, OR = 2.28, 95% CI = 1.56 – 7.52), lower left limb fracture decreased likelihood ($p < 0.01$, OR = 0.47, 95% CI = 0.28 – 0.77), pneumothorax decreased the likelihood ($p < 0.01$, OR = 0.36, 95% CI = 0.18 – 0.73), haemothorax also decreased the likelihood ($p < 0.001$, OR = 0.39, 95% CI = 0.25 – 0.60), and front seat passengers had a decreased likelihood of head injury relative to drivers ($p < 0.01$, OR = 0.46, 95% CI = 0.27 – 0.76), as did rear seat passengers ($p < 0.01$, OR = 0.26, 95% CI = 0.10 – 0.64).

The results of a confusion matrix using the training dataset to validate model accuracy and fit found the current model had predictive accuracy of 66.6% (95% CL 61.7-71.73). The model correctly identified head injury in 42.0% of cases and correctly identified no head injury in 83.6% of cases. For full model and confusion matrix output, please see Appendix C.8.

A secondary analysis using a conditional inference decision tree was performed to determine which factors predict head injury type stratified into the three categories as in Table 3. However, the model output was not consistently repeatable, indicating that there is volatility and variability within the data. In other words, the current factors included in the dataset cannot account for the data variability and so other external factors may be influencing the outcomes. One of possible reasons for the difficulty in predicting head injury outcome is the mechanism of head injury, whereby single impacts can cause a lethal head injury without additional injuries. It is possible that the factors which distinguish between types of head injury are factors that relate to crash impact patterns, such as intrusion into the

vehicle and point of impact, which were unfortunately not available to be included in this study. Therefore, the current study is limited in its ability to determine factors with distinguish between types of head injury. This does, however, create an avenue for future research. For full model and confusion matrix output, please see Appendix C.9.

6.8.2 Ring and hinge fracture

Closed head injury with skull fracture is the most common form of head injury. Skull fractures may involve the cranial vault, fronto-basilar bones and the base of the skull. Basilar skull fractures are common in vehicle crashes but there are two specific types of basilar skull fracture, the hinge and ring fracture, which commonly occur in high impact crashes. Transverse hinge fractures are a type of crush fracture, occurring in the middle of the skull base with the fracture lines passing through the left and right middle anterior fossa (Zhang et al. 2022). Hinge fractures may be the result of crushing of the left and right temporal regions or unilateral blunt force to the temporal regions (Zhang et al. 2022). Ring fractures occur through the floor of the middle fossa of the base of the skull and the squamous region of the temporal and occipital bones. These may result in an incomplete or complete separation of the rim of the foramen magnum from the base of the skull (Jang & Kim 2014; Zhang et al. 2022,) One possible mechanism of ring fractures is the upward force of the spine against the skull base (Jang & Kim 2014).

While ring and hinge fractures are relatively infrequent compared to other skull fracture types, the mechanisms of injury are specific and therefore the following logistic regression was performed to determine which factors predict hinge fractures and ring fractures, respectively. Overall, there was a lower proportion of hinge fractures (n=155) in those with skull fractures than those without hinge fractures (n=319). The probability of hinge fracture further decreased with increasing age ($p < 0.05$, OR = 0.98, 95% CI = 0.97 – 1.0) and decreased with the presence of a nasal/zygoma fracture ($p < 0.01$, OR = 0.41, 95%

CI = 0.23 – 0.70) (Appendix C.10). The decreased probability of hinge fracture in the presence of a nasal/zygoma fracture could suggest that, in cases in which the head impact is to the middle face region, there is a reduction in the dispersion of force through the temporal bones. Ring fractures were present in a significantly lower proportion (n=72, p<0.001) of those with skull fractures (n=400). The probability of ring fracture increased with brainstem injury (p < 0.001, OR = 3.34, 95% CI = 1.84 – 5.98), the presence of a left lower limb fracture (p < 0.01, OR = 2.12, 95% CI = 1.20 – 3.69) and the presence of pelvic fracture (p < 0.01, OR = 2.22, 95% CI = 1.29 – 3.82) (Appendix C.11). That fractures to the left lower limb and pelvis increased the probability of ring fracture is likely to be indicative of higher crash severity rather than representing factors involved specifically in the causation of ring fracture.

6.8.3 Brainstem injury

Brainstem injury is known to be associated with ring fractures, as predicted by the current model (see the previous section), and hinge fractures (Zivkovic et al. 2010; Živković et al. 2012). Brainstem injuries can range from haemorrhage within the brainstem through to lacerations and complete transection, mainly at the pontomedullary junction, which are instantaneously fatal.

A stepwise logistic regression was used to determine if injuries suggested by previously literature predicted brainstem injury in the current study. Brainstem injury was present in a significantly lower proportion (n=121, p<0.001) of those in the sample population (n=1000), with the likelihood of brainstem injury increasing with the presence of atlanto-occipital injury (p < 0.001 , OR = 3.10, 95% CI = 1.98 – 4.81), hinge fracture (p < 0.001, OR = 2.94, 95% CI = 1.80 – 4.73), ring fracture (p < 0.001, OR = 5.71, 95% CI = 3.22 – 9.96) and transection of the spinal cord (p < 0.001, OR = 2.78, 95% CI = 1.67 – 4.53). For the full model, please see Appendix C.12.

6.9 Atlanto-occipital fracture

While atlanto occipital injury strongly predicts brainstem injury (see the previous section), atlanto-occipital injury can occur without injury to the brainstem. A stepwise logistic regression was used to determine if age was a predictor of atlanto-occipital fracture and whether other injuries, such as injury to the other levels of the spinal column, predict atlanto-occipital fracture. Atlanto-occipital injury was present in a significantly lower proportion ($n=200$, $p<0.001$) of those in the sample population ($n=929$), with the probability increasing with the presence of closed head injury without skull fracture ($p < 0.001$, OR = 2.98, 95% CI = 2.10 – 4.21), brainstem injury ($p < 0.001$, OR = 3.64, 95% CI = 2.33 – 5.64) and transection of the spinal cord ($p < 0.001$, OR = 2.50, 95% CI = 1.64 – 3.78). Unexpectedly, closed head injury without skull fracture was included in the final model. This may be related to the consequences of atlanto-occipital injury, such as subarachnoid haemorrhage, that may travel upwards into the skull. It may also be possible that, in crashes involving a head impact, the impact force is transferred through the skull, whilst in crashes without a head impact, the rotational forces act on the brain parenchyma as well as concentrating at the atlanto-occipital joint, resulting in closed head injury without skull fracture and atlanto-occipital joint injury. For the full model output, please see Appendix C.13.

6.10 Factors predicting the level of spinal injury

Spinal injuries occurred in just over a quarter of occupants (25.97%), with 9.6% cervical, 15.49% thoracic and 2.31% lumbar spinal injuries. These injuries included fractures of the vertebral body, wedge and burst fractures, as well as fracture dislocations. Table 6.4 shows a summary of the occupant and crash characteristics at the various levels of spinal injury.

Table 6.4 Occupant and crash characteristics at the various levels of spinal injury

	Cervical spine, n = 342	Thoracic spine, n = 396	Lumbar spine, n = 59
	mean, (range)	*mean, (range)*	*mean, (range)*
Age	46 (18 – 93)	50 (18 – 94)	43 (18 – 88)
BMI	26.8 (16.7 -54.6)	28.4 (17.0 – 55.6)	27.5 (17.6 – 44.9)
	n (%)	*n (%)*	*n (%)*
Sex			
Male	209 (61)	269 (68)	32 (54)
Female	133 (39)	127 (32)	27 (46)
Seat belt wearing			
Worn	139 (41)	188 (47.5)	22 (37)
Not worn	56 (16)	80 (20.2)	17 (29)
Unknown	147 (43)	128 (32.3)	20 (34)
Seating Position			
Driver	229 (67)	294 (74.2)	41 (69.5)
Front seat passenger	88 (25.7)	82 (20.7)	15 (25.4)
Rear seat passenger	22 (6.4)	17 (4.3)	2 (3.4)
Unknown	3 (0.9)	3 (0.8)	1 (1.7)
Ejection			
Ejected	28 (8)	34 (9)	15 (25)
Not Ejected	314 (92)	58 (91)	44 (75)

To explore the relationship between spinal fractures and specific occupant factors and crash factors, stepwise logistic regressions were used to determine which factors predicted cervical spinal injury, thoracic spinal injury, and lumbar spinal injury, respectively. BMI was separated into height and weight for the following analysis of spinal injuries and crash type was also included a possible relationship between height and roll overs was investigated. As expected, increasing age was the most significant predictor of cervical spinal fracture ($p < 0.001$, OR= 1.02, 95% CI = 1.01, 1.03). Increasing age was also a predictor of thoracic spinal injury ($p < 0.001$, OR= 1.03, 95% CI = 1.02, 1.03) as was greater weight ($p < 0.001$, OR= 1.01, 95% CI = 1.01, 1.02). Not being ejected from the vehicle decreased the risk of lumbar spinal injury ($p < 0.01$, OR= 0.32, 95% CI = 0.16, 0.69), while newer vehicles were associated with an increased likelihood of lumbar injury ($p < 0.01$, OR= 1.06, 95% CI = 1.02,

1.10). Although these factors are statistically significant, the chance of having a lumbar spine was extremely low and injury of the lumbar spine was unlikely even when the factors above were present. For the full model output, please see Appendix C.14, C.15 and C.16.

6.11 Blunt chest injury

Blunt chest injury was the third leading cause of death, and some level of chest injury is common in MVCs. Drivers in particular are vulnerable to contact with the steering wheel during crashes increasing the likelihood of chest injury. It is likely that the lethality associated with blunt chest injuries is the rapid blood loss that occurs with injury to the major organs and vasculature in the chest cavity. Injuries to the heart, aorta, subsidiary cardiac vessels and lung avulsion are highly lethal or indicative of high energy impacts. These have been summarised in Table 6.5.

Table 6.5 Occupant and crash characteristics related to blunt chest injuries				
	Heart, n = 500	**Cardiovascular, n = 70**	**Aorta, n = 725**	**Lung avulsion, n = 31**
	mean, (range)	*mean, (range)*	*mean, (range)*	*mean, (range)*
Age	43 (18 – 93)	46 (18 – 87)	44 (18 – 100)	41 (18 – 80)
BMI	26.3 (13.3 - 57.2)	26.8 (13.3 – 54.6)	26.9 (14.4 – 55.6)	27.1 (18.1 – 47.1)
	n (%)	*n (%)*	*n (%)*	*n (%)*
Sex				
Male	349 (70)	44 (63)	499 (69)	21 (68)
Female	151 (30)	26 (37)	226 (31)	10 (32)
Seat belt wearing				
Worn	191 (38)	21 (30)	256 (35)	6 (19)
Not worn	99 (20)	23 (33)	113 (16)	5 (16)
Unknown	210 (42)	26 (37)	356 (49)	20 (65)
Seating Position				
Driver	342 (68.5)	47 (67)	518 (71.5)	24 (77)
Front seat passenger	122 (24.5)	18 (25.7)	159 (21.9)	4 (13)
Rear seat passenger	31 (6)	4 (5.7)	43 (5.9)	3(10)
Unknown	5 (1)	1 (1.4)	5 (0.7)	0
Ejection				
Ejected	46 (9)	12 (17)	47 (6.5)	4 (13)
Not Ejected	454 (91)	58 (83)	678 (94)	27 (87)

Injuries to the lungs are also common, with contusion and lacerations often associated with rib fractures. Rib fractures are more common than not within the current dataset, with 72.2% of cases having one or more rib fractures. Although cases have been removed in which the rib fractures were attributed to resuscitation attempts, there are still potentially some cases in the sample in which ribs were fractured or fractures were exacerbated by resuscitation attempts. As a single rib fracture is not considered a severe injury, a binary category determining whether one or more rib fractures were present is unsuitable to fully investigate rib fractures and the injuries associated with them.

6.11.1 Publication: Fatal blunt chest trauma: an evaluation of rib fracture patterns and age

S. O'Donovan, C. Van Den Heuvel, M.R.J. Baldock, R.W. Byard

To investigate the relationship between the number and pattern of rib fractures to occupant characteristics, crash factors and other injuries, a study was undertaken using a subset of the current sample to explore the patterns of rib fractures in MVCs.

Statement of Authorship

Title of Paper	Fatal blunt chest trauma: An evaluation of rib fracture patterns and age
Publication Status	☒ Published ☐ Accepted for Publication ☐ Unpublished and Unsubmitted work written in manuscript style ☐ Submitted for Publication
Publication Details	O'Donovan, S, Van Den Heuvel, C, Baldock, MRJ, Humphries, MA & Byard, RW 2022, 'Fatal blunt chest trauma: an evaluation of rib fracture patterns and age', *International Journal of Legal Medicine*, advance online publication, DOI: 10.1007/s00414-022-02866-2.

Principal Author

Name of Principal Author (Candidate)	Siobhan O'Donovan		
Contribution to the Paper	Collected autopsy data, performed statistical analysis, wrote manuscript, acted as corresponding author		
Overall percentage (%)	85%		
Certification:	This paper reports on original research I conducted during the period of my Higher Degree by Research candidature and is not subject to any obligations or contractual agreements with a third party that would constrain its inclusion in this thesis. I am the primary author of this paper.		
Signature		Date	27/05/2022

Co-Author Contributions

By signing the Statement of Authorship, each author certifies that:

- the candidate's stated contribution to the publication is accurate (as detailed above);
- permission is granted for the candidate in include the publication in the thesis; and
- the sum of all co-author contributions is equal to 100% less the candidate's stated contribution.

Name of Co-Author	Corinna Van Den Heuvel		
Contribution to the Paper	Primary supervisor of Siobhan, helped to evaluate and edit the manuscript		
Signature		Date	04/07/2022

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Contribution to the Paper	Co-supervisor of Siobhan, provide access to Traffic Accident Reporting System through Centre for Automotive Safety Research, helped to evaluate and edit the manuscript		
Signature		Date	27/5/2022

Please cut and paste additional co-author panels here as required.

Name of Co-Author	Melissa Humphries		
Contribution to the Paper	Performed statistical analysis, assisted in data interpretation and visualisation, evaluation of manuscript		
Signature		Date	20/06/2022

Name of Co-Author	Roger Byard		
Contribution to the Paper	Co-supervisor of Siobhan, supervised collection of autopsy data, helped to evaluate and edit the manuscript		
Signature		Date	08/07/2022

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ORIGINAL ARTICLE

Fatal blunt chest trauma: an evaluation of rib fracture patterns and age

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Abstract
The following study was undertaken to determine if any specific occupant characteristics, crash factors, or associated injuries identified at autopsy could predict the occurrence or number of fractured ribs in adults. Data were accrued from the Traffic Accident Reporting System (TARS) and coronial autopsy reports from Forensic Science SA, Adelaide, South Australia, from January 2000 to December 2020. A total of 1475 motor vehicle fatalities were recorded in TARS between January 2000 and December 2020, and 1082 coronial autopsy reports were identified that corresponded to TARS fatal crash data. After applying exclusion criteria involving missing data, 874 cases were included in the analysis. Of the 874 cases, 685 cases had one or more rib fractures. The leading cause of death for those with rib fractures was multiple trauma (54%), followed by head injury (17%) and chest injuries (10%). The strongest predictor of one or more rib fractures was increasing age ($p < 0.001$). Other factors found in the regression to be predictive of the number of rib fractures were the presence of a variety of other injuries including thoracic spinal fracture, lower right extremity fracture, splenic injury, liver injury, pelvic fracture, aortic injury, lung laceration, and hemothorax. Age is most likely associated with increasing rib fractures due to reduced tolerance to chest deflection with greater injuries occurring at lower magnitudes of impact. The association of other injuries with rib fractures may be a marker of higher impact severity crashes.

Keywords Rib fractures · Fatal vehicle crashes · Age · Injury · Forensic

Introduction

Blunt force chest injuries with associated rib fractures are frequent in motor vehicle crashes. Injuries to the thorax can occur from impacts with the steering wheel or instrument panel in frontal crashes, from side doors in side impact crashes, or from a combination of these. Fractured ribs may lead to injury of thoracic organs, particularly with the sharp ends of the ribs having the potential to tear/puncture vessels and the lungs, or the pleura leading to pneumothorax or hemothorax [1, 2].

Cadaveric studies and finite element models are often used to simulate and predict rib fracture patterns in injury research, and studies have used clinical data to identify patterns of rib fracture occurring with multiple causes of trauma including motor vehicle crashes [3–8].

The following study evaluates the patterns of rib fracture in adult occupants involved in fatal motor vehicle crashes and combines coronial autopsy report injury descriptions with crash data information. Seat belt wearing is mandatory for all motor vehicle occupants in Australia. The aims of this study are to determine if any specific occupant characteristics, crash factors, or associated injuries identified at autopsy can predict the occurrence or number of ribs fractured.

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Materials and methods

Crash data—Traffic Accident Reporting System

The crash data used in the present study were extracted from the Traffic Accident Reporting System (TARS). TARS is comprised of all crashes reported to the South Australia Police and is maintained by the Department of Transport and Infrastructure. The study time period was between January 2000 and December 2020. Data from TARS were made available through the Centre for Automotive Safety Research at the University of Adelaide. All cases were motor vehicle occupant fatalities ≥ 18 years of age reported in TARS. Pediatric motor vehicle occupants were excluded as the relative size difference between children and adults can alter the performance and function of seat belts and change the pattern of injury [9, 10]. Furthermore, infants and young children may have a higher injury tolerance to chest impact due to the elasticity of the pediatric thorax [11]. Due to the difference in crash circumstances and therefore patterns of injury, heavy vehicles (e.g., trucks, buses) were excluded from the study. Crash factors selected from TARS for analysis were position in vehicle, restraint status, ejection, crash type, vehicle type, and year of vehicle manufacture at the time of the crash.

Fatal injury data—coronial autopsy reports

Cases identified in TARS were then cross-matched with corresponding coronial autopsy reports from Forensic Science SA. Occupant characteristics such as body mass index (BMI), cause of death, and the presence of other injuries were extracted from the autopsy reports. Rib fracture information was also extracted from autopsy reports identified through gross examination by the examining pathologist. Concomitant injuries were recorded if the injury description in the autopsy report satisfied the requirements for an Abbreviated Injury Severity score of 3 or above [12]. Individual cases were excluded from the dataset if the pattern of rib fractures were attributed by the examining pathologist to resuscitation attempts or the body had been incinerated.

As the coronial autopsy files contained varying levels of detail in regard to rib fracture descriptions, the sample sizes for each level of analysis varied depending on the amount of detail on rib injuries that was available (Fig. 1). Cases with missing data for belt status, seating position, and year of vehicle manufacture were excluded from the logistic regression modelling. However, cases with missing data were still included in the dataset used for heat mapping if data relating to rib number, side, and anatomical location were available (Fig. 1).

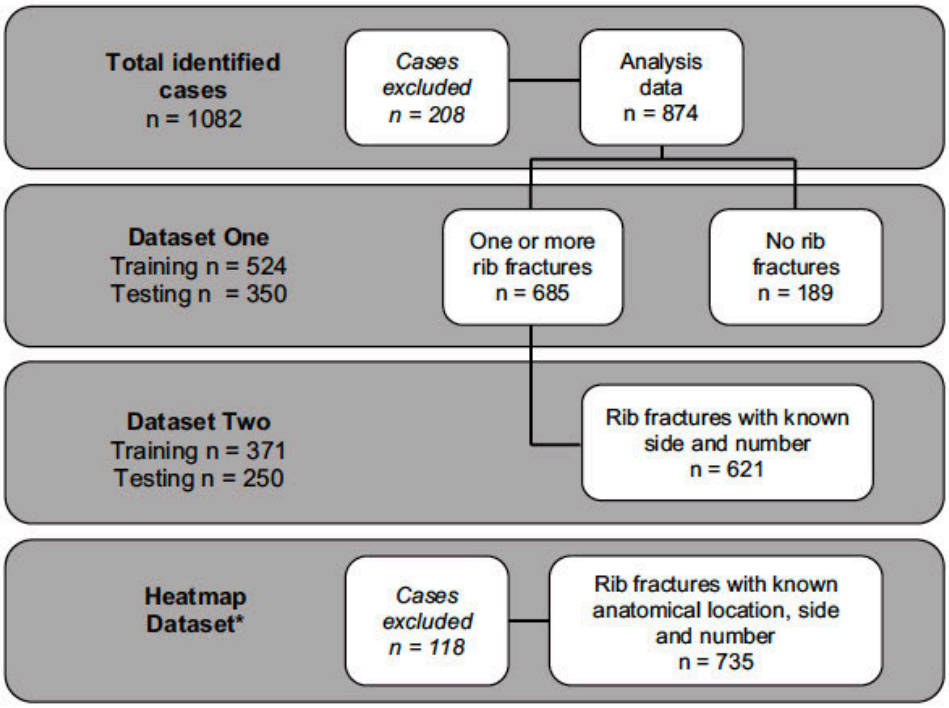


Fig. 1 Summary of dataset inclusion and exclusion. All cases were derived from the initial pool of coronial autopsy reports identified with corresponding TARS crash data. Of the 1082 identified cases, 853 cases had rib fractures, 229 cases had no rib fractures. As outlined in the data analysis, cases with missing data are required to be excluded for model development ($n = 208$) however, the development of heat maps permits cases to have missing data. The sample of 735 cases for the heat map dataset was derived from the 853 cases with rib fractures (asterisk symbol). The remaining 118 cases with rib fractures identified in the initial dataset ($n = 853$) were excluded in the final heat map dataset due to missing anatomical, side, or number of ribs fractured

Statistical methods

All analyses were conducted using R studio version 1.4 [13]. Two separate datasets were created for the analyses (Fig. 1). Training (60%) and test (40%) subsets were extracted from the data for individual model development and validation.

Logistic regression modelling was performed ($\alpha = 0.05$) on the training data to predict whether or not a vehicle occupant had sustained at least one rib fracture, including injury type, occupant, and crash characteristics as independent variables. Increasing age is recognised as the strongest predictor for rib fractures and was therefore included in all models as a covariate [6, 14, 15].

The results of the logistic regression are presented as odds ratios (OR) with 95% confidence intervals (CI). Model development was performed using a stepwise method with selection determined using Bayesian information criterion (BIC) scoring. Sternal injury was excluded from the model as sternal fractures rarely occur without rib fractures and therefore behave as a proxy for rib fractures. The testing dataset was then used to assess the fit of the model with performance measured using a confusion matrix.

A linear regression (with $\alpha = 0.05$) was performed to determine if any of the occupant characteristics, crash characteristics, or associated injuries could significantly predict the number of rib fractures (among those who had sustained at least one fractured rib). This linear regression was performed using the training data of dataset two. Results of the linear regression are reported using adjusted R squared and F statistics with degrees of freedom. All model outputs and goodness of fit tests are included in the Appendix.

Heat maps were generated for selected occupant characteristics, crash factor, and injuries to visualise the anatomical pattern of rib fractures. The proportion of total ribs fractured for the condition of interest was calculated to standardise the appearance of the heat maps across varying sample sizes for each condition.

Results

A total of 1475 motor vehicle fatalities were recorded in TARS between January 2000 and December 2020, and 1082 coronial autopsy reports were identified that corresponded to TARS fatal crash data. After applying the exclusion criteria involving missing data, 874 cases were included in the analysis (Fig. 1). To summarize the excluded occupant cases, 153 cases had unknown belt status, 12 cases had unknown seating position, 14 cases had indeterminable BMI (due to amputations etc.), and there were 29 cases for which the year of vehicle manufacture was unknown.

Cause of death

Of the 874 cases, 685 cases had one or more rib fractures. The leading cause of death for those with rib fractures was multiple trauma (54%), followed by head injury (17%) and chest injuries (10%). The remaining 189 cases did not have any rib fractures at autopsy. The main cause of death for occupants without rib fractures was head injury (52%), followed by multiple trauma (20%) and spinal injuries (6.9%). It should be noted that the cause of death was taken as the main injury that resulted in death. Table 1 provides a descriptive summary of the occupant and crash characteristic in fatal cases with and without rib fractures.

Predicting rib fracture patterns

The strongest predictor of one or more rib fractures was increasing age ($p < 0.001$, OR = 1.04, 95% CI = 1.03–1.06). Liver, lung contusion, and hemothorax were also significant factors in predicting one or more rib fractures. Please see appendix for full model output. The accuracy of the model as determined by a confusion matrix was 83% (accuracy = 0.831, 95% CI = 0.788–0.869). The model had sensitivity of 47% (0.474) and specificity of 93% (0.930) when predicting whether an occupant sustained one or more rib fractures.

Predicting the number of ribs fractured

Multiple linear regression was used to test if any of the occupant characteristics, crash characteristics, or associated injuries significantly predicted the number of ribs fractured. The overall regression was statistically significant (adjusted $R^2 = 0.399$, F (8,362) = 31.72, $p < 0.001$). The factors found

Table 1 Demographic characteristics of 874 occupant fatalities with and without rib fracture in South Australia (2000–2020)

Characteristic	No rib fracture, $N = 189$	Rib fracture, $N = 685$
Age	32 (18–87)	46 (18–96)
BMI	26 (16–61)	28 (16–57)
Year of vehicle manufacture	1995 (1970–2018)	1996 (1942–2020)
Sex		
Male	147 (78%)	468 (68%)
Female	42 (22%)	217 (32%)
Seat belt		
Not wearing	59 (31%)	201 (29%)
Wearing	130 (69%)	484 (71%)
Ejected from vehicle		
No	159 (84%)	599 (87%)
Yes	30 (16%)	86 (13%)

in the regression to be predictive of the number of rib fractures were increasing age (Fig. 2) and the presence of a variety of other injuries including thoracic spinal fracture, lower right extremity fracture, splenic injury, pelvic fracture, aortic injury, lung laceration, and hemothorax. Please see appendix for full model output.

The following heat maps show the proportion of rib fractures at various anatomical locations on the rib cage for different variable conditions (Figs. 3, 4, 5, and 6).

Discussion

Increasing age was the strongest predictor of rib fractures as has been noted in previous research [6, 14, 15]. However, the current study demonstrates that the effect of age is stronger than any other occupant or crash characteristic. This effect is clearly shown in Fig. 3 where the proportion of ribs fractured for occupants 65 years and older is far greater than for younger vehicle occupants.

Impact to the thorax results in "posterior displacement of the sternum relative to the spine", referred to as chest deflection [16]. The number of rib fractures is associated with the magnitude of chest deflection rather than the impact force, and the threshold of injury during chest deflection is dependent on age [16, 17]. Increasing age results in reduced chest deflection tolerance and therefore greater injury at lower magnitudes of deflection [16]. Age-related demineralisation and degradation of cortical bone result in bone remodelling, which causes rib deterioration, while cartilaginous sections of rib undergo calcification and hardening, which reduces rib flexibility. Thus, the ability of the aging ribcage to deflect thoracic impact is reduced and this explains why aged ribs fracture more readily [18].

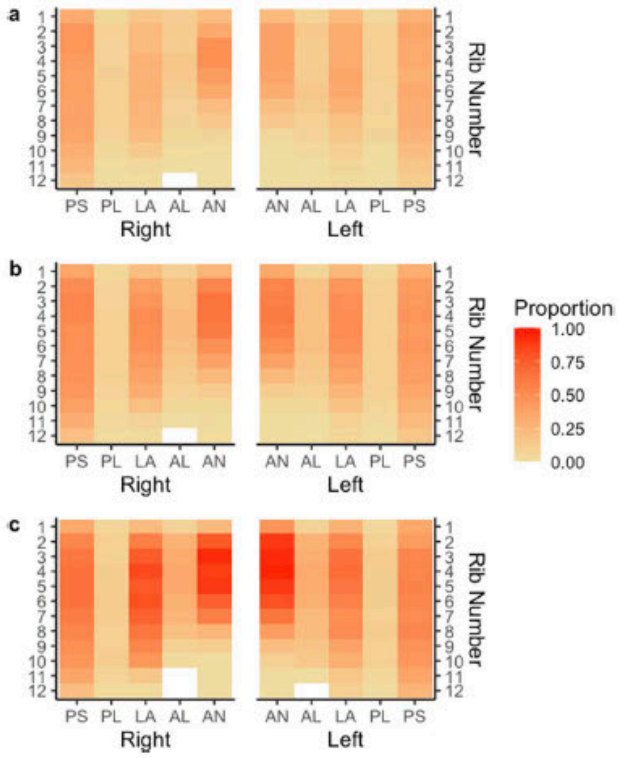
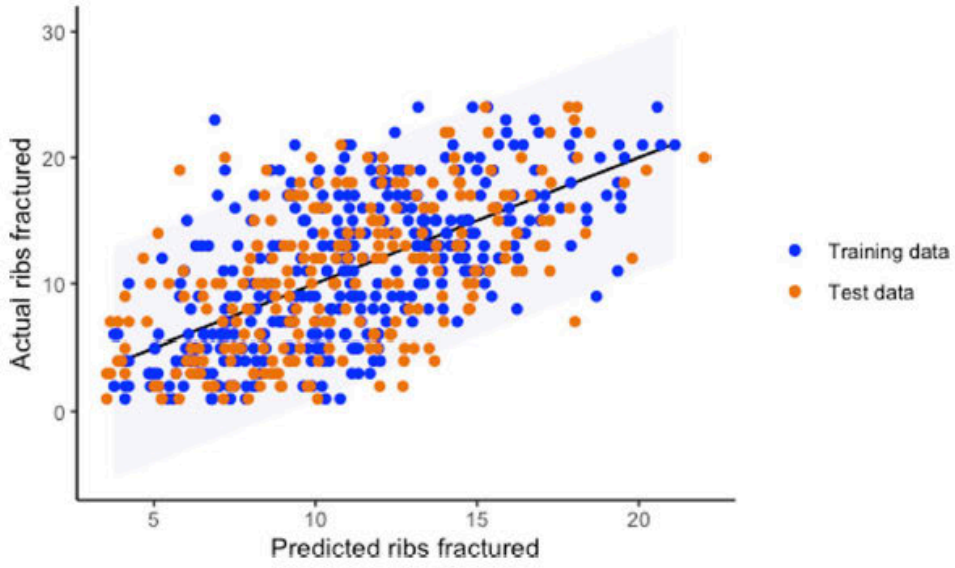


Fig. 3 Proportion of rib fractures by rib number, rib cage side, and anatomical location by age. Panel **a** describes the proportion of rib fractures for ages 18–39. Panel **b** describes the proportion of rib fractures for age 40–64. Panel **c** describes the proportion of rib fractures for age > 65 years of age. The abbreviations of anatomical locations are as follows: AN, anterior; AL, anterolateral; L, lateral; PL, posterolateral; PS, posterior

Fig. 2 Linear regression of training dataset predicting number of ribs fractured plotted against test dataset. Along the *x*-axis are the values predicted by the linear regression using the training data. The *y*-axis shows the actual values within the test dataset. 95% prediction intervals for number of ribs fractured



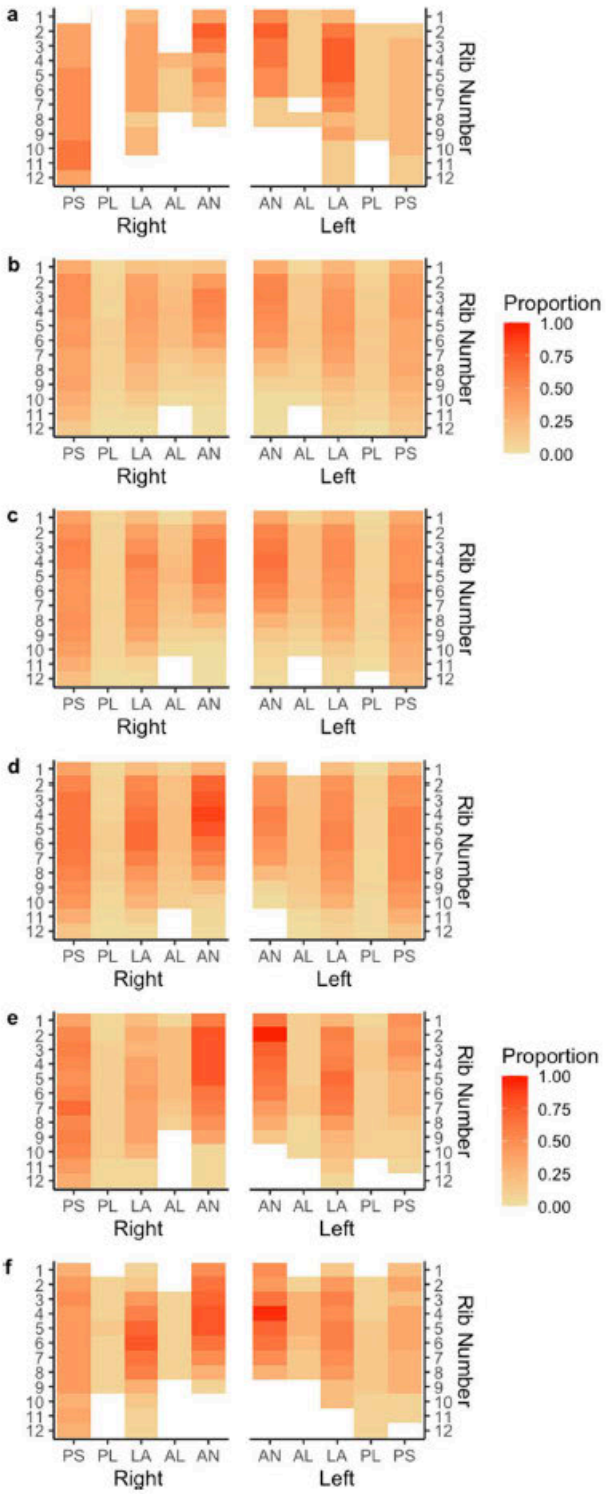


Fig. 4 Proportion of rib fractures by rib number, rib cage side, and anatomical location by BMI. Panel **a** describes the proportion of rib fractures for BMI < 17.99. Panel **b** describes the proportion of rib fractures for BMI 18–24.99. Panel **c** describes the proportion of rib fractures for BMI 25–29.99. Panel **d** describes the proportion of rib fractures for BMI 30–34.99. Panel **e** describes the proportion of rib fractures for BMI 35–39.99. Panel **f** describes the proportion of rib fractures for BMI > 40. The abbreviations of anatomical locations are as follows: AN, anterior; AL, anterolateral; L, lateral; PL, posterolateral; PS, posterior

Increased risk of fatality is associated with increased number of ribs fractured for older occupants. In a study of hospitalised trauma patients, a majority of whom were motor vehicle occupants, Bulger et al. found that, for every additional rib fracture in elderly patients (> 65 years of age), the risk of pneumonia increased by 29% and mortality by 19% [19]. Additionally, rib fractures are frequently associated with other thoracic injuries such as aortic damage and pulmonary contusions. The combined effects of rib fractures and underlying injuries are particularly detrimental to the already reduced physiological reserve and decreased ventilatory capacity of older people. Stiffening of the chest wall, reduced cardiac output, reduced lung capacity, poor blood oxygen exchange, and pain-related limitation of mechanical ventilation in older persons with rib fractures also increase associated mortality [14].

Importantly, restraint use was not predictive of rib fracture or of a greater number of ribs fractured in either model. While seat belts load the thorax during crash impacts, the likelihood of rib fracture is not greater with restraint use compared to no restraint use. Fatalities of seat belt wearers, however, often involve significant impacts that would likely be fatal irrespective of belt wearing [20, 21].

Liver injury was the second strongest predictor of one or more rib fractures. Lung contusion and hemothorax also appeared as significant factors in the model. Similarly, when predicting the number of ribs fractured, fractures of the right leg, splenic injury, and pelvic fracture all appeared more significant than other typical blunt injuries (e.g., aortic injury). The association with non-chest injuries most likely suggests an overall higher level of injury severity. Thoracic spinal fracture is an expected predictor of the number of ribs fractured as the thoracic spine connects to all ribs and, consequently, fracture of one of thoracic vertebrae may produce one or more rib fractures. Figure 6 demonstrates that the posterior anatomical location has a higher proportion of rib fractures for those with a thoracic spine fracture compared to no thoracic spine fracture.

Although linear regression was able to predict the number of ribs fractured, the model had a low predictive power suggesting that there are other factors affecting the number of rib fractures that are not included in this dataset such as speed of impact, vehicle intrusion and point of impact. It may also be the case that the relationship between ribs fractured and injury severity is cumulative whereby the aggregated number of ribs fractured creates distinct levels of injury severity as opposed to the incremental increase in injury severity for every additional rib fractured [22].

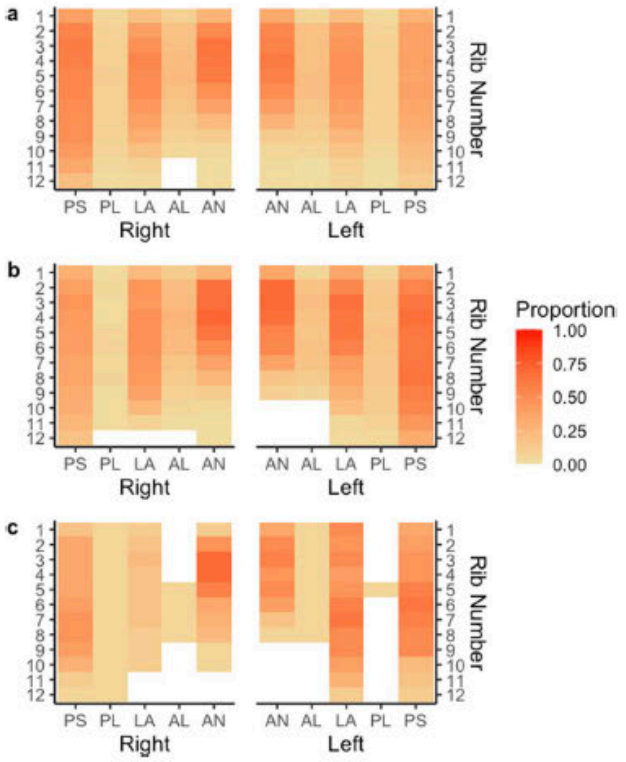


Fig. 5 Proportion of rib fractures by rib number, rib cage side, and anatomical location by position in vehicle. Panel **a** describes the proportion of rib fractures for drivers. Panel **b** describes the proportion of rib fractures for front seat passengers. Panel **c** describes the proportion of rib fractures for rear seat passengers. The abbreviations of anatomical locations are as follows: AN, anterior; AL, anterolateral; L, lateral; PL, posterolateral; PS, posterior

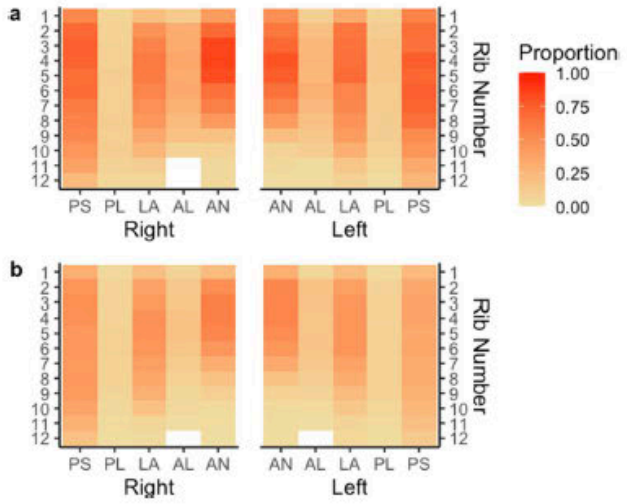


Fig. 6 Proportion of rib fractures by rib number, rib cage side, and anatomical location by thoracic spinal fracture. Panel **a** describes the proportion of rib fractures with a thoracic spinal fracture. Panel **b** describes the proportion of rib fractures without a thoracic spinal fracture. The abbreviations of anatomical locations are as follows: AN, anterior; AL, anterolateral; L, lateral; PL, posterolateral; PS, posterior

While position in a vehicle and BMI were not significant predictors in any of the models, both are well-established modifiers of injury severity and patterns of injury [23–27]. The extremes of BMI (Fig. 4, panels a and f) both have a higher incidence of anterior rib fractures, particularly for rib numbers two to seven. Notably, there were fewer rib fractures in rear-seated passengers (Fig. 5).

There are several limitations to this study, namely the absence of crash data for variables associated with injury severity such as speed of impact, vehicle intrusion, and point of crash impact [28–30]. Other safety devices, such as airbags, are not routinely reported in TARS or coronial autopsy reports and were therefore omitted from the study. Airbags protect the head and thorax during impact, and therefore the extent to which this protective effect is contributing or preventing rib fractures or other injuries remains unclear. Additionally, rib fractures may have been missed during gross examination, predominantly in the posterior anatomical location. Although cases with rib fracture patterns attributable to were removed from the study cohort, it is possible that some of the rib fractures documented at autopsy, particularly those in the anterior anatomical location, were caused by or worsened during resuscitation attempts.

This study demonstrates the effect of increasing age on rib fractures in vehicle crashes, as well as revealing an association with liver injury, pulmonary contusion, and hemothorax. Right leg fractures, splenic injury, and pelvic fractures may reflect the severity of impact.

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Research involving human participants and/or animals Not applicable.

Informed consent Not applicable.

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Supplementary Material to 6.11.1

Appendix A.

Table A1
Model output predicting one or more rib fractures

Deviance Residuals:

Min	1Q	Median	3Q	Max
-2.9129	0.1188	0.3145	0.5817	1.5832

Coefficients:

	Estimate	Standard Error	z value	Pr(>|z|)
(Intercept)	-1.74721	0.34785	-5.023	5.09e-07 ***
Age	0.04613	0.00808	5.709	1.14e-08 ***
Liver injury (1)	1.42924	0.41109	3.477	0.000508 ***
Lung contusion (1)	0.71049	0.28090	2.529	0.011428*
Haemothorax (1)	2.00830	0.27669	7.258	3.92e-13***

Signif. codes: 0 '***' 0.001 '**' 0.01 '*' 0.05 '.' 0.1 ' ' 1

(Dispersion parameter for binomial family taken to be 1)

Null deviance: 546.37 on 523 degrees of freedom
Residual deviance: 396.98 on 519 degrees of freedom
AIC: 406.98
BIC: 428.29

Number of Fisher Scoring iterations: 6

Table A1 is predicting the probability of rib fracture using the training data from Dataset 1, where the presence of one or more rib fracture was equal to 1.

Table A2

Model output predicting the number of ribs fractured

Deviance Residuals:

Min	1Q	Median	3Q	Max
-9.7630	-3.2974	-0.5812	3.0947	16.1159

Coefficients:

	Estimate	Standard Error	z value	Pr(>|z|)
(Intercept)	1.51507	0.73201	2.070	0.039186 *
Age	0.11278	0.01222	9.231	<2e-16 ***
Thoracic spinal fracture (1)	1.89514	0.60018	3.158	0.001724 **
Lower right extremity fracture (1)	1.48338	0.52424	2.830	0.004921 **
Spleen (1)	1.76754	0.59250	2.983	0.003046 **
Pelvic fracture (1)	1.29633	0.51216	2.531	0.011792 *
Aortic injury (1)	2.09845	0.54559	3.846	0.000142 ***
Lung laceration (1)	1.44577	0.58725	2.462	0.014284 *
Haemothorax (1)	2.52645	0.56345	4.484	9.8e-06 ***

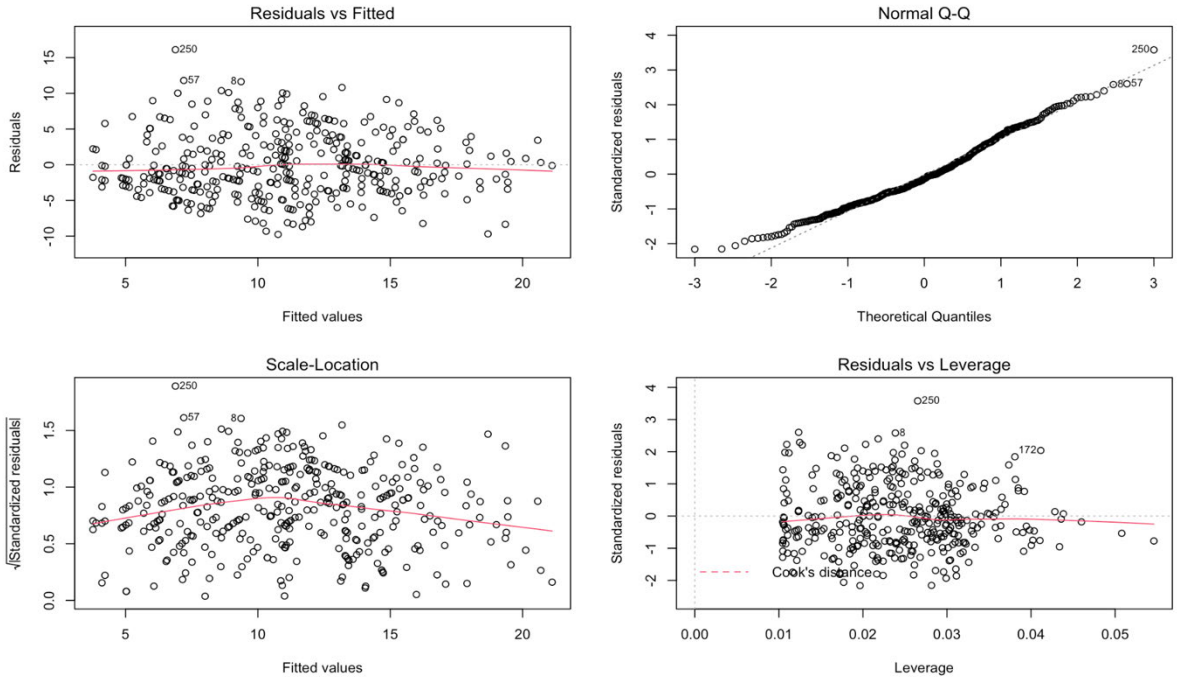
Signif. codes: 0 ‘***’ 0.001 ‘**’ 0.01 ‘*’ 0.05 ‘.’ 0.1 ‘ ’ 1

(Dispersion parameter for binomial family taken to be 1)

Residual standard error: 4.563 on 362 degrees of freedom
Multiple R-squared: 0.4121, Adjusted R-squared: 0.3991
F-statistic: 31.72 on 8 and 362 DF, p value: <2.2e-16

Table A2 is predicting the number of ribs fracture using the training data from Dataset 2.

Plot A1



Plot A1 describes the goodness of fit for the linear regression used to predict the number of rib fractures using training data from Dataset 2.

6.12 Seat belt wearing and abdominal injury

The previous study (Chapter 5.4) which assessed the relationship between seat belt marking and injury found that the presence of seat belt marking is not associated with an increase in the probability of abdominal injury and that BMI may have protective properties. The impact of BMI on injury severity remains unclear and it is likely that the relationship is complex. To explore this further, a series of logistic regressions were performed to determine if a relationship exists between BMI, belt wearing and abdominal injury. As it is difficult to categorise abdominal injury, a model was fitted for each abdominal organ, including the liver, spleen, kidney, and intestinal/mesenteric injury. Overall, there was a lower proportion of liver (n=336, $p<0.001$), kidney (n=80, $p<0.001$), splenic (n=214, $p<0.001$) and mesenteric/intestinal injury (n=136, $p<0.001$). Seat belt wearing or other variables included in the analysis were associated with liver or splenic injury. Increasing age was related to a decrease in the likelihood of kidney injury ($p <0.01$, OR= 0.98, 95% CI = 0.97, 0.99). As BMI increased, so too did the likelihood of intestinal/mesenteric injury ($p <0.001$, OR= 1.05, 95% CI = 1.02, 1.08). However, the overall probability of mesenteric/intestinal injury was low overall. For the full model output, please see Appendix C.17, C.18, C.19 and C.20.

6.13 Predicting pelvic injury

Pelvic fractures are usually the result of high energy impacts in a MVC. A summary of pelvic fractures observed at autopsy, including if they were unilateral or bilateral, with the occupant and crash characteristics is presented in Table 6.6.

Table 6.6 Occupant and crash characteristics of pelvic fractures

	Pelvic fracture, n = 587	Unilateral*, n = 249	Bilateral*, n = 209
	Mean, (range)	*Mean, (range)*	*Mean, (range)*
Age	44 (18 – 92)	44 (18 – 91)	45 (18 – 92)
BMI	27.0 (12.7 – 55.6)	26.7 (17.4– 54.3)	27.4 (12.7 – 55.6)
	n (%)	*n (%)*	*n (%)*
Sex			
Male	366 (62)	154 (62)	133 (64)
Female	221 (38)	95 (38)	76 (36)
Seat belt wearing			
Worn	285 (48.5)	123 (49.5)	119 (57)
Not worn	87 (14.8)	36 (14.4)	39 (19)
Unknown	215 (36.7)	90 (36.1)	51 (24)
Seating Position			
Driver	404 (68.9)	174 (69.9)	143 (68)
Front seat passenger	140 (23.8)	55 (22.1)	55 (26)
Rear seat passenger	42 (7.1)	19 (7.6)	11 (6)
Unknown	1 (0.2)	1 (0.4)	0
Ejection			
Ejected	59 (10)	20 (8)	29 (14)
Not Ejected	528 (90)	229 (92)	180 (86)

* The sum of unilateral and bilateral pelvic fractures does not equal total number of pelvic fractures as the location of some pelvic fractures was not specified in the autopsy report

Pelvic fractures were present in a significantly lower proportion (n=328, $p<0.01$) of those with skull fractures (n=818) with all factors included in the logistic regression increasing the probability. The first two factors in the regression model are related to crash factors. Pelvic fracture was found to be more likely for occupants who were wearing seatbelts ($p <0.01$, OR= 1.65, 95% CI = 1.18, 2.32) and occupants of newer vehicles compared to older vehicles ($p <0.01$, OR= 1.02, 95% CI = 1.01, 1.04). The remaining factors included in the model were injuries, including ring fractures, heart injury and femur fracture, which are all associated with high energy impacts. This suggests that the presence of a pelvic fracture is an indicator of high crash impact severity. For the full model output, please see Appendix C.21.

6.14 Extremities

Fractures to the upper and lower extremities are extremely common in MVCs. These can occur when the force of the crash is transferred through the limbs, such as through the knee-thigh-hip axis when there is forward excursion of the pelvis or when there is intrusion into the vehicle causing the instrument panel to make contact with the knees. Direct impacts can also result in fractures, particularly with vehicle intrusion in the foot pan or door-side impacts to the upper limbs. While single fractures are common and often non-life threatening, bilateral fractures of the larger limb bones, the humerus and the femur, usually indicate significant impact forces. Table 6.7 provides a summary of the occupant and crash characteristics associated with the bilateral humeral and femoral fractures.

Table 6.7 Occupant and crash characteristics of bilateral humeral and femoral fractures

	Bilateral humeral fractures, n=35	**Bilateral femoral fractures, n=110**
	mean, (range)	*mean, (range)*
Age	35 (18 – 77)	40 (18 – 100)
BMI	26.7 (19.4 – 42.5)	28.3 (17.3– 54.6)
	n (%)	*n (%)*
Sex		
Male	21 (60)	68 (62)
Female	14 (40)	42 (38)
Seat belt wearing		
Worn	20 (57.1)	55 (50)
Not worn	7 (20)	11 (10)
Unknown	8 (22.9)	44 (40)
Seating Position		
Driver	22 (63)	71 (64.6)
Front seat passenger	11 (31)	27 (24.5)
Rear seat passenger	2 (6)	9 (8.2)
Unknown	0	3 (2.7)
Ejection		
Ejected	7 (20)	5 (5)
Not Ejected	28 (80)	105 (95)

6.14.1 Upper and lower limb amputation in vehicle-related fatalities

S. O'Donovan, C. Van Den Heuvel, M.R.J. Baldock, R.W. Byard

Amputations in MVCs are rare as occupants are protected by the vehicle cabin by the sorts of impacts that can cause them. However, several cases of limb amputations were identified in the current sample and a study was conducted to establish common occupant or crash characteristics seen in upper and lower limb amputations in MVCs.

Statement of Authorship

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Publication Status	☒ Published ☐ Accepted for Publication ☐ Submitted for Publication ☐ Unpublished and Unsubmitted work written in manuscript style
Publication Details	O'Donovan, S, Van Den Heuvel, C, Baldock, MRJ & Byard, RW 2021, 'Upper and lower limb amputations in vehicle-related fatalities', *Journal of Forensic and Legal Medicine*, vol. 82, p. 102225, DOI: 10.1016/j.jflm.2021.102225.

Principal Author

Name of Principal Author (Candidate)	Siobhan O'Donovan		
Contribution to the Paper	Collected autopsy data, performed statistical analysis, wrote manuscript, acted as corresponding author		
Overall percentage (%)	85%		
Certification:	This paper reports on original research I conducted during the period of my Higher Degree by Research candidature and is not subject to any obligations or contractual agreements with a third party that would constrain its inclusion in this thesis. I am the primary author of this paper.		
Signature		Date	27/05/2022

Co-Author Contributions

By signing the Statement of Authorship, each author certifies that:

- the candidate's stated contribution to the publication is accurate (as detailed above);
- permission is granted for the candidate in include the publication in the thesis; and
- the sum of all co-author contributions is equal to 100% less the candidate's stated contribution.

Name of Co-Author	Corinna Van Den Heuvel		
Contribution to the Paper	Primary supervisor of Siobhan, helped to evaluate and edit the manuscript		
Signature		Date	04/07/2022

Name of Co-Author	Matthew Baldock

Contribution to the Paper	Co-supervisor of Siobhan, provide access to Traffic Accident Reporting System through Centre for Automotive Safety Research, helped to evaluate and edit the manuscript		
Signature		Date	27/5/2022

Please cut and paste additional co-author panels here as required.

Name of Co-Author	Roger Byard		
Contribution to the Paper	Co-supervisor of Siobhan, supervised collection of autopsy data, helped to evaluate and edit the manuscript		
Signature		Date	08/07/2022

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Research Paper

Upper and lower limb amputations in vehicle-related fatalities

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ABSTRACT

Traumatic limb amputation is rare in occupants following a motor vehicle collision (MVC). A retrospective analysis of autopsy reports at Forensic Science South Australia (FSSA) over a 19 year period from January 2000 to December 2018 was performed to determine the incidence of limb amputation in lethal collisions and to identify predisposing factors. Only 18 cases (1.54%) of occupant fatalities had a traumatic limb amputation with an age range of 18–78 years (mean 44.2 years), male to female ratio 13:5, and an average body mass index (BMI) of 28.5 (overweight). There were nine cases of upper limb amputation and nine cases of lower limb amputation (one case had both upper and lower limb amputations). Head on impacts without subsequent rollover were the most common collision type in both upper and lower amputation. The likely cause of limb amputation in vehicle crashes is, therefore, speed on impact rather than rollovers as has been previously suggested.

1. Introduction

Limb amputations are rare in motor vehicle collisions (MVCs) as the external structure of the vehicle often provides a level of protection to occupants that does not exist for motorcyclists or pedestrians. A review of the literature suggests that upper limb amputation are closely associated with vehicle rollovers[1–3] with fewer cases of lower limb amputations occurring. To evaluate amputations in vehicle occupants further, autopsy files at Forensic Science South Australia (FSSA) were retrospectively examined for cases of traumatic amputation limb in fatal MVCs.

2. Materials and methods

The autopsy files at FSSA were searched over a 19-year period from January 2000 to December 2018 for all occupant vehicle-related fatalities in which there had been upper or lower limb amputations. All vehicle fatalities within the state over that time had been reported to the State Coroner, and all had undergone full police, coronial and pathological investigations. Amputations were defined as either complete separation of a leg or arm, or part of a leg or arm, from the body, or where a devitalized limb was held only by strips of skin or soft tissue. Cases of amputation where there was severe disruption of the body with separation of multiple body parts were excluded from the study as were subsequent surgical amputations of devitalized limbs. The age and sex of de-identified decedents were recorded, along with the circumstances of the crash, the nature of the injury, the body mass index (BMI) and the cause of death. As the speeds of the collisions were not able to be accurately discerned from the available records these data were not included.

3. Results

Out of the total of 1164 vehicle-related deaths there were 18 cases (1.54%) with limb amputations consisting of 13 males and five females (Table 1). The age range was 18–78 years (average 44.2 years). The BMI ranged from 20.8 to 42.8 with an average of 28.5 (overweight). The BMI was not available for four cases due to the fracture or amputation of lower limbs. Fourteen of the decedents were drivers of the vehicle with three front (left) seat passengers and a single front middle passenger. Twelve decedents were wearing seat belts at the time of the crash and four were not. In two cases it is unknown if a seatbelt was worn at the time of the collision. Five occupants had been ejected from the vehicle and of those one had been wearing a seat belt at time of the collision.

The deaths in all of the decedents were attributed to multiple injuries occurring at the scene of the crashes. There were nine cases of single upper limb amputation: three through the forearm, five mid-humeral and one forequarter amputation (Table 2). All upper limb amputations

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1752-928X/

Table 1
Characteristics of Upper and Lower Limb amputations in South Australian road collisions from 2000 to 2018.

Case	Casualty type	Sex	Age	BMI	Belt	Ejected	Vehicle type	Limb amputation	Collision description
1	Driver	M	45	20.8	Yes	Yes	Car	LL, LR	Car vs. truck, head on
2	Front middle passenger	F	24	NA	No	Yes	Passenger van	LL, LR	Van vs. utility, head on
3	Driver	M	38	26.5	Yes	No	Utility	UR	Driver's side to driver's side, head on
4	Driver	M	62	26.2	Yes	No	Car	UR	head on
5	Driver	M	30	21.6	Yes	No	Car	LR	Car vs. prime mover, head on
6	Driver	M	45	33.8	Yes	No	Utility	UR, LR	Utility vs. truck, head on
7	Driver	M	53	26.5	Yes	No	Utility	UR	Utility vs. truck, head on
8	Driver	F	78	27.1	Yes	No	Car	UR	Car vs. prime mover, head on
9	Front left passenger	M	67	29.7	Yes	No	Car	UL	Car vs. prime mover, head on
10	Front left passenger	M	25	22.2	Yes	No	Car	LL, LR	Head on
11	Driver	M	57	34.8	Fitted – Unknown^	No	Station wagon	UR	Head on
12	Driver	F	67	42.2	Yes	No	Car	LL	Car vs. utility, head on
13	Driver	M	25	23.2	Fitted – Unknown^	Yes	Utility	LL, LR	Utility vs. stone wall, hit fixed object with roll over
14	Driver	M	23	27.9	No	No	Car	LL	Car vs. stobie pole, hit fixed object
15	Driver	M	18	NA	No	Yes	Car	LR	Car vs. prime mover, right angle
16	Driver	F	44	35.3	Yes	No	Car	UR	Car vs. prime mover, struck driver's side, right angle
17	Front left passenger	F	69	NA	Yes	No	Station wagon	UL	Car vs. freight train, right angle
18	Driver	M	26	NA	No	Yes	Passenger van	LL, LR	Van vs. 4WD, head on

* Driver positioned on the right of the vehicle, M = Male, F = Female, UR = Upper right limb, UL = Upper left limb, LR = Lower right limb, LL = Lover left limb, ^refers to a seat belt fitted in the vehicle it is unknown if it was worn at time of collision.

Table 2
Upper limb amputations with associated limb fractures and skull fractures.

Case	Casualty type	Collision type	Belt	Ejected	Amputation	Upper limb fractures	Lower limb fractures	Skull fractures
3	Driver[a]	Head on	Yes	No	R Mid-humerus	No	Bilateral	Yes; Closed
4	Driver[a]	Head on	Yes	No	R Forearm	No	No	No
6	Driver[a]	Head on	Yes	No	R Mid-humerus	Left	Right amputation	Yes; Open
7	Driver[a]	Head on	Yes	No	R Forearm	Left	Bilateral	Yes; Closed
8	Driver[a]	Head on	Yes	No	R Upper limb	Left	No	Yes; Open
9	Front left passenger	Head on	Yes	No	L Forearm	No	Bilateral	Yes; Open
11	Driver[a]	Head on	UK	No	R Mid-humerus	No	No	Yes; Closed
16	Driver[a]	Right angle	Yes	No	R Upper limb	No	No	Yes; Closed
17	Front left passenger	Right angle	Yes	No	L Forequarter	Right	Bilateral	Yes; Open

[a] Driver positioned on the right of the vehicle, L = Left, R = Right, UK = Unknown.

Table 3
Lower limb amputations with associated limb fractures and skull fractures.

Case	Casualty type	Collision type	Belt	Ejected	Amputation	Upper limb fractures	Lower limb fractures	Skull fractures
1	Driver[a]	Head on	Yes	Yes	BL Hindquarter	Right arm	NA	Yes; Open
2	Front middle passenger	Head on	No	Yes	BL Below knee	No	No	Yes; Closed
5	Driver[a]	Head on	Yes	No	R Above knee	No	L	No
6	Driver[a]	Head on	Yes	No	R Above knee	Left fracture and right amputation	No	Yes; Open
10	Front left passenger	Head on	Yes	No	BL Foot	Bilateral	No	Yes; Open
12	Driver[a]	Head on	Yes	No	L Foot	No	No	No
13	Driver[a]	Hit fixed object	UK	Yes	BL Above knee	Bilateral	NA	No
14	Driver[a]	Hit fixed object	No	No	L Above knee	No	No	Yes; Closed
15	Driver[a]	Right angle	No	Yes	R Hindquarter	Bilateral	No	Yes; Open
18	Driver[a]	Head on	No	Yes	BL Below Knee	Bilateral	NA	Yes; Closed

[a] Driver positioned on the right of the vehicle, L = Left, R = Right, BL = Bilateral, UK = Unknown.

occurred on the window side (ipsilateral) for both drivers or passengers.

Ten cases had lower limb amputations, with five being bilateral. The latter involved amputations of the feet, two cases through the upper calves, above the knees and hindquarters. Single lower limb amputations included a foot, three above the knee and a single hindquarter amputation (Table 3). One decedent had both upper and lower limb amputations.

The most common crash type involved head on collisions (13 cases) with three collisions involving right angle impacts and two collisions caused by impact with a fixed object. Data on vehicle kinematics following the initial impact was sometimes limited in some cases and therefore the possibility of a roll over following the head on collision could not be excluded in three cases. It appeared, however, that all vehicle were either travelling at high speed on impact or were in high speed road zones (80 km/h and above).

4. Discussion

Extremity fractures are frequent in high energy vehicle collisions as impact forces cause intrusion of the vehicle structure into the cabin with

lower limb entrapment and uneven force distribution disproportionately transferred to the limbs.[4] Amputations are infrequent, as demonstrated in the current study with only 1.54% of autopsies examined having a limb amputation. In the literature amputations of the upper limb have been associated with vehicle rollovers and unbelted occupants with the occupant either being ejected or moving freely within the vehicle cabin during the collision.[1–3] A potential mechanism of upper limb amputation may involve having a limb outside the vehicle (i.e. positioned through a window) when a rollover collision occurs. As the vehicle rolls, the arm may then be crushed between the vehicle structure and the ground causing either amputation or more commonly, devitalisation necessitating surgical amputation.[2] Ball et al. and Bakker et al. describe a subset of surgical amputations with the majority of collisions being a roll over.[1,2] The occupants were often belted preventing ejection and exposing the limb to the majority of the roll over force.[1] This is consistent with Barmparas et al.'s finding that occupants with upper limb injuries, when compared to lower limb injuries, often had less severe injuries overall.[3] The details of eight cases of upper limb amputations are summarised in Table 1 including head-on impacts without subsequent rollovers or right (driver) sided impacts. These findings differ from previous cases reported by Ball et al. and Bakker et al.[1–3] All upper limb amputations occurred on the window (ipsilateral) side arm, and eight of the nine cases were known to be wearing seat belts.

Lower limb amputations are seldom reported in the literature, possibly because lower extremity trauma is associated with increased mortality and injury severity which results in these cases being excluded from clinical trauma datasets as they do not reach hospital.[3,5,6] The current study is, therefore, useful in specifically assessing occupant lower limb amputations.

Restraint use was identified in five cases of lower limb amputation and occupant ejection was reported in three. It is likely that the safety benefit of the seat belts in this cohort is negligible due to the magnitude of the impact forces. Additionally, there may be restraint failure or ejection despite restraint use. There are two proposed mechanisms for restraint failure: the first involves inertial unlatching of the buckle component, often during a roll over or lateral impact and the second being a failure in the restraint webbing.[7,8] Crash testing of a restraint system can be used to detect restraint failure however, testing was not performed as part of this retrospective study.

Thus the primary reason for upper and lower limb amputations in lethal collisions in this series was speed on impact rather than collision type or occupant kinematics during the collision. Doecke et al. outlined the significance of speed as a primary moderator of collision outcome and associated injury severity.[9,10] It has been shown, for example, that cardiac injuries are more likely to occur with higher speeds at the time of impact.[11] Unfortunately a limitation to this study is that the exact speed on impact was not available for all cases. However, using the crashes details as reported in the coronial autopsy files it was appears that all vehicles were either travelling at high speed on impact or were in high speed road zones (80 km/h and above). Head on collisions are also characterised by limited driver reaction time and opportunity to reduce speed,[10] thus carrying an increased risk of severe injury and fatality risk and, as this series has shown, an increased risk of amputation.

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Ethical approval

By Forensic Science SA management (no number provided).

Declaration of competing interest

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

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Chapter Seven: Thesis general discussion and future considerations

7.1 Summary of publications

This thesis combined an existing police-reported crash database of motor vehicle occupant fatalities with injury information from coronial autopsy reports in order to investigate relationships between occupant characteristics, crash factors and injury patterns. The wide-ranging subjects evaluated in the publications presented in this thesis point to the multi-faceted ways in which the combination of crash data and injury data can be used to explore questions related to fatal MVCs. The following is a summary of each of the publications in this thesis.

The study presented in Chapter 3.5 found that there was an upward trend in methamphetamine (MA) detection in driver fatalities, which, in the context of increasing use of MA in SA, highlights the need for continued monitoring of MA detection rates and the application of countermeasures. The significant downward trend in BAC above 0.05g/100mL in driver fatalities may be attributable to the success of safety countermeasures such as random breath testing but this decline appears to have plateaued, indicating that there is likely to be a subset of drivers who continue to drink and drive and are not deterred by random breath testing.

The study of cardiac disease and driver fatality (Chapter 3.6) found no difference in the incidence of severe coronary artery atherosclerosis or cardiomegaly in drivers compared to a control passenger group and so it was concluded that cardiac disease does not appear to be contributing to fatal crash risk. The case series and overview of vehicle-related suicides (Chapter 3.7) addressed some of the difficulties in determining if a vehicle crash is deliberate. Although deliberate crashes and other forms of vehicle-related suicide are not included in TARS, this overview highlights the broader effects of the readily accessible nature of motor vehicles that facilitates suicide.

The study presented in Chapter 5.9 assessed the incidence of seat belt marking by season of year to elucidate a common understanding in forensic investigation that had yet to be substantiated in the literature. Season of year was used as a proxy measure for the amount of clothing worn, with more clothing being worn in winter than in summer. The incidence of seat belt marking was significantly lower in the months of winter than in the months of summer. Although imposition of clothing has been thought to mask or protect the skin from seat belt marking, this was the first study of its kind to demonstrate this phenomenon empirically.

Five cases of drivers who were suspended upside down by the lap component of the seat belt following vehicle roll-over were also reported in Chapter 5.10. The cause of death in four of these cases was attributed to positional asphyxia and one was attributed to significant head trauma combined with positional asphyxia. The suspension by the seat belt across the abdomen in the upside down position could contribute to the compression of the lungs from the abdominal viscera. An important feature of these cases was the angular position of the neck which further compromised the upper airway.

Evaluating the number and patterns of rib fractures (Chapter 6.11.2) found increasing age was the most significant predictor of rib fractures. This is likely related to the reduced tolerance to chest deflection in older occupants as the result of age-related changes to the rib cage. An increasing number of rib fractures was associated with the presence of other injuries suggesting a relationship between the number of ribs fractured and overall injury severity. In the same chapter, a series of cases of limb amputation in MVCs (Chapter 6.13.2) suggested a major role for speed of impact rather than roll-overs as had been suggested previously. Although exact impact speeds were not available in the larger data set, the autopsy reports of this small case series all reported crash impacts indicative of a high speed of impact.

7.2 Discussion

Several distinct themes emerged from the results presented in Chapter Six and the findings of the various publications. Age was considered a co-variate factor throughout the analysis and, expectedly, a strong relationship between increasing age and increased risk of cervical and thoracic spine and rib fractures was found (Chapter 6.11.2). The increased risk of these injuries is likely associated with age-related degeneration and increased fragility (i.e., greater susceptibility to trauma), as discussed in Chapter Four (Li, Braver & Chen 2003). The effect of frailty could explain the results of the decision trees in Chapter 6.6, whereby location of death was more likely to be in hospital if the occupant was older than 73 years old. Frailty decreases physiological reserve and affects an older occupant's capacity to respond to injury, even if that injury may not be considered severe in a younger occupant (Kent et al. 2009). However, increasing age did not predict atlanto-occipital injury despite the strong relationship between increasing age and cervical spinal injury. Furthermore, increasing age decreased the likelihood of head injury, hinge fractures and kidney injury. There are two possible explanations for these findings. The first of these is that these injuries are likely to be related to high severity impact forces, which are more likely among younger drivers exhibiting risk taking behaviour, such as high speeds. Secondly, it may be that the occurrence of these injuries is heavily dependent on particular injury mechanisms and so the factors more useful for prediction of these injuries are those related to these mechanisms (e.g., closed head injury without skull fracture predicting atlanto-occipital injury).

In addition to older age, the likelihood of thoracic spinal fracture was also linked to greater body mass. Analysis in Chapter 6.12 also found that the likelihood of mesenteric/intestinal injury increased with higher BMI, although the increase was minor. Increasing BMI was also shown to increase the likelihood of seat belt marking, whilst there was no increase in abdominal injury related to the seat belt wearing or presence of a marking

(Chapter 5.8). These findings suggest that BMI may have protective properties for some injury types, as suggested in the literature in Chapter Five (Arbabi et al. 2003; Wang, SC et al. 2003)**,** whilst the likelihood of other injury types increases (Arbabi et al. 2003; Hoebee et al. 2021; Kent, Forman & Bostrom 2010; Reed, Ebert-Hamilton & Rupp 2012; Rupp et al. 2013), potentially due to the increased kinetic energy generated by more mass. Further research on the relationship between BMI and injury is required.

Overall, seat belt wearing did not increase the presence of chest or internal abdominal injury. This is possibly due to the sample being based on fatal crashes in which the majority of occupant fatalities experienced high energy impacts that would have been equally if not more injurious without a seat belt. Seat belt wearing did increase the probability of pelvic fractures (Chapter 6.13) and bilateral pelvic fractures were also more common in the presence of seat belt marking (along with increasing BMI) (Chapter 5.8). These findings do not suggest that seat belts cause pelvic injuries but, rather, these findings more likely indicate that, when an occupant is in crash of a significant magnitude, the seat belt is correctly applying the counterforce across the pelvis, preventing forward excursion and slipping above the anterior superior iliac spine. The inclusion of other injuries in the model predicting pelvic fracture also suggests that this relationship is related to crash magnitude and severity.

As discussed in Chapter 6.5, improved vehicle safety may be increasing the threshold of impact severity that is required in a crash to result in a fatal outcome. It is noteworthy that there was a small but statistically significant increase in average vehicle age over the course of the time period studied, suggestive of a protective effect of new vehicles. This protective effect would incorporate improvements in vehicle design that enhance occupant protective but could also reflect the effect of vehicle safety technologies (e.g., advanced braking systems) that can reduce impact severity in the event of a crash (Doecke et al. 2021; Doecke, Thompson & Stokes 2020; Jermakian 2011; Reagan et al. 2018). In additional to the effects

of vehicle safety, advances in emergency response and treatment may be reducing the mortality associated with certain injuries. Comparing patters of fatal and serious injury with reference to crash severity and vehicle safety measures could be an avenue of future research (see Section 7.3 below).

7.3 Limitations and future directions

The retrospective design of the current study inevitably resulted in limitations in data capture. Having such an extensive time frame for the study (40 years) meant that data capture was limited in the early years of the time frame due to improper record keeping or files that could not be verified. While actions were made to minimise the impact of incomplete data capture, there were inevitably periods in time or certain variables for which important data were missing. This was further complicated by the de-identification of cases in TARS, which introduced difficulties in case matching.

Another limitation in regard to data capture was the unavailability of data for a number of important variables. Speed of impact, vehicle intrusion, point of impact and airbag deployment could not be verified through autopsy reports and was not included in the TARS database. As highlighted in Chapter 6.8.2, this likely limited some of the analysis in this thesis, particularly in distinguishing between types of head injury. This does, however, create an avenue for future research. For example, future smaller scale studies could extract a subset of these data using full Coroner's files to undertake analyses that were not possible using the dataset available for this thesis.

Future research could also incorporate injuries from occupants who sustained injuries in a MVC but who survived. Such studies could consider injuries along a spectrum, with fatalities as the end point, to further investigate which factors most significantly contribute to fatal occupant outcomes. Variables related to vehicle safety, such as occupant protection features and crash mitigation technologies, could be incorporated into the analysis to

demonstrate the effects on outcomes of newer vehicles. Furthermore, a combination of fatal and serious injury information could help address the question raised in Chapter 6.5 to determine if fatal crashes now typically involve high impact severity resulting in multiple injuries more than crashes resulting in serious injury. It may also help elucidate some of the relationships that remain ambiguous when examined only using fatal injury data (e.g., age and BMI).

Future research could also benefit from the use of data downloaded from vehicle Event Data Recorders (EDRs) (Brumbelow 2019; Doecke 2017; Elsegood, Doecke & Ponte 2020; Gabauer & Gabler 2005). These provide accurate data for a number of vehicle control variables in the lead up to, and including, the point of impact in a crash. These typically include speed of impact (which can be inaccurate when based on crash reconstruction), braking patterns and steering input. EDRs provide a new data source that could readily be incorporated into existing databases such as TARS or used to establish new databases that are multifaceted, and which also incorporate injury data.

The decline in the number of full internal autopsies being conducted on occupant fatalities in SA may present a challenge for future research into the patterns of injury. The number of cases captured dropped considerably in the final five years of the study time frame, with the majority of cases undergoing pathology review. For the proportion of cases that survive to hospital, autopsy may not be as crucial as there is clinical documentation and radiographic imaging can provide sufficient injury information. However, other crashes may result in rapid, if not instantaneous, death for which an autopsy is the only method of obtaining an accurate description of the fatal injuries sustained. The reduction in the proportion of fatally injured road users for whom full autopsies are conducted will hinder future research that aims to link crash and occupant characteristics to specific injury patterns or types.

In summary, the work presented in this thesis explores the relationship between occupant characteristics and crash factors that influence the patterns of injury seen at autopsy. The series of publications and analyses highlight the nexus of factors that alter the pattern of fatal injuries and contribute to fatal outcomes in MVCs. The amalgamation of various crash-related databases can provide greater understanding of these contributing factors in the aim of reducing and eliminating motor vehicle fatalities.

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Appendix A

A.1 Analysis
Logistic regression predicting fatal crashes from hospital admitted crashes with post-hoc testing

Sample size of training dataset: 6653
Sample size of test dataset: 4436

Table A.1.1 Logistic regression model output predicting fatality using training data

```
Call:
glm(formula = crash severity ~ age + sex + ejected +
    belt + crash type + speed limit + road alignment, family =
"binomial")

Deviance Residuals:
    Min       1Q   Median       3Q      Max
-1.9461  -0.5222  -0.3666  -0.2478   2.9345

Coefficients:
                   Estimate Std. Error    z value   Pr(>|z|)
(Intercept)          -0.462460   0.423699  -1.091  0.275061
Age                   0.014819   0.002031   7.295  2.98e-13 ***
Sex (Female)         -0.608468   0.087586  -6.947  3.73e-12 ***
Not ejected          -1.418586   0.184308  -7.697  1.40e-14 ***
Belted (No)           1.305212   0.117342  11.123  < 2e-16 ***
Belted (Unknown)     -0.402043   0.126167  -3.187  0.001440 **
Belted (Not fitted)   0.931275   0.464645   2.004  0.045041 *
Hit fixed object     -0.452105   0.116182  -3.891  9.97e-05 ***
Side swipe           -0.774777   0.245644  -3.154  0.001610 **
Right angle          -0.633287   0.145031  -4.367  1.26e-05 ***
Rear end             -1.480348   0.244551  -6.053  1.42e-09 ***
Hit object^          -1.011492   0.401857  -2.517  0.011834 *
Roll over            -1.231244   0.158465  -7.770  7.86e-15 ***
Right turn           -0.797656   0.224837  -3.548  0.000389 ***
Hit parked car       -1.149045   0.348350  -3.299  0.000972 ***
Left road - OFC      -1.594390   0.486175  -3.279  0.001040 **
Other                -0.994332   0.674217  -1.475  0.140268
speedlimit50         -1.052946   0.390651  -2.695  0.007031 **
speedlimit60         -0.702466   0.366775  -1.915  0.055460 .
speedlimit70         -0.806869   0.528938  -1.525  0.127147
speedlimit80          0.020673   0.370906   0.056  0.955552
speedlimit90          0.714720   0.443878   1.610  0.107360
speedlimit100         0.231159   0.361832   0.639  0.522917
speedlimit110         0.435906   0.361090   1.207  0.227357
Curved - obscured    -0.616666   0.161311  -3.823  0.000132 ***
```

```
Curved - open         0.104261   0.101412   1.028  0.303904
---

OFC – Out of control
^ - Hit object includes pedestrian, animal or object on road

Signif. codes:  0 ‘***’ 0.001 ‘**’ 0.01 ‘*’ 0.05 ‘.’ 0.1 ‘ ’ 1

(Dispersion parameter for binomial family taken to be 1)

    Null deviance: 4916.6  on 6652  degrees of freedom
Residual deviance: 4228.5  on 6627  degrees of freedom
AIC: 4280.5
BIC: 4457.4

Number of Fisher Scoring iterations: 6
```

Table A.1.2 Confusion matrix output predicting fatality using test data (Kuhn, 2021)

```
Confusion Matrix and Statistics

                   Reference
  Predicted        Hospital admitted    Fatal
  Hospital admitted       3903           439
  Fatal                     25           69

               Accuracy : 0.8954
                 95% CI : (0.886, 0.9043)
    No Information Rate : 0.8855
    P-Value [Acc > NIR] : 0.01916

                  Kappa : 0.2006

 Mcnemar's Test P-Value : < 2e-16

            Sensitivity : 0.9936
            Specificity : 0.1358
         Pos Pred Value : 0.8989
         Neg Pred Value : 0.7340
             Prevalence : 0.8855
         Detection Rate : 0.8798
   Detection Prevalence : 0.9788
      Balanced Accuracy : 0.5647

       'Positive' Class : FALSE
```

The number of crashes that were predicted correctly as hospital admitted was 3903 crashes. 69 crashes were correctly predicted as fatal crashes however, 25 crashes were incorrectly predicted as fatal crashes when they were hospital admitted and 439 fatal crashes were correctly predicted as hospital admitted crash.

Table A.1.3 Post-hoc test using Tukey on speed limit (Hothorn, Bretz & Westfall 2008)

```
Simultaneous Tests for General Linear Hypotheses

Multiple Comparisons of Means: Tukey Contrasts


Linear Hypotheses:
               Estimate Std. Error  z value Pr(>|z|)
50 - 40 == 0    -0.8966     0.4439  -2.020   0.41940
60 - 40 == 0    -0.6065     0.4275  -1.419   0.82020
70 - 40 == 0    -0.7480     0.5763  -1.298   0.87875
80 - 40 == 0     0.1342     0.4296   0.312   0.99998
90 - 40 == 0     0.8507     0.4978   1.709   0.63732
100 - 40 == 0    0.4808     0.4224   1.138   0.93612
110 - 40 == 0    0.7259     0.4249   1.708   0.63774
60 - 50 == 0     0.2901     0.2092   1.386   0.83734
70 - 50 == 0     0.1486     0.4390   0.338   0.99997
80 - 50 == 0     1.0309     0.2150   4.794   < 0.001 ***
90 - 50 == 0     1.7473     0.3299   5.297   < 0.001 ***
100 - 50 == 0    1.3774     0.2048   6.727   < 0.001 ***
110 - 50 == 0    1.6225     0.2112   7.681   < 0.001 ***
70 - 60 == 0    -0.1415     0.4084  -0.347   0.99996
80 - 60 == 0     0.7407     0.1590   4.658   < 0.001 ***
90 - 60 == 0     1.4572     0.2932   4.970   < 0.001 ***
100 - 60 == 0    1.0873     0.1560   6.969   < 0.001 ***
110 - 60 == 0    1.3324     0.1629   8.178   < 0.001 ***
80 - 70 == 0     0.8823     0.4158   2.122   0.35495
90 - 70 == 0     1.5987     0.4826   3.313   0.01633 *
100 - 70 == 0    1.2288     0.4160   2.954   0.05082 .
110 - 70 == 0    1.4739     0.4183   3.523   0.00795 **
90 - 80 == 0     0.7165     0.2972   2.411   0.20053
100 - 80 == 0    0.3465     0.1490   2.325   0.23998
110 - 80 == 0    0.5916     0.1575   3.757   0.00328 **
100 - 90 == 0   -0.3699     0.2906  -1.273   0.88922
110 - 90 == 0   -0.1248     0.2923  -0.427   0.99984
110 - 100 == 0   0.2451     0.1115   2.198   0.30849
---
Signif. codes:  0 ‘***’ 0.001 ‘**’ 0.01 ‘*’ 0.05 ‘.’ 0.1 ‘ ’ 1
(Adjusted p values reported -- single-step method)
```

Higher speed limits, as compared to lower speed limits, were consistently more likely to predict fatal crash

Table A1.4 Post-hoc test using Tukey on area of crash (Inner Adelaide, Outer Adelaide and Country) (Hothorn et al. 2008)

```
Simultaneous Tests for General Linear Hypotheses

Multiple Comparisons of Means: Tukey Contrasts


Linear Hypotheses:
                  Estimate Std.  Error   z value  Pr(>|z|)
Outer – Inner   == 0   0.5850     0.6197   0.944   0.5833
Country – Inner == 0   0.2629     0.6292   0.418   0.8990
Country - Outer == 0  -0.3221     0.1186  -2.717   0.0146 *
---
Signif. codes:  0 ‘***’ 0.001 ‘**’ 0.01 ‘*’ 0.05 ‘.’ 0.1 ‘ ’ 1
(Adjusted p values reported -- single-step method)
```

Area of crash either does not predict fatality or is being captured by another variable in the model (e.g., speed limit). Crashes that occur in the country have a significantly more likely to predict a hospital admitted crash that crashes that occur in outer Adelaide. This may be related to more hospital admitted crashes occurring in outer Adelaide.

Reference list

Hothorn, T, Bretz, F & Westfall, P 2008, ‘Simultaneous Inference in General Parametric Models’, *Biometrical Journal,* vol. 50. no. 3, pp. 346-363.

Kuhn, M. 2021. caret: Classification and Regression Training. R package version 6.0-90. https://CRAN.R-project.org/package=caret

R Core Team. 2021. R: A Language and Environment for Statistical Computing. Vienna, Austria. Available at: https://www.R-project.org/.

Appendix B

Table B.1 Classifications of causes of death

Cause of Death	Description
Closed head injury, no skull fracture	A blunt force head injury (e.g., diffuse axonal injury, focal contusion, brainstem laceration) without the presence of a skull fracture
Closed head injury, skull fracture	A blunt force head injury (e.g., focal contusion, cerebral laceration) that involves a skull fracture
Open head injury	A blunt force head injury where the dura is exposed and open, may involve the avulsion of brain matter, includes penetrating head injury
Chest injury	Blunt force chest injury involving one or more of the following injuries: rib fracture, rib cage disruption, heart, lungs and cardiopulmonary vasculature
Abdominal injury	Blunt force abdominal injury involving one or more of the following injuries: liver, kidneys, gastrointestinal tract, mesentery, spleen, genitourinary
Spinal injury	Injury (e.g., fracture dislocation, spinal cord transection) to the spine at any level
Atlanto-occipital	Injury (e.g., subluxation, distraction, fracture dislocation) of the atlanto-occipital joint
Injury to the pelvis/extremities	Injury (e.g., significant laceration, fracture, amputation) of any of the extremities
Asphyxial	Cause of death due to asphyxia which include crush, postural, aspiration of gastric contents
Crush injury	Crush injury to any part of the body that results in death
Decapitation	Decapitation of head
Complication	Hypoxic ischaemic encephalopathy, pulmonary oedema, bronchopneumonia, respiratory failure, multi-organ failure, congestive heart failure, myocardial infarction, pericarditis, sepsis, adult respiratory distress syndrome
Multiple	Multiple injuries
Incineration	Incineration

Table B.2 Variables used in data collection from coronial autopsy reports and injury descriptions of each variable

Variable	Injury type	Injury Description	Variable type
bf_nf	Blunt force head injury, no skull fracture, closed	Subarachnoid; epidural; subdural; extradural haemorrhage; cerebral contusion and/or laceration; brain swelling; intraventricular haemorrhage; neuropathological (APP positive), dura mater intact	0 = No 1 = Yes
brainstem	Brainstem	brainstem laceration; pontomedullary transection	0 = No 1 = Yes
bf_f	Blunt force head injury, skull fracture, closed	Fractures to the skull including to the calvarium; fronto-basal region and basilar region, dura mater intact	0 = No 1 = Yes
basilar	Basilar skull fracture	Fractures involving the middle cerebral fossa and/or posterior cerebral fossa, petrous temporal bones, sphenoid and sella turcica	0 = No 1 = Yes
hinge	Hinge type skull fracture	Bilateral fracture petrous temporal bones causing distraction from anterior section of the basilar skull and posterior	0 = No 1 = Yes
ring	Ring type skull fracture	Fracture through floor of the middle fossa of the base of the skull and the squamous region of the temporal and occiptial bones resulting in a incomplete or complete separation of the rim of the foramen magnum the base of skull	0 = No 1 = Yes
fronto-basal	Fracture to the fronto-basal region	Infra-orbital ring fracture: orbital blow out; orbital roof; ethmoid bone; anterior cranial fossa	0 = No 1 = Yes
calvarium	Fracture to the calvarium	Fracture to the temporal; frontal; parietal; occipital bones	0 = No 1 = Yes
nasal_zygoma	Fracture of the zygoma and/or nasal bone		0 = No 1 = Yes
maxilla	Maxilla fracture		0 = No 1 = Yes
mandible	Mandible fracture		0 = No 1 = Yes
panfacial	Panfacial fracture	Facial fractures involving 2 or more regions of the face	0 = No 1 = Yes
bf_ext	Blunt force head injury, open with skull fracture	Open skull fracture with disruption to dura mater; may involve the expulsion of brain tissue or complete brain avulsion	0 = No 1 = Yes

cf_spine	Cervical spine fracture	Vertebral fracture or fracture dislocation of cervical spine (does not indicate spinal cord transection)	0 = No 1 = Yes
tf_spine	Thoracic spinal fracture	Vertebral fracture or fracture dislocation of thoracic spine (does not indicate spinal cord transection)	0 = No 1 = Yes
lf_spine	Lumbar spinal fracture	Vertebral fracture or fracture dislocation of lumbar spine (does not indicate spinal cord transection)	0 = No 1 = Yes
atlanto	Atlanto-occipital fracture	Subluxation, distraction or fracture dislocation of the atlanto-occipital joint	0 = No 1 = Yes
trans_spinal_cord	Spinal cord transection (any location)	Spinal cord transection at any spinal injury level	0 = No 1 = Yes
ul_ext	Upper left extremities fracture	Fracture to the upper left limb, any location	0 = No 1 = Yes
ur_ext	Upper right extremity fracture	Fracture to the upper right limb, any location	0 = No 1 = Yes
scapula	Scapula fracture		R = Right L = Left B = Bilateral
humerus	Humerus fracture		R = Right L = Left B = Bilateral
radius_ul	Radius and/or Ulna fracture		R = Right L = Left B = Bilateral
wrist_hand	Hand fracture		R = Right L = Left B = Bilateral
u_ext	Upper Extremity fracture (any location)	Fracture to the upper limbs, any location	0 = No 1 = Yes
ll_ext	Lower left extremity fracture	Fracture to the lower left limb, any location	0 = No 1 = Yes
lr_ext	Lower right extremity fracture	Fracture to the lower right limb, any location	0 = No 1 = Yes
femur	Femur fracture		R = Right L = Left B = Bilateral
tib_fib	Tibia or Fibula fracture		R = Right L = Left B = Bilateral
ankle_foot	Foot fracture		R = Right L = Left B = Bilateral
l_ext	Lower extremity fracture (any location)	Fracture to the lower limb, any location	0 = No 1 = Yes

liver	Liver injury	3+ contusion = subcapsular, >50% surface area; ruptured subcapsular or parenchymal; intraparenchymal >10cm in diameter or expanding; major 3+ laceration = >3cm parenchymal depth; major duct involvement; moderate [OIS I, II]	0 = No 1 = Yes
kidney	Kidney injury	3+ contusion = subcapsular, >50% surface area; major; large 3+ laceration = >1cm parenchymal depth of renal cortex, no collecting system rupture or urinary extravasation; moderate [OIS III]	0 = No 1 = Yes
abd_vas	Abdominal vascular injury	Injury (laceration, transection, avulsion) to the renal, mesenteric, iliac or other abdominal arteries or veins	0 = No 1 = Yes
spleen	Splenic injury	3+ contusion = subcapsular, >50% surface area; ruptured subcapsular or parenchymal; intraparenchymal >5cm diameter; major [OIS III] 3+ laceration = >3cm parenchymal depth or involving trabecular vessels; moderate [OIS III]	0 = No 1 = Yes
mesentery	Mesenteric or intestinal injury	3+ mesenteric = laceration major 3+ small bowel and large bowel = perforation; full thickness laceration <50% circumference [OIS II]	0 = No 1 = Yes
pelvis	Pelvic fracture (any location)	Fracture of pelvis at any location	0 = No 1 = Yes
pelvis_ps	Pubic Symphysis	Fracture or widening	0 = No 1 = Yes
pelvis_sij	Sacro iliac joint	Distraction, dislocation, fracture, separation	R = Right L = Left B = Bilateral
pelvis_uni	Pelvic fracture Unilateral		R = Right L = Left B = Bilateral
pelvis_bi	Pelvic fracture Bilateral		0 = No 1 = Yes
heart	Heart injury	3+ contusion = major 3+ laceration = laceration NFS; no perforation; no chamber invovlement	0 = No 1 = Yes

cardio_vas	Heart vascular injury	Injury (laceration, transection, avulsion) to the cardiac arteries or veins	0 = No 1 = Yes
aorta	Aortic injury	Laceration, transection or avulsion of thoracic or abdominal aorta	0 = No 1 = Yes
contusion_lung	Lung contusion	3+ = contusion NFS; unilateral major; extensive; massive, bilateral NFS	0 = No 1 = Yes
laceration_lung	Lung laceration	3+ = laceration NFS Lacerations under 3cm were not included	0 = No 1 = Yes
avulsion_lung	Lung avulsion	Avulsion of the lung from the bronchi or trachea	0 = No 1 = Yes
pneumothorax	Pneumothorax	Collapsed lung	0 = No 1 = Yes
haemothorax	Haemothorax	Greater than 100mL of frank blood in one or both pulmonary cavities	0 = No 1 = Yes
lung_vas	Lung vascular injury	Injury (laceration, transection, avulsion) to the pulmonary arteries or veins	0 = No 1 = Yes
chestwall	One or more rib fracture	One or more rib fractures present	0 = No 1 = Yes
sternum	Sternum fracture		0 = No 1 = Yes
diaphragm	Diaphragm injury	Laceration or rupture of diaphragm and/or herniation of gastric content into thoracic cavity	0 = No 1 = Yes

Semicolon separate injury descriptor that are comparable in severity

Appendix C

The following packages were used in R studio

Packages:

caret
Max Kuhn (2021). caret: Classification and Regression Training. R package version 6.0-90. https://CRAN.R-project.org/package=caret

c.tree
Torsten Hothorn, Kurt Hornik and Achim Zeileis (2006). Unbiased Recursive Partitioning: A Conditional Inference Framework. Journal of Computational and Graphical Statistics, 15(3), 651--674, DOI: 10.1198/106186006X133933

data.frame
Matt Dowle and Arun Srinivasan (2021). data.table: Extension of`data.frame`. R package version 1.14.2. https://CRAN.R-project.org/package=data.table

dplyr
Hadley Wickham, Romain François, Lionel Henry and Kirill Müller (2021). dplyr: A Grammar of Data Manipulation. R package version 1.0.7. https://CRAN.R-project.org/package=dplyr

gtsummary
Daniel D. Sjoberg, Michael Curry, Margie Hannum, Joseph Larmarange, Karissa Whiting and Emily C. Zabor (2021). gtsummary: Presentation-Ready Data Summary and Analytic Result Tables. R package version 1.4.2. https://CRAN.R-project.org/package=gtsummary

partykit
Torsten Hothorn, Achim Zeileis (2015). partykit: A Modular Toolkit for Recursive Partytioning in R. Journal of Machine Learning Research, 16, 3905-3909. URL https://jmlr.org/papers/v16/hothorn15a.html

tidyverse
Wickham et al., (2019). Welcome to the tidyverse. Journal of Open Source Software, 4(43), 1686, https://doi.org/10.21105/joss.01686

Table C.1 Percentage of occupant autopsies able to be identified from TARS by year and by category of autopsy

Year	External (%)	CT (%)	Full autopsy (%)
1981	0	0	41.9
1982	0	0	39.6
1983	0	0	31.3
1984	0	0	41.3
1985	0	0	78.3
1986	0	0	87.5
1987	0	0	87.0
1988	0	0	92.4
1989	0	0	88.7
1990	0	0	92.9
1991	0	0	93.2
1992	0	0	95.7
1993	0	0	94.1
1994	0	0	89.8
1995	0	0	92.1
1996	0	0	88.8
1997	0	0	83.5
1998	0	0	74.7
1999	0	0	85.6
2000	0	0	68.8
2001	0	0	56.3
2002	0	0	64.3
2003	0	0	83.3
2004	2.1	0	72.3
2005	0	0	83.2
2006	1.4	0	90.0
2007	0	0	90.0
2008	0	1.7	96.6
2009	0	0	88.2
2010	1.4	0	85.7
2011	0	0	87.0
2012	0	0	78.9
2013	1.7	1.7	81.0
2014	0	1.6	76.6
2015	0	0	86.2
2016	0	0	73.1
2017	1.9	3.8	56.6
2018	0	29.2	47.9
2019	0	12.3	35.1
2020	0	60.7	5.4

C.2 Analysis
Logistic regression predicting the effect of sex on occupant age
Variables included: Occupant age and sex

Sample size of model: 2800

Table C.2.1 Logistic regression model output predicting sex where 0 = Male and 1 = Female

```
Call:
glm(formula = sex ~ age, family = "binomial")

Deviance Residuals:
    Min       1Q   Median       3Q      Max
-1.6906  -1.3389   0.7662   0.8660   1.2441

Coefficients:
             Estimate    Std. Error  z value    Pr(>|z|)
(Intercept)  1.442911     0.095648    15.086    < 2e-16 ***
Age         -0.015984     0.002021    -7.911    2.56e-15 ***
---
Signif. codes:  0 ‘***’ 0.001 ‘**’ 0.01 ‘*’ 0.05 ‘.’ 0.1 ‘ ’ 1

(Dispersion parameter for binomial family taken to be 1)

    Null deviance: 3456.9  on 2773  degrees of freedom
Residual deviance: 3394.3  on 2772  degrees of freedom
AIC: 3398.3
BIC: 3410.12

Number of Fisher Scoring iterations: 4
```

Table C.2.2 Summary of logistic regression predicting sex where 0 = Male and 1 = Female with odds ratios and 95% confidence intervals

Characteristic	OR[1]	95% CI[1]	p-value
age	0.98	0.98, 0.99	<0.001
[1]OR = Odds Ratio, CI = Confidence Interval			

C.3 Analysis

Linear regression predicting the effect of the year of the crash on the age of the vehicle
Variables included: age of the vehicle(derived from year of crash and year of vehicle manufacture) and year of crash
Variable excluded: vehicle year was restricted from 1950 onwards. Three vehicles were older than 40 years, include vehicles made in 1901, 1942, 1949

Sample size of model: 2543

Table C.3.1 Linear regression model output predicting age of the vehicle

```
Call:
lm(formula = age of vehicle ~ year of crash)

Residuals:
    Min      1Q  Median      3Q     Max
-14.936  -5.474  -0.445   5.000  33.204

Coefficients:
                Estimate   Std. Error   t value     Pr(>|t|)
(Intercept)    -267.94075   27.55521    -9.724     <2e-16 ***
Year of crash     0.14004    0.01379    10.156     <2e-16 ***
---
Signif. codes:  0 ‘***’ 0.001 ‘**’ 0.01 ‘*’ 0.05 ‘.’ 0.1 ‘ ’ 1

Residual standard error: 7.26 on 2548 degrees of freedom
  (247 observations deleted due to missingness)
Multiple R-squared:  0.03891,      Adjusted R-squared:  0.03853
F-statistic: 103.2 on 1 and 2548 DF,  p-value: < 2.2e-16
```

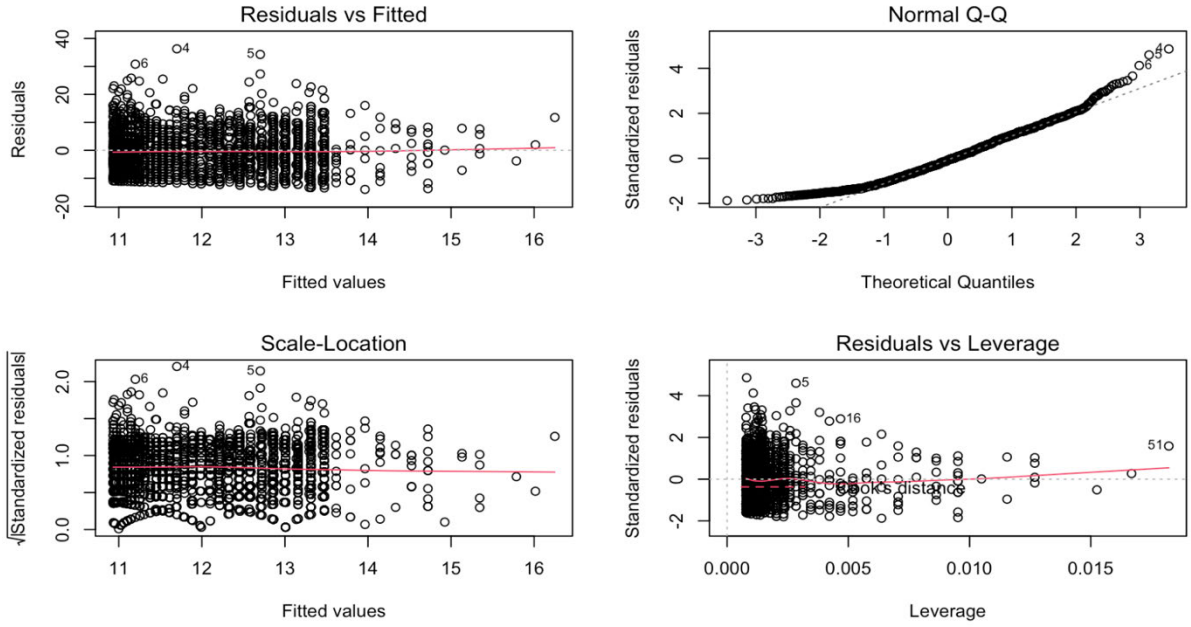

Figure C.3.1. Residuals of the linear model describing the effect of the year of crash on the age of the vehicle

C.4 Analysis

Quadratic regression predicting the effect of the driver age on the age of the vehicle
Variables included: age of the vehicle(derived from year of crash and year of vehicle manufacture) and age of the driver
Variable excluded: Only drivers were included in this analysis Vehicle year was restricted from 1950 onwards. Three vehicles were older than 40 years, include vehicles made in 1901, 1942, 1949

Sample size of model: 1783

Table C.4.1 Quadratic regression model output predicting the age of the vehicle

```
Call:
lm(formula = age of vehicle ~ I(driver age^2) + driver age)

Residuals:
    Min      1Q  Median      3Q     Max
-13.965  -5.792  -0.530   4.874  36.301

Coefficients:
                 Estimate Std.    Error     t value    Pr(>|t|)
(Intercept).      17.2741988    1.0171381   16.983     < 2e-16 ***
I(driver age^2)     0.0026342   0.0004988    5.281.     1.44e-07 ***
Driver age         -0.2585117   0.0484700   -5.333      1.09e-07 ***
---
Signif. codes:  0 ‘***’ 0.001 ‘**’ 0.01 ‘*’ 0.05 ‘.’ 0.1 ‘ ’ 1

Residual standard error: 7.474 on 1780 degrees of freedom
Multiple R-squared:  0.01576,  Adjusted R-squared:  0.01465
F-statistic: 14.25 on 2 and 1780 DF,  p-value: 7.239e-07
```

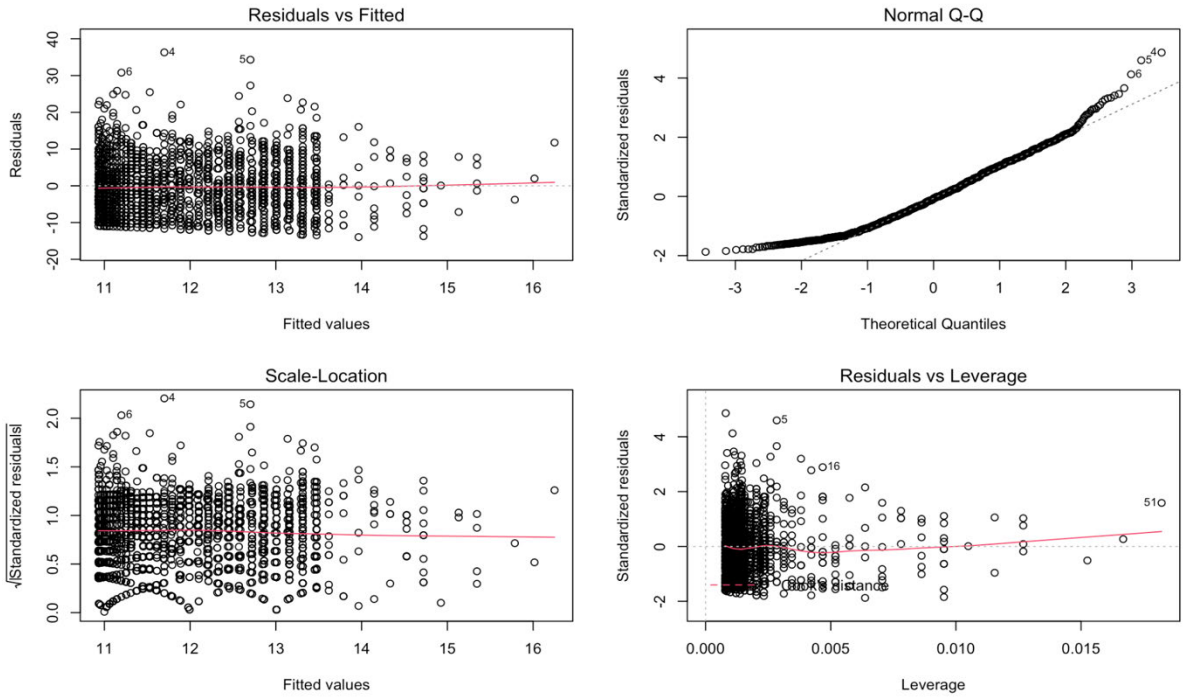

Figure C.4.1 Residuals of the quadratic model describing the effect of the driver age on the age of the vehicle

C.5 Analysis
Quadratic regression predicting the effect of the age and sex on BMI
Variables included: BMI, age and sex (males = 0, female = 1)

Sample size of model: 2204

Table C.5.1 Quadratic regression model output predicting BMI

```
Call:
lm(formula = bmi ~ age + I(age^2) + sex)

Residuals:
    Min      1Q  Median      3Q     Max
-14.677  -3.400  -0.731   2.445  37.149

Coefficients:
             Estimate    Std. Error   t value   Pr(>|t|)
(Intercept)  17.7080703   0.6564865   26.974    <2e-16 ***
Age           0.3527665   0.0301422   11.703    <2e-16 ***
I(age^2)     -0.0030045   0.0003066   -9.799    <2e-16 ***
Female        0.5105127   0.2462470    2.073     0.0383 *
---
Signif. codes:  0 ‘***’ 0.001 ‘**’ 0.01 ‘*’ 0.05 ‘.’ 0.1 ‘ ’ 1

Residual standard error: 5.297 on 2200 degrees of freedom
Multiple R-squared:  0.09076,      Adjusted R-squared:  0.08952
F-statistic:  73.2 on 3 and 2200 DF,  p-value: < 2.2e-16
```

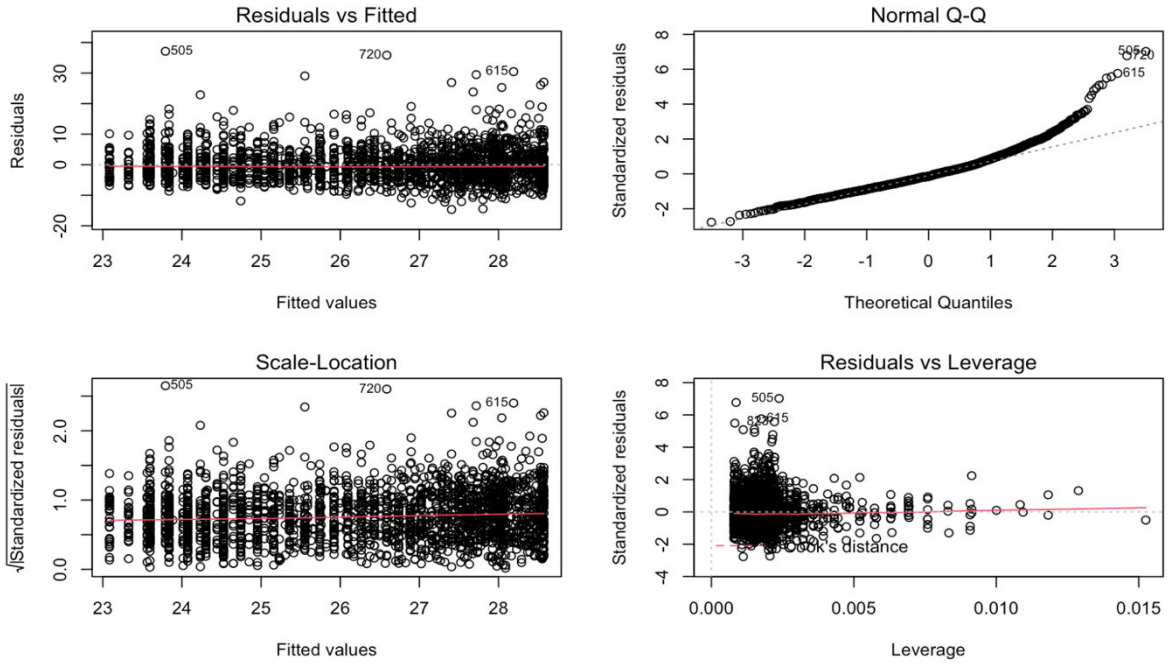

Figure C.5.1 Residuals of the quadratic model describing the effect of the sex and age on the age of the vehicle

C.6 Analysis

Decision tree predicting the location of death

Variables included in the model: Ejection, seat belt wearing, position, vehicle type, vehicle year, sex, age, BMI, alcohol, drug, closed head injury no skull fracture, closed head injury with skull fracture, nasal/zygomatic fracture, maxilla fracture, mandible fracture, panfacial fracture, open head injury, cervical spinal fracture, thoracic spinal fracture, lumbar spinal fracture, atlanto-occipital injury, upper right limb fracture, upper left limb fracture, lower right limb fracture, lower left limb fracture, liver injury, kidney injury, abdominal vasculature injury, splenic injury, mesenteric/intestinal injury, pelvic fracture, heart injury, aortic injury, other cardiovascular injury, lung contusion, lung laceration, lung avulsion, pulmonary vasculature, one or more ribs fractured, sternum fracture, diaphragm

Sample size of training data: 539
Sample size of testing data: 360

Table C.6.1 Decision tree output trained to predict location of death using the training dataset

```
Fitted party:
[1] root
|   [2] aorta in 0
|   |   [3] age <= 73: 1 (n = 346, err = 17.1%)
|   |   [4] age > 73: 3 (n = 41, err = 48.8%)
|   [5] aorta in 1: 1 (n = 152, err = 5.3%)

Number of inner nodes:    2
Number of terminal nodes: 3
```

Table C.6.2 Confusion matrix output testing the fit of the decision tree predicting location of death using the testing dataset

```
Confusion Matrix and Statistics

          Reference
Prediction   1   2   3
         1 280  16  41
         2   0   0   0
         3  15   0   8

Overall Statistics

               Accuracy : 0.8
                 95% CI : (0.7549, 0.8401)
    No Information Rate : 0.8194
    P-Value [Acc > NIR] : 0.8478

                  Kappa : 0.108
```

```
 Mcnemar's Test P-Value : NA

Statistics by Class:

                     Class: 1 Class: 2 Class: 3
Sensitivity            0.9492  0.00000  0.16327
Specificity            0.1231  1.00000  0.95177
Pos Pred Value         0.8309      NaN  0.34783
Neg Pred Value         0.3478  0.95556  0.87834
Prevalence             0.8194  0.04444  0.13611
Detection Rate         0.7778  0.00000  0.02222
Detection Prevalence   0.9361  0.00000  0.06389
Balanced Accuracy      0.5361  0.50000  0.55752
```

C.7 Analysis

Decision tree predicting the location of death in seat belt wearing, non-ejected occupants
Variables included: The same variables were included as in C.6 Analysis. Only occupants wearing a seat belt (1) and were not ejected (2) were included in the model
Variable excluded: No or unknown seat belt wearing (0,2), ejected occupants (1)
Number omitted: 352

Sample size of training data: 328
Sample size of testing data: 219

Table C.7.1 Decision tree output trained to predict location of death in seat belt wearing, non-ejected occupants using the training dataset

```
Fitted party:
[1] root
|   [2] age <= 73: 1 (n = 471, err = 14.4%)
|   [3] age > 73: 1 (n = 76, err = 35.5%)

Number of inner nodes:    1
Number of terminal nodes: 2
```

Table C.7.2 Confusion matrix output testing the fit of the decision tree predicting location of death in seat belt wearing, non-ejected occupants using the testing dataset

```
Confusion Matrix and Statistics

          Reference
Prediction   1   2   3
         1 180   9  30
         2   0   0   0
         3   0   0   0

Overall Statistics

               Accuracy : 0.8219
                 95% CI : (0.7647, 0.8702)
    No Information Rate : 0.8219
    P-Value [Acc > NIR] : 0.5427

                  Kappa : 0

 Mcnemar's Test P-Value : NA

Statistics by Class:

                     Class: 1 Class: 2 Class: 3
Sensitivity            1.0000   0.0000    0.000
Specificity            0.0000   1.0000    1.000
```

```
Pos Pred Value         0.8219      NaN      NaN
Neg Pred Value            NaN   0.9589    0.863
Prevalence             0.8219   0.0411    0.137
Detection Rate         0.8219   0.0000    0.000
Detection Prevalence   1.0000   0.0000    0.000
Balanced Accuracy      0.5000   0.5000    0.500
```

C.8 Analysis

Logistic regression predicting head injury

Variables included: Ejection, seat belt wearing, position, vehicle year, sex, age, BMI, nasal/zygomatic fracture, maxilla fracture, mandible fracture, cervical spinal fracture, thoracic spinal fracture, lumbar spinal fracture, atlanto-occipital injury, transection of spinal cord, upper right limb fracture, upper left limb fracture, lower right limb fracture, lower left limb fracture, liver injury, kidney injury, abdominal vasculature injury, splenic injury, mesenteric/intestinal injury, pelvic fracture, heart injury, aortic injury, other cardiovascular injury, lung contusion, lung laceration, pneumothorax, haemothorax, lung avulsion, pulmonary vasculature, one or more ribs fractured, sternum fracture, diaphragm

Modified variables: head injury were dichotomised into no head injury (0) and head injury which included closed head injury no skull fracture, head injury with skull fracture, open head injury (1)

Variable excluded: panfacial, brainstem, only variables with known seat belt wearing status (0=not wearing, 1=wearing) were included

Sample size of training data: 532

Sample size of testing data: 356

Table C.8.1 Logistic regression model output predicting head injury, where 0 = no head injury and 1 = head injury, using the training dataset

```
Call:
glm(formula = head injury ~ age + mandible + ul_ext + ll_ext +
pneumothorax + haemothorax + position, family = "binomial")

Deviance Residuals:
    Min       1Q   Median       3Q      Max
-2.8265  -0.8949   0.4531   0.8187   2.3037

Coefficients:
               Estimate Std.    Error     z value   Pr(>|z|)
(Intercept)           2.479026   0.303186   8.177   2.92e-16 ***
Age                  -0.027875   0.005324  -5.236   1.64e-07 ***
Mandible fracture     2.026438   0.424148   4.778   1.77e-06 ***
UL limb fracture      1.016193   0.340829   2.982   0.00287 **
LL limb fracture     -0.765539   0.260681  -2.937   0.00332 **
Pneumothorax         -1.008633   0.359245  -2.808   0.00499 **
Haemothorax          -0.943831   0.219111  -4.308   1.65e-05 ***
Front seat position^ -0.781466   0.259660  -3.010   0.00262 **
Rear seat position   -1.346745   0.465786  -2.891   0.00384 **
---
Signif. codes:  0 ‘***’ 0.001 ‘**’ 0.01 ‘*’ 0.05 ‘.’ 0.1 ‘ ’ 1

(Dispersion parameter for binomial family taken to be 1)

    Null deviance: 700.23  on 531  degrees of freedom
Residual deviance: 549.56  on 523  degrees of freedom
AIC: 567.56
```

```
BIC: 606.05

Number of Fisher Scoring iterations: 5
^ front seat position include front left passenger and front middle
passenger
```

Table C.8.2 Summary of logistic regression predicting head injury with odds ratios and 95% confidence intervals

Characteristic	OR[1]	95% CI[1]	p-value
Age	0.97	0.96, 0.98	<0.001
Mandible fracture	7.59	3.52, 19.0	<0.001
Upper left limb fracture	2.76	1.44, 5.50	0.003
Lower left limb fracture	0.47	0.28, 0.77	0.003
Pneumothorax	0.36	0.18, 0.73	0.005
Haemothorax	0.39	0.25, 0.60	<0.001
Driver	REF	REF	
Front seat passenger	0.46	0.27, 0.76	0.003
Rear seat passenger	0.26	0.10, 0.64	0.004

[1] OR = Odds Ratio, CI = Confidence Interval

The output of all binary independent variables are compared to reference category 0 i.e., no injury present

Table C.8.3 Confusion matrix output testing the fit of the logistic regression model predicting head injury using the training dataset

```
Confusion Matrix and Statistics


       FALSE TRUE
  FALSE    60   35
  TRUE     83  178

               Accuracy : 0.6685
                 95% CI : (0.617, 0.7173)
    No Information Rate : 0.5983
    P-Value [Acc > NIR] : 0.003741

                  Kappa : 0.2702

 Mcnemar's Test P-Value : 1.514e-05

            Sensitivity : 0.4196
            Specificity : 0.8357
         Pos Pred Value : 0.6316
         Neg Pred Value : 0.6820
             Prevalence : 0.4017
         Detection Rate : 0.1685
   Detection Prevalence : 0.2669
      Balanced Accuracy : 0.6276

       'Positive' Class : FALSE
```

C.9 Analysis

Decision tree predicting head injury

Variables included: Ejection, seat belt wearing, position, vehicle year, sex, age, BMI, nasal/zygomatic fracture, maxilla fracture, mandible fracture, cervical spinal fracture, thoracic spinal fracture, lumbar spinal fracture, atlanto-occipital injury, transection of spinal cord, upper right limb fracture, upper left limb fracture, lower right limb fracture, lower left limb fracture, liver injury, kidney injury, abdominal vasculature injury, splenic injury, mesenteric/intestinal injury, pelvic fracture, heart injury, aortic injury, other cardiovascular injury, lung contusion, lung laceration, pneumothorax, haemothorax, lung avulsion, pulmonary vasculature, one or more ribs fractured, sternum fracture, diaphragm

Variable excluded: only variables with known seat belt wearing status (0=not wearing, 1=wearing) were included

Sample size of training data: 698

Sample size of testing data: 466

Table C.9.1 Decision tree output trained to predict head injury using the training dataset

```
Model formula:
head ~ ejected + seatbelt + vehicleyear + sex + age + bmi +
nasal_zygoma +  maxilla + mandible + cf spine + tf spine + lf_spine
+ atlanto +  trans_spinal_cord + ul_ext + ur_ext + ll_ext + lr_ext +
liver + kidney + abd_vas + spleen + mesentery + pelvis + heart +
cardio_vas + aorta + contusion_lung + laceration_lung +
avulsion_lung + pneumothorax + haemothorax + lung_vas + chestwall +
sternum +  diaphragm + position

Fitted party:
[1] root
|   [2] maxilla in 0
|   |   [3] age <= 54
|   |   |   [4] atlanto in 0
|   |   |   |   [5] haemothorax in 0
|   |   |   |   |   [6] heart in 0
|   |   |   |   |   |   [7] cf_spine in 0
|   |   |   |   |   |   |   [8] aorta in 0: 2 (n = 110, err = 26.4%)
|   |   |   |   |   |   |   [9] aorta in 1: 0 (n = 10, err = 60.0%)
|   |   |   |   |   |   [10] cf_spine in 1: 1 (n = 15, err = 53.3%)
|   |   |   |   |   [11] heart in 1: 0 (n = 13, err = 30.8%)
|   |   |   |   [12] haemothorax in 1
|   |   |   |   |   [13] ul_ext in 0: 2 (n = 160, err = 62.5%)
|   |   |   |   |   [14] ul_ext in 1: 0 (n = 26, err = 65.4%)
|   |   |   [15] atlanto in 1: 1 (n = 82, err = 54.9%)
|   |   [16] age > 54: 0 (n = 181, err = 49.7%)
|   [17] maxilla in 1: 2 (n = 101, err = 28.7%)

Number of inner nodes:    8
```

```
Number of terminal nodes: 9
  7)*  weights = 132
```

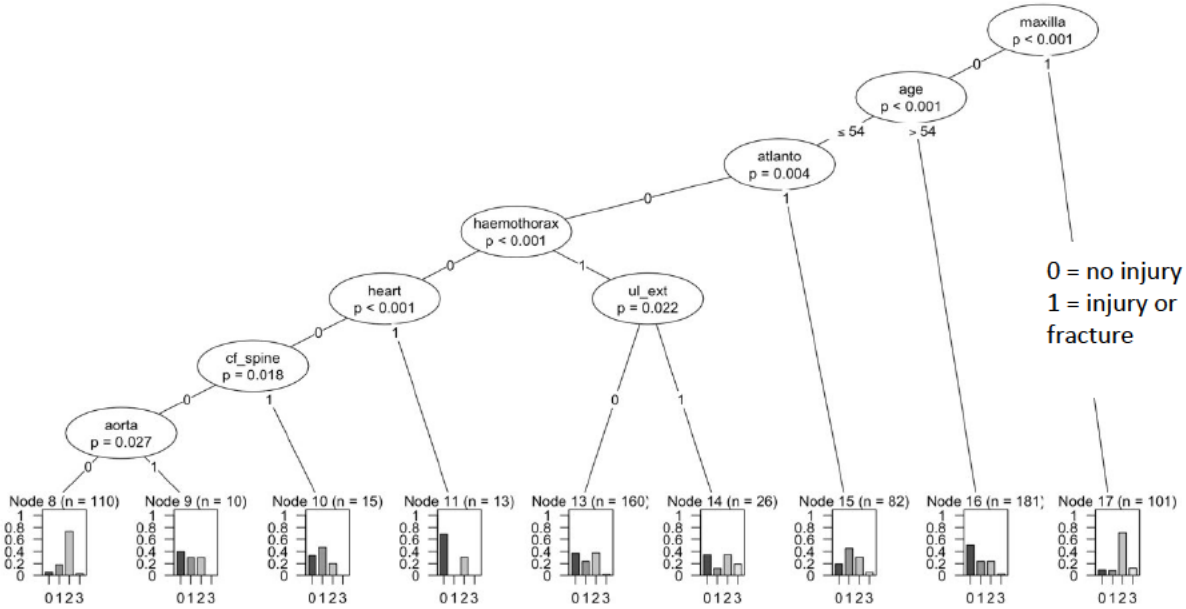

Figure C.9.1 Output of decision tree where the labels on the x axis on the terminal nodes correlate to 0 = no head injury, 1 = closed head injury without skull fracture, 2 = closed head injury without skull fracture and 3 = open head injury

Table C.9.2 Confusion matrix output testing the fit of the decision tree predicting head injury, where 0 = no head injury, 1 = closed head injury without skull fracture, 2 = closed head injury without skull fracture and 3 = open head injury, using the testing dataset

```
Confusion Matrix and Statistics

          Reference
Prediction   0   1   2   3
         0  57  50  36   4
         1  14  28  19   2
         2  60  39 134  23
         3   0   0   0   0

Overall Statistics

               Accuracy : 0.47
                 95% CI : (0.4239, 0.5164)
    No Information Rate : 0.4056
    P-Value [Acc > NIR] : 0.00283

                  Kappa : 0.1902

 Mcnemar's Test P-Value : 1.647e-11
```

```
Statistics by Class:

                     Class: 0 Class: 1 Class: 2 Class: 3
Sensitivity            0.4351  0.23932   0.7090  0.00000
Specificity            0.7313  0.89971   0.5596  1.00000
Pos Pred Value         0.3878  0.44444   0.5234      NaN
Neg Pred Value         0.7680  0.77916   0.7381  0.93777
Prevalence             0.2811  0.25107   0.4056  0.06223
Detection Rate         0.1223  0.06009   0.2876  0.00000
Detection Prevalence   0.3155  0.13519   0.5494  0.00000
Balanced Accuracy      0.5832  0.56951   0.6343  0.50000
```

C.10 Analysis

Logistic regression predicting hinge fracture

Variables included: Ejection, seat belt wearing, position, vehicle year, sex, age, BMI, brainstem, hinge fracture, nasal/zygomatic fracture, maxilla fracture, mandible fracture, panfacial fracture, transection of spinal cord, cervical spinal fracture, thoracic spinal fracture, lumbar spinal fracture, atlanto-occipital injury, upper right limb fracture, upper left limb fracture, lower right limb fracture, lower left limb fracture, liver injury, kidney injury, abdominal vasculature injury, splenic injury, mesenteric/intestinal injury, pelvic fracture, heart injury, aortic injury, other cardiovascular injury, lung contusion, lung laceration, pneumothorax, haemothorax, lung avulsion, pulmonary vasculature, one or more ribs fractured, sternum fracture, diaphragm

Variable excluded: Only cases where a skull fracture was identified were included in the dataset, only variables with known seat belt wearing status (0=not wearing, 1=wearing) were included

Sample size of model: 484

Table C.10.1 Logistic regression model output predicting hinge fracture from occupants with a skull fracture

```
Call:
glm(formula = hinge ~ age + nasal/zygoma, family = "binomial")

Deviance Residuals:
    Min       1Q   Median       3Q      Max
-1.0579  -0.9428  -0.7264   1.3221   1.9622

Coefficients:
                     Estimate Std. Error    z value   Pr(>|z|)
(Intercept)           -0.010450   0.249172  -0.042   0.96655
Age                   -0.015405   0.006235  -2.471   0.01349 *
Nasal/zygoma fracture -0.894423   0.285103  -3.137   0.00171 **
---
Signif. codes:  0 ‘***’ 0.001 ‘**’ 0.01 ‘*’ 0.05 ‘.’ 0.1 ‘ ’ 1

(Dispersion parameter for binomial family taken to be 1)

    Null deviance: 599.17  on 473  degrees of freedom
Residual deviance: 582.70  on 471  degrees of freedom
AIC: 588.7
BIC: 601.18


Number of Fisher Scoring iterations: 4
```

Table C.10.2 Summary of logistic regression predicting hinge fracture with odds ratios and 95% confidence intervals

Characteristic	OR[1]	95% CI[1]	p-value
Age	0.98	0.97, 1.00	0.013
Nasal/zygoma fracture	0.41	0.23, 0.70	0.002
[1] OR = Odds Ratio, CI = Confidence Interval			

The output of all binary independent variables are compared to reference category 0 i.e., no injury present

C. 11 Analysis

Logistic regression predicting ring fracture

Variables included: Ejection, seat belt wearing, position, vehicle year, sex, age, BMI, brainstem, ring fracture, nasal/zygomatic fracture, maxilla fracture, mandible fracture, panfacial fracture, cervical spinal fracture, thoracic spinal fracture, lumbar spinal fracture, atlanto-occipital injury, transection of spinal cord, upper right limb fracture, upper left limb fracture, lower right limb fracture, lower left limb fracture, liver injury, kidney injury, abdominal vasculature injury, splenic injury, mesenteric/intestinal injury, pelvic fracture, heart injury, aortic injury, other cardiovascular injury, lung contusion, lung laceration, pneumothorax, haemothorax, lung avulsion, pulmonary vasculature, one or more ribs fractured, sternum fracture, diaphragm

Variable excluded: Only cases where a skull fracture was identified were included in the dataset, only variables with known seat belt wearing status (0=not wearing, 1=wearing) were included

Sample size of model: 482

Table C.11.1 Logistic regression model output predicting ring fracture from occupants with a skull fracture

```
Call:
glm(formula = ring ~ brainstem + ll_ext + pelvis, family =
"binomial")

Deviance Residuals:
    Min       1Q   Median       3Q      Max
-1.3023  -0.5880  -0.4037  -0.4037   2.2572

Coefficients:
            Estimate Std.  Error    z value Pr(>|z|)
(Intercept)      -2.4661     0.2097 -11.762  < 2e-16 ***
Brainstem         1.2056     0.2996   4.024  5.73e-05 ***
LL limb fracture  0.7508     0.2850   2.634  0.00844 **
Pelvic fracture   0.7986     0.2770   2.883  0.00393 **
---
Signif. codes:  0 ‘***’ 0.001 ‘**’ 0.01 ‘*’ 0.05 ‘.’ 0.1 ‘ ’ 1

(Dispersion parameter for binomial family taken to be 1)

    Null deviance: 403.18  on 471  degrees of freedom
Residual deviance: 368.89  on 468  degrees of freedom
AIC: 376.89
BIC: 393.52


Number of Fisher Scoring iterations: 5
```

Table C.11.2 Summary of logistic regression predicting ring fracture with odds ratios and 95% confidence intervals

Characteristic	OR[1]	95% CI[1]	p-value
Brainstem injury	3.34	1.84, 5.98	<0.001
Lower left limb fracture	2.12	1.20, 3.69	0.008
Pelvic fracture	2.22	1.29, 3.82	0.004

[1] OR = Odds Ratio, CI = Confidence Interval

The output of all binary independent variables are compared to reference category 0 i.e., no injury present

C.12 Analysis

Logistic regression predicting brainstem injury

Variables included: Ejection, seat belt wearing, position, vehicle year, sex, age, BMI, brainstem, nasal/zygomatic fracture, maxilla fracture, mandible fracture, panfacial fracture, hinge fracture, ring fracture, cervical spinal fracture, thoracic spinal fracture, lumbar spinal fracture, atlanto-occipital injury, transection of spinal cord, upper right limb fracture, upper left limb fracture, lower right limb fracture, lower left limb fracture, liver injury, kidney injury, abdominal vasculature injury, splenic injury, mesenteric/intestinal injury, pelvic fracture, heart injury, aortic injury, other cardiovascular injury, lung contusion, lung laceration, pneumothorax, haemothorax, lung avulsion, pulmonary vasculature, one or more ribs fractured, sternum fracture, diaphragm

Modified variables: Interaction with atlanto-occipital injury was included in the model

Variable excluded: Only variables with known seat belt wearing status (0=not wearing, 1=wearing) were included

Sample size of model: 1131

Table C.12.1 Logistic regression model output predicting brainstem injury

```
Call:
glm(formula = brainstem ~ atlanto + hinge + ring + transection of
spinal cord,
    family = "binomial")

Deviance Residuals:
    Min       1Q   Median       3Q      Max
-1.5634  -0.5303  -0.3167  -0.3167   2.4567

Coefficients:
                  Estimate Std. Error z value Pr(>|z|)
(Intercept)        -2.9674     0.1578 -18.805  < 2e-16 ***
atlanto             1.1301     0.2259   5.004 5.62e-07 ***
hinge               1.0769     0.2461   4.377 1.20e-05 ***
ring                1.7416     0.2872   6.064 1.33e-09 ***
trans spinal cord   1.0220     0.2535   4.032 5.53e-05 ***
---
Signif. codes:  0 ‘***’ 0.001 ‘**’ 0.01 ‘*’ 0.05 ‘.’ 0.1 ‘ ’ 1

(Dispersion parameter for binomial family taken to be 1)

    Null deviance: 769.45  on 1130  degrees of freedom
Residual deviance: 679.95  on 1126  degrees of freedom
AIC: 689.95
BIC: 715.10

Number of Fisher Scoring iterations: 5
```

Table C.12.2 Summary of logistic regression predicting brainstem injury with odds ratios and 95% confidence intervals

Characteristic	OR[1]	95% CI[1]	p-value
Atlanto-occipital fracture	3.10	1.98, 4.81	<0.001
Hinge fracture	2.94	1.80, 4.73	<0.001
Ring fracture	5.71	3.22, 9.96	<0.001
Transection spinal cord	2.78	1.67, 4.53	<0.001

[1] OR = Odds Ratio, CI = Confidence Interval

The output of all binary independent variables are compared to reference category 0 i.e., no injury present

C.13 Analysis

Logistic regression predicting atlanto-occipital injury

Variables included: Ejection, seat belt wearing, position, vehicle year, crash type, sex, age, BMI, brainstem, closed head injury no skull fracture, closed head injury with skull fracture, hinge fracture, ring fracture, nasal/zygomatic fracture, maxilla fracture, mandible fracture, panfacial fracture, open head injury, cervical spinal fracture, thoracic spinal fracture, lumbar spinal fracture, atlanto-occipital injury, transection of spinal cord, upper right limb fracture, upper left limb fracture, lower right limb fracture, lower left limb fracture, liver injury, kidney injury, abdominal vasculature injury, splenic injury, mesenteric/intestinal injury, pelvic fracture, heart injury, aortic injury, other cardiovascular injury, lung contusion, lung laceration, pneumothorax, haemothorax, lung avulsion, pulmonary vasculature, one or more ribs fractured, sternum fracture, diaphragm

Variable excluded: Only variables with known seat belt wearing status (0=not wearing, 1=wearing) were included

Sample size of model: 1143

Table C.13.1 Logistic regression model output predicting atlanto-occipital injury

```
Call:
glm(formula = atlanto ~ bf_nf + brainstem + trans_spinal_cord,
    family = "binomial")

Deviance Residuals:
    Min       1Q   Median       3Q      Max
-1.6712  -0.7031  -0.4608  -0.4608   2.1425

Coefficients:
                  Estimate Std. Error z value Pr(>|z|)
(Intercept)        -2.1890     0.1196 -18.301  < 2e-16 ***
Closed head nf              1.0913     0.1767   6.174 6.64e-10 ***
brainstem1          1.2923     0.2249   5.745 9.19e-09 ***
trans spinal cord   0.9175     0.2125   4.317 1.58e-05 ***
---
Signif. codes:  0 ‘***’ 0.001 ‘**’ 0.01 ‘*’ 0.05 ‘.’ 0.1 ‘ ’ 1

(Dispersion parameter for binomial family taken to be 1)

    Null deviance: 1054.58  on 1128  degrees of freedom
Residual deviance:  966.24  on 1125  degrees of freedom
AIC: 974.24
BIC: 994.35

Number of Fisher Scoring iterations: 4
```

Table C.13.2 Summary of logistic regression predicting atlanto-occipital injury with odds ratios and 95% confidence intervals

Characteristic	OR[1]	95% CI[1]	p-value
Closed head injury, no skull fracture	2.98	2.10, 4.21	<0.001
Brainstem injury	3.64	2.33, 5.64	<0.001
Transection of spinal cord	2.50	1.64, 3.78	<0.001
[1] OR = Odds Ratio, CI = Confidence Interval			

The output of all binary independent variables are compared to reference category 0 i.e., no injury present

C.14 Analysis

Logistic regression predicting cervical spinal fracture
Variables included: Ejection, seat belt wearing, position, vehicle year, sex, age, height, weight, cervical spinal fracture, crash type, vehicle type
Variable excluded: Only variables with known seat belt wearing status (0=not wearing, 1=wearing) were included

Sample size of model:1176

Table C.14.1 Logistic regression model output predicting cervical spinal fracture

```
Call:
glm(formula = cf_spine ~ age, family = "binomial")

Deviance Residuals:
    Min       1Q   Median       3Q      Max
-0.8444  -0.5983  -0.5098  -0.4637   2.1516

Coefficients:
             Estimate Std. Error z value Pr(>|z|)
(Intercept) -2.525180   0.200816 -12.575  < 2e-16 ***
Age          0.017473   0.003839   4.551 5.33e-06 ***
---
Signif. codes:  0 ‘***’ 0.001 ‘**’ 0.01 ‘*’ 0.05 ‘.’ 0.1 ‘ ’ 1

(Dispersion parameter for binomial family taken to be 1)

    Null deviance: 992.82  on 1175  degrees of freedom
Residual deviance: 972.39  on 1174  degrees of freedom
AIC: 976.39
BIC: 986.53

Number of Fisher Scoring iterations: 4
```

Table C.14.2 Summary of logistic regression predicting cervical spinal fracture with odds ratios and 95% confidence intervals

Characteristic	**OR**[1]	**95% CI**[1]	**p-value**
Age	1.02	1.01, 1.03	<0.001
[1]OR = Odds Ratio, CI = Confidence Interval			

C.15 Analysis

Logistic regression predicting thoracic spinal fracture
Variables included: Ejection, seat belt wearing, position, vehicle year, sex, age, height, weight, thoracic spinal fracture, crash type, vehicle type
Variable excluded: Only variables with known seat belt wearing status (0=not wearing, 1=wearing) were included

Sample size of model: 1177

Table C.15.1 Logistic regression model output predicting thoracic spinal fracture

```
Call:
glm(formula = tf_spine ~ age + weight, family = "binomial")

Deviance Residuals:
    Min       1Q   Median       3Q      Max
-1.1596  -0.6910  -0.5298  -0.4190   2.2406

Coefficients:
             Estimate Std. Error z value Pr(>|z|)
(Intercept) -3.802610   0.367327 -10.352  < 2e-16 ***
Age          0.026710   0.003577   7.467 8.17e-14 ***
Weight^       0.014460   0.003653   3.958 7.55e-05 ***
---
Signif. codes:  0 ‘***’ 0.001 ‘**’ 0.01 ‘*’ 0.05 ‘.’ 0.1 ‘ ’ 1

(Dispersion parameter for binomial family taken to be 1)

    Null deviance: 1174.1  on 1176  degrees of freedom
Residual deviance: 1101.0  on 1174  degrees of freedom
AIC: 1107
BIC: 1122.22

Number of Fisher Scoring iterations: 4
^ weight in kilograms
```

Table C.15.2 Summary of logistic regression predicting thoracic spinal fracture with odds ratios and 95% confidence intervals

Characteristic	OR[1]	95% CI[1]	p-value
Age	1.03	1.02, 1.03	<0.001
Weight	1.01	1.01, 1.02	<0.001
[1]OR = Odds Ratio, CI = Confidence Interval			

C.16 Analysis

Logistic regression predicting lumbar spinal fracture
Variables included: Ejection, seat belt wearing, position, vehicle year, sex, age, height, weight cervical spinal fracture, crash type, vehicle type
Variable excluded: Only variables with known seat belt wearing status (0=not wearing, 1=wearing) were included

Sample size of model:1177

Table C.16.1 Logistic regression model output predicting lumbar spinal fracture

```
Call:
glm(formula = lf_spine ~ casejected + vehicleyear, family =
"binomial")

Deviance Residuals:
    Min       1Q   Median       3Q      Max
-0.6622  -0.2770  -0.2207  -0.1706   3.0263

Coefficients:
              Estimate   Std. Error  z value   Pr(>|z|)
(Intercept)  -117.61833   35.72248   -3.293   0.000993 ***
Not ejected    -1.14346    0.37284   -3.067   0.002163 **
Vehicleyear     0.05770    0.01788    3.227   0.001249 **
---
Signif. codes:  0 ‘***’ 0.001 ‘**’ 0.01 ‘*’ 0.05 ‘.’ 0.1 ‘ ’ 1

(Dispersion parameter for binomial family taken to be 1)

    Null deviance: 335.68  on 1176  degrees of freedom
Residual deviance: 315.83  on 1174  degrees of freedom
AIC: 321.83
BIC: 337.05

Number of Fisher Scoring iterations: 7
```

Table C.16.2 Summary of logistic regression predicting lumbar spinal fracture with odds ratios and 95% confidence intervals

Characteristic	OR[1]	95% CI[1]	p-value
Not ejected	0.32	0.16, 0.69	0.002
Year of vehicle manufacture	1.06	1.02, 1.10	0.001
[1]OR = Odds Ratio, CI = Confidence Interval			

The output of all binary independent variables are compared to reference category 0 i.e., no injury present

C.17 Analysis
Logistic regression predicting liver injury
Variables included: Seat belt wearing, sex, age, BMI, liver injury
Variable excluded: Only variables with known seat belt wearing status (0=not wearing, 1=wearing) were included

Sample size of model:1228

Table C.17.1 Logistic regression model output predicting liver injury

```
Call:
glm(formula = liver ~ 1, family = "binomial")

Deviance Residuals:
    Min       1Q   Median       3Q      Max
-0.7996  -0.7996  -0.7996   1.6100   1.6100

Coefficients:
            Estimate Std. Error z value Pr(>|z|)
(Intercept) -0.97635    0.06401  -15.25   <2e-16 ***
---
Signif. codes:  0 ‘***’ 0.001 ‘**’ 0.01 ‘*’ 0.05 ‘.’ 0.1 ‘ ’ 1

(Dispersion parameter for binomial family taken to be 1)

    Null deviance: 1441.2  on 1227  degrees of freedom
Residual deviance: 1441.2  on 1227  degrees of freedom
AIC: 1443.2
BIC: 1448.35

Number of Fisher Scoring iterations: 4
```

C.18 Analysis
Stepwise logistic regression predicting kidney injury
Variables included: Seat belt wearing, sex, age, bmi, kidney
Variable excluded: Only variables with known seat belt wearing status (0=not wearing, 1=wearing) were included

Sample size of model: 1227

Table C.18.1 Logistic regression model output predicting kidney injury

```
Call:
glm(formula = kidney ~ age, family = "binomial")

Deviance Residuals:
    Min       1Q   Median       3Q      Max
-0.4394  -0.4148  -0.3631  -0.2981   2.6113

Coefficients:
             Estimate Std. Error z value Pr(>|z|)
(Intercept) -1.978647   0.263950  -7.496 6.56e-14 ***
Age         -0.017249   0.006418  -2.688   0.0072 **
---
Signif. codes:  0 ‘***’ 0.001 ‘**’ 0.01 ‘*’ 0.05 ‘.’ 0.1 ‘ ’ 1

(Dispersion parameter for binomial family taken to be 1)

    Null deviance: 591.51  on 1226  degrees of freedom
Residual deviance: 583.58  on 1225  degrees of freedom
AIC: 587.58
BIC: 597.80

Number of Fisher Scoring iterations: 5
```

Table C.18.2 Summary of logistic regression predicting kidney injury with odds ratios and 95% confidence intervals

Characteristic	**OR**[1]	**95% CI**[1]	**p-value**
Age	0.98	0.97, 0.99	0.007
[1] OR = Odds Ratio, CI = Confidence Interval			

C.19 Analysis
Stepwise logistic regression predicting splenic injury
Variables included: Seat belt wearing, sex, age, bmi, spleen
Variable excluded: Only variables with known seat belt wearing status (0=not wearing, 1=wearing) were included

Sample size of model:1228

Table C.19.1 Logistic regression model output predicting splenic injury

```
Call:
glm(formula = spleen ~ 1, family = "binomial")

Deviance Residuals:
    Min       1Q   Median       3Q      Max
-0.6188  -0.6188  -0.6188  -0.6188   1.8693

Coefficients:
            Estimate Std. Error z value Pr(>|z|)
(Intercept) -1.55568    0.07523  -20.68   <2e-16 ***
---
Signif. codes:  0 ‘***’ 0.001 ‘**’ 0.01 ‘*’ 0.05 ‘.’ 0.1 ‘ ’ 1

(Dispersion parameter for binomial family taken to be 1)

    Null deviance: 1136.1  on 1227  degrees of freedom
Residual deviance: 1136.1  on 1227  degrees of freedom
AIC: 1138.1
BIC: 1143.23

Number of Fisher Scoring iterations: 4
```

C.20 Analysis
Stepwise logistic regression predicting intestinal/mesenteric injury
Variables included: Seat belt wearing, sex, age, bmi, intestinal/mesenteric
Variable excluded: Only variables with known seat belt wearing status (0=not wearing, 1=wearing) were included

Sample size of model:1224

Table C.20.1 Logistic regression model output predicting intestinal/mesenteric injury

```
Call:
glm(formula = mesentery ~ bmi, family = "binomial")

Deviance Residuals:
    Min       1Q   Median       3Q      Max
-1.0283  -0.5038  -0.4580  -0.4167   2.3133

Coefficients:
            Estimate Std. Error z value Pr(>|z|)
(Intercept) -3.51016    0.41056  -8.550  < 2e-16 ***
BMI          0.05167    0.01410   3.664 0.000248 ***
---
Signif. codes:  0 ‘***’ 0.001 ‘**’ 0.01 ‘*’ 0.05 ‘.’ 0.1 ‘ ’ 1

(Dispersion parameter for binomial family taken to be 1)

    Null deviance: 853.94  on 1223  degrees of freedom
Residual deviance: 841.32  on 1222  degrees of freedom
AIC: 845.32
BIC: 855.54

Number of Fisher Scoring iterations: 5
```

Table C.20.2 Summary of logistic regression predicting mesenteric/intestinal injury with odds ratios and 95% confidence intervals

Characteristic	OR[1]	95% CI[1]	p-value
BMI	1.05	1.02, 1.08	<0.001
[1] OR = Odds Ratio, CI = Confidence Interval			

C.21 Analysis

Stepwise logistic regression predicting pelvic fracture

Variables included: Ejection, seat belt wearing, position, vehicle year, sex, age, BMI, brainstem, ring fracture, nasal/zygomatic fracture, maxilla fracture, mandible fracture, panfacial fracture, cervical spinal fracture, thoracic spinal fracture, lumbar spinal fracture, atlanto-occipital injury, upper right limb fracture, upper left limb fracture, lower right limb fracture, lower left limb fracture, liver injury, kidney injury, abdominal vasculature injury, splenic injury, mesenteric/intestinal injury, pelvic fracture, heart injury, aortic injury, other cardiovascular injury, lung contusion, lung laceration, pneumothorax, haemothorax, lung avulsion, pulmonary vasculature, one or more ribs fractured, sternum fracture, diaphragm

Modified variables: Interaction between age and sex was included in the model

Variable excluded: Only variables with known seat belt wearing status (0=not wearing, 1=wearing) were included

Sample size of model: 1146

Table C.21.1 Logistic regression model output predicting pelvic fracture

```
Call:
glm(formula = pelvis ~ seatbelt + vehicleyear + bf_nf + ring +
spleen + mesentery + heart + aorta + chestwall + diaphragm + femur2,
    family = "binomial")

Deviance Residuals:
    Min       1Q   Median       3Q      Max
-2.1877  -0.7467  -0.5152   0.7364   2.6068

Coefficients:
              Estimate.    Std. Error  z value   Pr(>|z|)
(Intercept).   -45.210956  14.821822  -3.050   0.002286 **
Seatbelt Worn    0.499740   0.171296   2.917   0.003530 **
Vehicleyear      0.021221   0.007444   2.851   0.004360 **
Closed head nf   0.594713   0.169268   3.513   0.000442 ***
Ring1            0.802060   0.289232   2.773   0.005553 **
Spleen1          0.684943   0.183969   3.723   0.000197 ***
Mesentery        0.655748   0.224534   2.920   0.003495 **
Heart            0.522597   0.175753   2.973   0.002945 **
Aorta            0.587180   0.167478   3.506   0.000455 ***
One or more rib  0.654906   0.221145   2.961   0.003062 **
Diaphragm        0.762587   0.206566   3.692   0.000223 ***
Bilateral femur  0.789306   0.162685   4.852   1.22e-06 ***
---
Signif. codes:  0 ‘***’ 0.001 ‘**’ 0.01 ‘*’ 0.05 ‘.’ 0.1 ‘ ’ 1

(Dispersion parameter for binomial family taken to be 1)

    Null deviance: 1372.3  on 1145  degrees of freedom
Residual deviance: 1131.3  on 1134  degrees of freedom
```

```
AIC: 1155.3
BIC: 1215.83

Number of Fisher Scoring iterations: 4
```

Table C.21.2 Summary of logistic regression predicting pelvic fracture with odds ratios and 95% confidence intervals

Characteristic	OR	95% CI	p-value
Seat belt wearing	1.65	1.18, 2.32	0.004
Year of vehicle manufacture	1.02	1.01, 1.04	0.004
Closed head injury, no skull fracture	1.81	1.30, 2.52	<0.001
Ring fracture	2.23	1.26, 3.92	0.006
Splenic injury	1.98	1.38, 2.84	<0.001
Mesenteric injury	1.93	1.24, 2.99	0.003
Heart injury	1.69	1.19, 2.38	0.003
Aortic injury	1.80	1.29, 2.50	<0.001
Chest wall injury	1.92	1.26, 3.01	0.003
Diaphragm injury	2.14	1.43, 3.21	<0.001
Bilateral femur fracture	2.20	1.60, 3.03	<0.001
OR = Odds Ratio, CI = Confidence Interval			

The output of all binary independent variables are compared to reference category 0 i.e., no injury present