SUBDURAL HEMATOMA AND AGING: CRASH CHARACTERISTICS AND ASSOCIATED INJURIES

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ABSTRACT

Among motor vehicle crash head injuries, subdural hematomas (SDH) are both frequent and life-threatening, especially for older occupants. Previous research on the mechanism of injury and on the increased vulnerability of older individuals to SDH has focused on the failure of bridging veins, which are one possible source of subdural bleeding. For all age groups, the injury mechanism and injury tolerance for SDH as a result of other bleeding sources has not been addressed. In the current study, two US crash databases were used to compare crash and injury characteristics for SDH cases in different age groups, with a focus on the original source of bleeding. Review of cases from the Crash Injury Research and Engineering Network (CIREN) database showed that both bridging veins *and* bleeding sources other than bridging veins are responsible for SDH among crash occupants in all age groups. Analysis of weighted data from the National Automotive Sampling System Crashworthiness Data System (NASS CDS) showed that the frequency of isolated SDH increases with age, potentially reflecting an increase in the frequency of SDH caused by bridging vein bleeding, particularly in frontal crashes and among women. SDH accompanied by brain contusions or other potential bleeding sources on the surface of the brain are also common, especially in side impacts and among occupants younger than 70. These cases potentially represent injuries where subdural bleeding came from sources other than bridging veins. Improved definition of SDH injury tolerance for all adult occupants will require a better understanding of the mechanism of injury from sources other than bridging veins, but determination of SDH injury tolerance for older occupants should focus on evaluation of the increasing risk of bridging vein failure with age.

INTRODUCTION

Subdural hematoma (SDH) is a frequent and life-threatening injury, especially for older adults. SDH has been reported in 26-36% of serious head injury cases [Gennarelli and Graham, 2005, Perel et al., 2009] and is reported to be among the most frequent injuries seen by neurosurgeons [Taussky et al., 2008]. Mortality rates are reported from 33 to 79% [Sawauchi and Abe, 2008, Servadei, 1997, Taussky et al., 2008]. For older occupants, SDH presents doubly-increased risks since it is not only more frequent among older individuals [Mallory, 2010, Seelig et al., 1981] but also presents an increasing rate of mortality and poor outcome with increasing age [Hanif et al., 2009, Hukkelhoven et al., 2003, Raju et al., 2004, Stitzel et al., 2008].

SDH are collections of blood that form in the dural border cell layer between the dura and arachnoid [Haines et al., 1993] or between the dura and the pial surface of the brain [Miller and Statham, 2000]. The original source for the bleeding that produces SDH can be from adjacent brain contusions, lacerations or intracerebral hematomas; from ruptured cortical vessels on the surface of the brain; or from the bridging veins that cross the meninges en route to the dural sinuses [Depreitere et al., 2006, Graham, 1996, Lee et al., 1987, Miller et al., 1996, Miller and Statham, 2000, Toyama et al., 2005]. Although ruptured bridging veins are generally believed to be a major source of SDH [Gennarelli and Thibault, 1982, Gennarelli and Graham, 2005, Lee and Haut, 1989], post-mortem studies of fatal traumatic subdural cases showed that as many as two thirds of SDH were the result of brain contusions [Maxeiner, 1997]. In the remaining third of cases, bleeding was attributed to bridging vein rupture and torn cortical arteries with

equal frequency [Maxeiner and Wolff, 2002]. Previously, in a study of surgically-treated SDH cases, bleeding from cerebral lacerations or intracranial hematoma was identified as the cause of SDH in 40.5% of cases [Jamieson and Yelland, 1972].

Subdural Hematoma Originating from Bridging Veins Experimental studies exploring injury mechanism and tolerance for SDH at any age have primarily focused on those that originate with the rupture of parasagittal bridging veins. Holbourn proposed that rotational motion of the brain relative to the skull stretched the bridging veins that cross the meningeal layers [Holbourn, 1943]. Ommaya et al. produced parasagittal SDH in rhesus monkeys by simulating a rear impact and attributed it to veins draining into the sagittal sinus [Ommaya et al., 1968]. Löwenhielm produced bridging vein rupture in frontal sled tests with post-mortem human subjects to estimate tolerance relative to anterior-posterior rotation [Löwenhielm, 1974]. Gennerelli and Thibault produced SDH in rhesus monkeys by applying anterior-posterior acceleration in a 60 degree arc, with demonstrable damage to bridging veins underlying the hematoma [Gennarelli and Thibault, 1982]. Depreitere documented parasagittal bridging vein failure in post-mortem human subjects by pressurizing the superior sagittal sinus with radiopaque fluid. Radiographs following anterior-posterior impacts were used to identify bridging vein rupture and predict tolerance to sagittal plane rotation/acceleration [Depreitere et al., 2005, Depreitere et al., 2006].

Age-related brain atrophy is thought to contribute to bridging vein failure in older individuals because it can lead to increased relative motion between the skull and brain, resulting in increased tension in the bridging veins [Hanif et al., 2009, Meaney, 1991, Yamashima and Friede, 1984]. Modeling studies have predicted a tripling of bridging vein strain in frontal impacts with modeled increase in the thickness of the subdural space to simulate age-related atrophy [Kleiven and von Holst, 2002]. Other explanations explored for decreased tolerance to SDH in older individuals associated with bridging vein rupture include age-related alterations in the mechanical properties of bridging veins [Meaney, 1991, Monson, 2001] and in the viscoelastic properties of the brain [Jane and Francel, 1996].

Subdural Hematoma Originating from Other Sources In contrast to the body of work on SDH resulting from bridging vein failure, there is limited documentation on the mechanism of SDH as a result of other bleeding sources, or on how tolerance to these types of SDH changes with age. Studies that have addressed SDH from sources other than bridging veins have focused on the conditions under which SDH from sources other than bridging veins occur rather than the mechanical loading required to produce both the original bleeding injury and the SDH.

Comparison Studies of Subdural Hematomas from Different Bleeding Sources A long-term study of surgically treated SDH cases found that in 40.5% of cases there was a laceration, intracerebral clot, or "exploded" temporal poles that was a cause of the SDH [Jamieson and Yelland, 1972]. These were categorized as "complicated" SDH and were more common among males but less common for adults over age 60. These complicated SDH were more common in motor vehicle crashes than "simple" SDH that were not associated with a surface contusion or laceration. In complicated SDH cases, the head impact was lateral 55% of the time. Among those lateral head impact cases, the impact and SDH were on the same side in 53% of cases and on contralateral sides in 47%.

In an autopsy study comparing 42 SDH cases with associated cerebral contusions to 30 cases of isolated SDH [Maxeiner, 1998], those with contusions were more common in falls (81%) or traffic events (17%), while isolated subdural cases occurred most often in falls (37%), from violence (26%), or from unclear sources [Maxeiner, 1998]. Isolated SDH were attributed to lateral impacts in only 12% of cases where direction was determined, while those with contusions were sustained in lateral impacts in 44% of cases.

While these two previous studies suggest that there are differences in the head impact conditions that produce SDH by bridging vein failure versus by other sources, they do not address the mechanical loading that results in SDH from sources other than bridging veins or address how tolerance might be affected by age.

Objective of Current Study To determine how tolerance to SDH changes for older occupants, the bleeding sources for SDH among different age groups need to be identified. With bleeding source information by age group, it can be determined if either bridging vein ruptures or other bleeding sources are primarily responsible for the increase in SDH for older occupants.

The current study uses available US crash data to explore how crash and injury characteristics for occupants who sustain SDH change with age. Specifically, the available US crash data was analyzed to look for any evidence that older occupants may be more susceptible to SDH as a result of bridging vein failure versus as a result of contusions or ruptured vessels on the surface of the brain.

METHODS

In order to understand how the risk of SDH changes for motor vehicle crash occupants as they age, US databases were used to compare crash and injury characteristics for adult SDH cases in different age groups, with a focus on evidence of the original bleeding source.

Medical information available in CIREN was used for detailed review of cases where the probable bleeding source in SDH cases could be identified. Cases were categorized by the bleeding source: *bridging veins*, *brain contusions*, or *other bleeding sources*.

Because NASS CDS does not contain the detailed medical information included in CIREN, a different approach was used to identify possible bleeding sources for SDH. Crash data from NASS CDS was compiled to determine the frequency of different serious bleeding head injuries that accompany SDH. SDH cases were sorted into three categories. Those sustained in the absence of other serious head injuries were identified as *isolated*. The remaining cases were categorized as SDH *with contusion* or SDH *with other serious head injury*. In the absence of specific information on bleeding sources in NASS CDS cases coded with SDH, it was assumed that isolated cases with no other serious head injury documented were more likely than other cases to be associated with bridging vein failure directly into the subdural border cell region since bridging veins are the only potential bleeding source that pass through the dural border cell layer. Similarly it was assumed that subdural cases that also had a documented brain contusion were the injury category most likely to include SDH sourced to contusions. Other potential subdural bleeding sources were combined in a third category with all other serious head injuries.

CIREN Case Review

All SDH cases in NHTSA's CIREN crash database up to August 2010 were selected. Those with recorded intracranial surgery for evacuation of SDH were reviewed individually. Operative reports were evaluated for documentation of bleeding source for each of these cases. SDH cases with operative reports were categorized into those associated with (1) bridging vein rupture, (2) brain contusions, or (3) other specified bleeding sources.

After categorizing the CIREN cases by associated bleeding source using the detailed medical descriptions available in the operative reports, the cases were also sorted by the accompanying injury categories used for the NASS CDS analysis using only the Abbreviated Injury Scale (AIS) coded injuries: *isolated, with contusion,* or *with other serious injury*. For example, a CIREN case that documented subdural bleeding from a bridging vein might have been coded as *isolated,* or *with other serious injury*, if it were sorted as in the NASS CDS analysis. A case-by-case comparison was made to determine if the NASS CDS accompanying injury categories, determined from AIS code only, were consistent with the CIREN categories based on the probable bleeding sources identified in the detailed records. This comparison was used to confirm the suitability of using the accompanying injuries in NASS CDS cases as surrogate variables for probable bleeding sources in the absence of explicitly coded information on bleeding source in NASS CDS.

NASS CDS Analysis

SDH cases in NASS CDS from 1993 to 2008 were selected based on seven-digit AIS codes. The AIS codes used in NASS CDS were based on the Association for the Advancement of Automotive Medicine's AIS-90 from 1993 to 1999, and on the

AIS-90/98 Update from 2000 to 2008. Serious injuries are those with an AIS score of 3 to 6.

All analyses compared three adult age groups: 20-49 years, 50-69 years, and age 70 and older. Occupants under age 20 were excluded. Occupants in all seat positions were included. Cases with unknown impact direction or velocity change were excluded only from analyses that included those variables. Weighted data was used for all NASS CDS analyses. Analysis was performed using survey analysis procedures in SAS, Version 9.2 (SAS Institute, Cary NC).

Accompanying Head Injuries The percentage of occupants in NASS CDS with SDH who also had other types of serious head injuries was estimated by age group. AIS 3+ head injuries were initially classified by their seven-digit AIS code into categories as follows:

- Skull fracture (*including crush, fracture)*
- Subarachnoid hemorrhage
- Contusions
- Intraventricular hemorrhage
- Epidural hematoma
- Other intracranial bleeding injury (*Including blood vessel injury, hematoma or hemorrhage not further specified, subpial hemorrhage, laceration or penetration, sinus bleeding, intracerebellar or intracerebral bleeding)*
- Closed head injury (*Including loss of consciousness or concussion, diffuse axonal injury, or closed head injury/blunt head injury/ traumatic brain injury not further specified)*
- Sequelae (*Including swelling, ischemia, pneumocephalus)*
- Brain stem injury (*Including any injury to the brain stem)*
- Other intracranial injury *(Any other injury to the AIS head region not included in the categories above, such as pituitary injury or serious scalp injury)*

SDH cases with no other serious injuries were classified as Isolated SDH.

Relative Frequency of Isolated Subdural Hematoma For the purpose of comparing the crash characteristics of SDH cases that were isolated to cases where the SDH was accompanied by a contusion, or by other serious head injury, SDH cases were grouped as follows:

Isolated
: No other documented AIS 3+ head injury

With contusion
: SDH combined with AIS 3+ coded brain contusion

With other serious injury
: SDH combined with serious head injuries (AIS 3+) other than contusion.

The frequency of isolated SDH was compared to the frequency of SDH accompanied by contusion or other serious injuries for different types of occupant and impact conditions. Frequency of each sub-type of SDH case was calculated as a percentage of all SDH cases among each occupant or crash type. Data is presented by sex, age group, vehicle impact direction, and head contact location.

Vehicle impact directions are defined by the direction of force (DOF1) variable in NASS CDS. Frontal impacts included those from 11 o'clock to 1 o'clock, as well as those at 10 or 2 o'clock only if the general area of damage (GAD1) was to the front of the vehicle. Side impacts were defined as all other cases with direction of force from 2 to 4 o'clock and 8 to 10 o'clock. Rear impacts included those from 5 to 7 o'clock and rollovers were any crash where the primary damage (variable TDD1) was overturn damage. Head contact locations were estimated based on the location of cutaneous and fracture injuries coded to the head and face. Injuries coded to the frontal aspect of the head were classified as frontal contacts and to the left or right aspects were classified as lateral contacts. Any face contact injury in combination with a frontal head contact, or without any other contact injuries to the head was classified as a frontal head contact. If face contact injuries were coded to the left or right aspect and were combined with a lateral head contact injury, the case was classified as a lateral head contact.

For vehicle side impact cases, the location of the SDH was compared to the vehicle impact direction. Cases where the vehicle impact and SDH were on the same side were categorized as ipsilateral, and those on opposite sides were contralateral. Side impact cases

with documented SDH on both sides of the brain were classified as bilateral.

Injury Rate Analysis For each of the three AIS-based accompanying injury categories (isolated, with contusion, and with other serious injury), injury rate was estimated by dividing the weighted number of injured occupants by the total weighted number of NASS CDS occupants in each age and impact direction category. The only cases excluded from the rate calculations were those where the occupant was listed as injured, but with details unknown (INJNO=97) since inclusion of these cases would increase the denominator of the rate calculations even though it was unknown whether they would be included in the numerator.

Injury rates were presented by age group for all occupants, then separately by impact direction for impact directions with sufficient numbers of cases to present rates by age group.

Odds Ratio Analysis Odds ratios were calculated to compare the odds of sustaining either isolated SDH or SDH with contusion for the oldest age group compared to the youngest age group. SDH cases with other serious head injuries were not included in the odds ratio analysis.

The SAS SURVEYLOGISTIC procedure was used to estimate odds ratios by age group separately for male and female occupants in frontal and side impacts. For each age and impact direction, a multinomial logistic regression model was developed, where the dependent injury variable could take on one of four values: no SDH, isolated SDH, SDH with contusion, or SDH with other serious injury. Categorical variables for age group were used to compare the 70 years and older group to a baseline 20 to 49 year-old group. To account for possible confounding differences in crashes for different age groups, independent variables reflecting potential differences in crash conditions and crash severity between young and old motor vehicle crash victims were included. The regression models for each sex and impact condition combination were built using a backward elimination stepwise procedure until the multinomial regression model contained only significant explanatory variables. Variables in the final models included a continuous variable for ΔV, dichotomous variables for the presence of a skull fracture and air bag deployment, and a categorical variable for seat position with possible values of driver, front seat passenger, or rear seat passenger.

Ninety-five percent confidence limits for the odds ratios were based on variance calculated by the Taylor series. In order to correct for simultaneous estimation of confidence intervals for each group (two-sided confidence interval for two injury types), Bonferroni correction was applied by dividing the significance level of 0.05 by 4 so that alpha was equal to 0.0125.

RESULTS

CIREN Search Results

A search of the CIREN database identified 277 cases with SDH. Case occupant age ranged from 4 days to 96 years, with a mean age of 37 years. Eighty-one cases were fatal, with the mean age of the fatal cases being 44 years. Operative reports were available for 37 individuals who underwent surgical evacuation of the SDH. These operative reports were reviewed for documentation of the source of subdural bleeding in each case.

In fourteen of the 37 cases with surgical records, a specific source of adjacent bleeding was identified. Two of these cases involved children and were excluded from the current analysis. The remaining 12 CIREN cases with documented bleeding sources adjacent to the evacuated SDH are summarized in Appendix A. Included are occupant age and sex, as well as CIREN-estimated impact direction and change in velocity (ΔV) or barrier equivalent velocity (BEV) for the primary impact. The location of the impact on the head and the head contact surface were drawn from CIREN investigation conclusions. Cases involving bridging vein bleeding sources are listed in Table A1, and cases involving only other bleeding sources are in Table A2.

In *four* of the twelve adult CIREN cases with documented bleeding source adjacent to the evacuated SDH, a bridging vein was the only identified bleeding source. In *two* of the twelve cases, bleeding was documented from a bridging vein as well as from cortical vessels: the vein of Labbé in the case

involving a 46 year-old woman and a posterior parietal superficial cortical vein in the case involving a 27 year-old woman (Table A1). In the remaining *six* adult CIREN cases with bleeding sources listed in the operative reports, bridging veins were not involved (Table A2). In three of those cases, documented bleeding adjacent to the evacuated SDH was attributed to contusions. Of the remaining three cases attributed to other bleeding sources, two documented intracerebral hematomas and the third documented an adjacent arterial bleeder on the brain surface.

The CIREN cases with information on subdural bleeding source were also reviewed to determine how they would have been categorized under the AIS-based accompanying injuries system used to sort cases in the NASS CDS analysis (Table 1). The objective was to determine whether the AIS-based categories to be used as surrogate variables for bleeding sources were consistent with the actual bleeding source identified in CIREN cases with additional medical information.

Table 1. Number of CIREN cases in each injury source category sorted by how they would have been categorized under AIS-based accompanying injury system used in NASS CDS analysis.

		Bleeding Source (from CIREN)		
		Bridging Vein	**Contusion**	**Other**
Based on AIS only	***Isolated***	**2 cases**		1 case
	With Contusion		**3 cases**	
	With Other Serious Injury	4 cases		2 cases

Of the CIREN cases, three would have been classified as isolated, three as with contusion, and six as with other serious injury by the AIS-based system. As shown in Table 1, two of the three cases that would have been categorized as isolated using the AIS-based system were associated with bridging vein failure in the CIREN operative report. All three cases that would have been categorized as with contusion using the AIS-based system were associated with contusion in CIREN records. This correspondence provides support for the use of isolated and with contusion injury categories as surrogate variables for SDH originating from bridging vein rupture and contusion, respectively. Of the CIREN cases that would have been categorized as with other serious injury by the AIS-based system, four were actually associated with bridging vein failure, and two were attributed to adjacent hematomas. Thus, the other serious injury category was not used as a surrogate variable for any specific bleeding source.

NASS CDS Search Results

In NASS CDS 1993-2008, there were 1,943 adult occupants coded with SDH, representing a weighted total of 105,980 cases. Among these, 46,907 cases were occupants age 20-49, 41,578 were age 50-69, and 17,494 were age 70 or older.

Accompanying Head Injuries The percentage of adult SDH cases where there were also other serious head injuries documented are shown in Figure 1. The percentages for each age group sum to greater than 100% since each SDH case may have multiple accompanying serious head injuries.

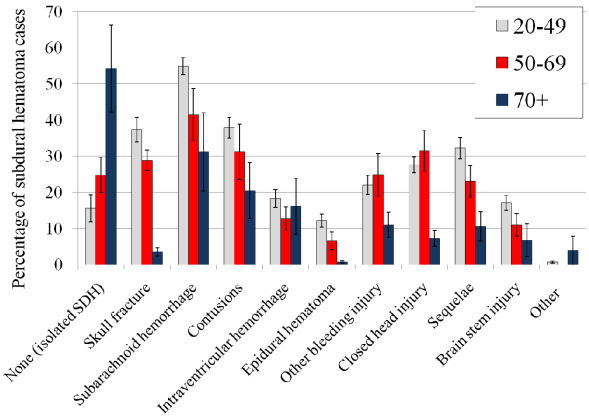

Figure 1. Percentage of SDH cases in each age group where other serious head injuries are also present (with standard error).

Among the youngest age group, 16% of those with SDH sustained no other serious head injuries. In the oldest age group, 54% of SDH cases were isolated. For adults in all age groups, subarachnoid hemorrhage and contusions were among the most common serious injuries to accompany SDH. For those younger than 70, skull fracture and closed head injury were recorded

in more than 25% of subdural cases. In contrast, among the oldest group of occupants with SDH, skull fractures were documented in fewer than 3% of cases and closed head injuries in fewer than 10% of cases. For the youngest age group, injuries in the sequelae category were also common.

Relative Frequency of Isolated Subdural Hematoma The frequency of isolated SDH versus SDH with contusion or with other serious injury was compared for occupants by age group and sex (Figure 2) as a percentage of all SDH cases. The percentage of subdural cases that are isolated among younger occupants is similar for men (15%) and women (17%). Among older occupants, however, the percentage of SDH that are isolated among men increases to only 33% while for women isolated SDH make up 65% of all subdural cases.

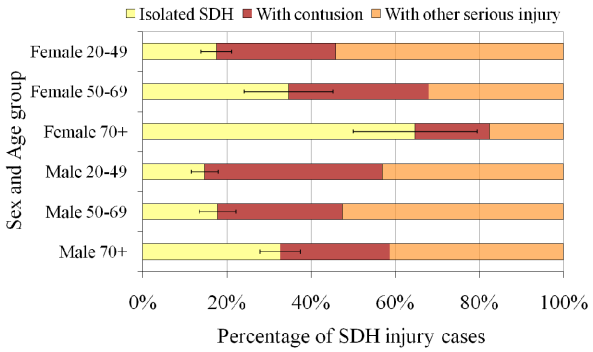

Figure 2. Percentage of SDH cases by sex and age group that are isolated versus with contusion or other serious head injury (standard error shown for percentage of isolated only).

The frequency of isolated SDH compared to SDH with contusion or SDH with other serious head injury is compared by crash impact direction in Figure 3. Of the vehicle impact directions, frontal impacts showed the highest percentage of isolated SDH, with over 30% of frontal SDH cases having no other serious head injuries.

Broken down by head impact location where cutaneous or bone injuries were present, Figure 4 shows that cases with isolated SDH were more likely to be associated with evidence of only frontal contact while cases with contusion were more likely to have evidence of a head impact on the side, either alone or with frontal contact.

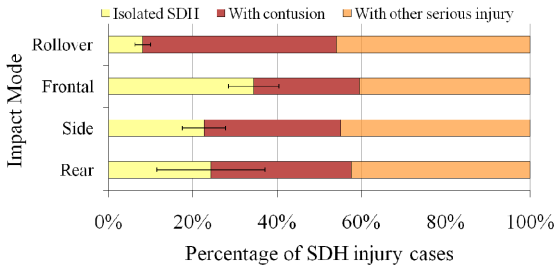

Figure 3. Percentage of SDH cases by impact direction that are isolated versus with contusion or other serious head injury (standard error shown for percentage of isolated only).

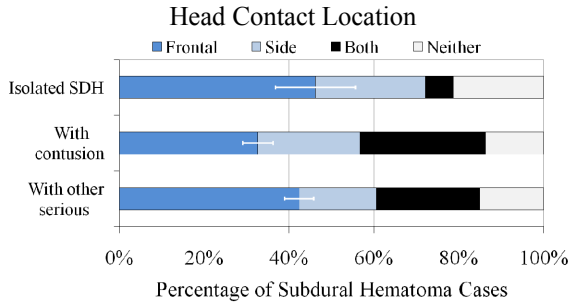

Figure 4. Percentage of SDH cases by accompanying injury category that have evidence of head contact location (with standard error shown for frontal cases only).

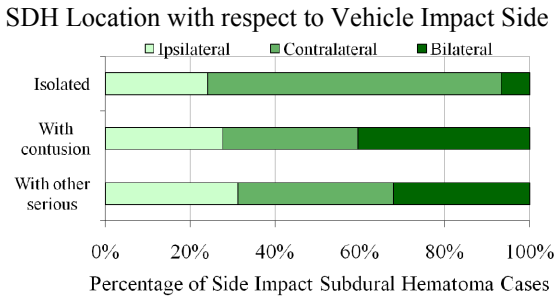

Figure 5. Percentage of side impact SDH cases by type (isolated versus accompanied by contusion or other serious head injuries) where SDH is ipsilateral or contralateral to vehicle impact side.

For side impact cases with SDH, the vehicle impact direction was compared to the side of the SDH (Figure 5). Cases where the vehicle impact and subdural were on the same side were categorized as ipsilateral, and those on opposite sides were contralateral. Cases with

isolated SDH were rarely bilateral (7%) compared to cases with subdural and contusion (41% bilateral) or subdural and other serious injury (32% bilateral). Isolated SDHs were predominantly contralateral (69%). In subdural cases with contusion or other serious head injury, ipsilateral or contralateral SDH occurred with similar frequency.

Injury Rate Analysis The rate of SDH injury goes up with age, regardless of whether the injury is isolated or in combination with contusion or other serious head injuries (Figure 6), but the increase in injury rate for occupants age 70 and older is especially dramatic for isolated SDH. The oldest group had a rate of isolated SDH that was 15 times higher than the rate of injury for younger occupants. For younger occupants, the rate of SDH in combination with contusion or other serious head injury is higher than the rate of isolated SDH. The opposite is true for older occupants, who are twice as likely to sustain an isolated SDH as one accompanied by contusion or other serious head injury.

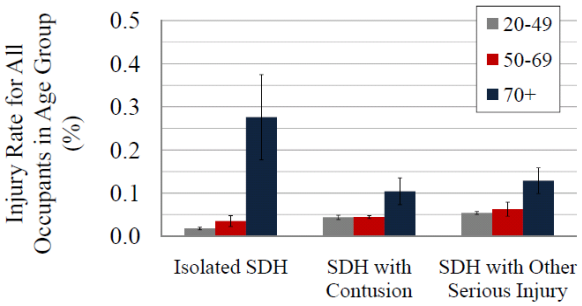

Figure 6. Injury rate for each type of SDH as percentage of all included occupants in all crash directions in each age group (with standard error).

When injury rates are calculated by impact direction (Figure 7), the age-related increase in rate of isolated SDH is especially notable in frontal impacts, while side impacts show a consistent increase in all types of SDH for the oldest occupants. The rate of SDH with contusions is higher in side impact than in frontal impact for all age groups.

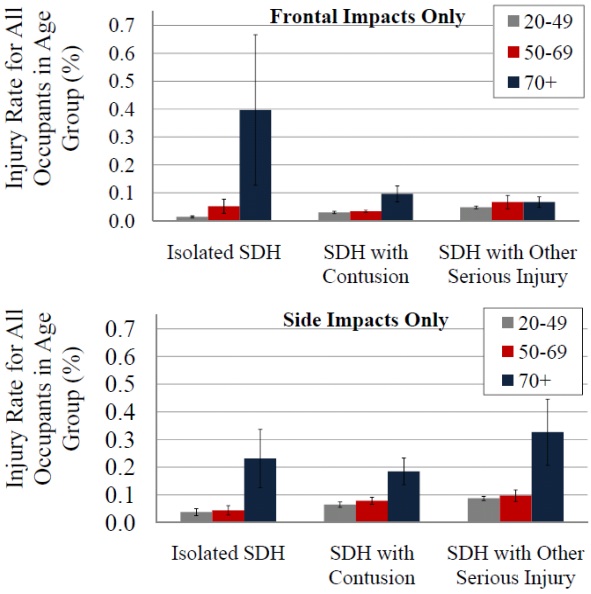

Figure 7. Injury rate for each type of SDH as percentage of all included occupants in frontal and side impact crashes (with standard error).

Odds Ratios Odds ratios were calculated to compare the odds of sustaining either isolated SDH or SDH accompanied by contusion for the oldest age group compared to the youngest age group. Regression models developed using backward elimination for males and females in frontal impacts included the potential confounding variables ΔV, skull fracture, and occupant seat location (driver, front passenger or rear passenger). Models for side impact for both males and females additionally included the potential confounder of air bag deployment.

Table 2 shows the point estimate for each odds ratio for males and females and for impact direction separately. To compare odds of each type of injury for older occupants versus younger adults, injuries that have a point estimate odds ratio greater than 1.0 and do not include 1.0 in the Bonferroni-corrected 95% confidence interval are annotated with an asterisk(*) to indicate significantly increased odds of injury for the older age groups. The resulting odds ratio for males in frontal crashes, for example, estimates that the odds of isolated SDH for an individual in the oldest age group were 11.4 times the odds of injury for a young adult *under the same conditions.* Since the entire confidence interval is greater than 1.0, this difference is significant at the 95% confidence level. As shown in Table 2, odds of injury for isolated SDH or for SDH

with contusion are especially elevated for older occupants in frontal crashes. The age-related increases in injury odds in frontals are particularly notable for isolated SDH and for women.

Table 2.
Odds ratios for types of SDH for 70+ age group compared to age 20 to 49 age group (Significant point estimates highlighted and marked with an asterisk*)

			Odds Ratio	95% CI
Frontal	Male	Isolated SDH	11.4*	2.0 - 64.9
		With Contusion	2.6*	1.03 - 6.7
	Female	Isolated SDH	118.4*	11.9 - >999
		With Contusion	11.5*	2.4 - 54.5
Side	Male	Isolated SDH	2.1	0.4 - 12.5
		With Contusion	2.4	0.7 - 7.8
	Female	Isolated SDH	4.5	0.7 - 30.4
		With Contusion	3.9*	1.6 - 9.4

DISCUSSION

Previously, the focus in SDH tolerance research has been on those originating from bridging vein failure. To begin to explore how tolerance to SDH changes with age, it needs to be determined whether bridging vein failure is the dominant injury mechanism for adults of all ages, or whether certain bleeding sources are particularly associated with the increase in SDH for older occupants.

Although autopsy and surgery studies have shown that SDH are commonly caused by bleeding from sources other than bridging veins [Jamieson and Yelland, 1972, Maxeiner, 1998], no large scale crash data set includes bleeding source information for SDH to evaluate the proportion of motor vehicle crash SDH cases that are caused by bleeding sources other than torn bridging veins for different age groups.

CIREN Results The current analysis of the CIREN database revealed a small number of cases where surgical reports documented adjacent bleeding that was assumed to be the most probable source of bleeding in each case. Review of these cases demonstrated that bridging veins are not the exclusive cause of SDH for any age group, and that other bleeding sources do need to be considered. SDH cases in CIREN were associated with surface contusions, intracerebral hematoma, and bleeding vessels on the surface of the brain as well as with bridging vein bleeding (Tables A1 and A2). The results of the CIREN search were consistent with previous studies that showed that SDH was not exclusively associated with bleeding from bridging veins and that SDH from non-bridging vein sources were relatively common in motor vehicle crashes [Jamieson and Yelland, 1972, Maxeiner, 1998].

Although review of the CIREN cases showed that bleeding sources other than bridging veins can contribute to SDH, there are too few cases with documentation on bleeding sources to analyze the distribution of age or crash conditions among cases originating either with bleeding from the bridging veins or from other bleeding sources.

Comparison of Probable Bleeding Source by Categories in CIREN and NASS CDS Analyses In contrast, NASS CDS has a large number of occupants coded with SDH, but no detailed medical information to specifically attribute those injuries to individual sources of bleeding. Therefore, the current study used available information on accompanying head injuries in NASS CDS to categorize the cases according to other serious head injuries present. In the absence of specific information on bleeding sources in cases coded with SDH, the accompanying injuries were used as surrogate variables for potential bleeding sources for the SDH in each case.

CIREN cases were used to evaluate the suitability of categorizing NASS CDS occupants into isolated SDH cases and SDH cases with contusion as surrogate variables for bridging-vein sourced subdural cases and contusion-sourced SDH cases, respectively. The CIREN cases with documented probable bleeding sources were reviewed and sorted by the same categories used in the NASS CDS search based on AIS codes (Table 1). The objective was to confirm whether the information drawn from AIS codes was consistent with the more detailed information available in operative reports. A good match would support use of the AIS-based categories as surrogate variables for probable bleeding source in NASS CDS which has no narrative injury detail.

Of the three CIREN cases that would have been classified as isolated from the categories used in the NASS analysis, two were indeed associated with bridging vein failure and one with a torn artery on the surface of the brain. Of the three cases that would have been classified as subdural with contusion, all three were associated with a contusion in the operative report. Among the six cases that would have been classified as subdural with other serious injury, operative reports indicated that two were associated with intracerebral hematoma, and four were associated with bridging vein failure or bridging vein failure as well as failure of other surface veins. This comparison suggests that cases categorized as isolated SDH are more likely to be associated with bridging vein rupture than with other sources and that cases in the SDH with contusion category are more likely to be from contusions than from bridging veins or other sources. The other serious head injury category may contain cases caused by bridging vein rupture or from bleeding sources on the surface of the brain. Therefore, although the injury categories used are only surrogates for variables describing the original source of bleeding, it is reasonable to use the trends in the isolated SDH and the SDH with contusion categories to approximate trends between cases where the SDH was produced by bridging vein bleeding versus by brain contusions, respectively. Since the true source of subdural bleeding is not available in NASS CDS or any other large-scale crash database, this data represents the best available estimate.

NASS CDS Results Based on the assumption that the injury categories used are reasonable surrogate variables for bleeding source for SDH, the increased tendency for older occupants to sustain isolated SDH (Figure 1) potentially reflects an increased frequency of SDH caused by bridging vein damage. Although the rate of SDH increases with age, regardless of accompanying injuries (Figure 6), cases with isolated SDH are 15 times more frequent among those aged 70 and older than among adults younger than 50. In comparison, SDH with contusion or other serious injury is only about 2.4 times as frequent among the oldest group compared to the youngest group.

The age-associated increase in isolated SDH is especially prominent for women and in frontal impacts (Table 2). Although isolated SDHs make up about the same proportion of SDH cases in women and men in the age 20 to 49 age group (15% and 17% respectively), SDH in the 70 and older age group are isolated in 65% of female cases but only 33% of male cases (Figure 2). SDH are isolated in 34% of cases sustained in frontals, compared to 23% of cases in side impacts (Figure 3). For the oldest age group, the injury rate for isolated SDH in frontals is almost twice that in side impacts (Figure 7). These results suggest that the age-related increase in bridging vein-associated SDH may especially affect women and those in frontal impacts.

Odds ratio analysis (Table 2) shows that the age-related increase in SDH in frontal crashes is present even after accounting for variables related to crash and head impact severity (ΔV, air bag deployment, skull fracture), especially for isolated SDH, and for women.

Assuming that isolated SDH are most likely to be associated with bridging vein bleeding, the current findings that the age-related increase in isolated SDH may be more frequent in frontal impacts are consistent with studies that have linked anterior-posterior motion to bridging vein failure [Depreitere et al., 2006, Gennarelli and Thibault, 1982, Löwenhielm, 1974, Ommaya et al., 1968] and consistent with the explanation that older individuals are more prone than younger individuals to SDH by bridging vein failure due to the increased relative brain motion and bridging vein tension induced by atrophy in the aging brain [Kleiven and von Holst, 2002, Meaney, 1991, Yamashima and Friede, 1984].

The case conditions for the SDH cases with contusion in the current study were compared to previous findings on cases with SDH with contusion [Maxeiner, 1998], or caused by bleeding sources that were intracerebral or on the surface of the brain rather than by bridging vein failure [Jamieson and Yelland, 1972]. In Jamieson and Yelland, the SDH cases with intracerebral or surface bleeds were more common among males. In the current study, SDH were accompanied by contusion more often among men than women, especially for *young* males under age 50 who sustained contusion with subdural more than twice as often as they sustained isolated SDH (Figure 2). Jamison and Yelland also reported that SDH caused by intracerebral or surface bleeding was *less*

common for adults over age 60. In the current study, although the injury rate for SDH with contusion increases for older occupants (Figure 6), the proportion of SDH that are accompanied by contusions decreases with age: only 20% of subdural cases among those age 70 and older are associated with contusion, compared to 38% of subdural cases for adults under age 50 (Figure 1). The reason for the reduction in the proportion of subdural injuries with contusion is that the age-related increase in rate of isolated SDH outpaces the age-related increase in rate of SDH with contusion. In previous studies, SDH associated with sources other than bridging veins were more frequently from lateral impact than those associated with bridging veins. In Jamieson and Yelland, 55% of complicated SDH cases were from lateral head impacts, while only 32% of isolated SDH cases were from lateral head impacts. Maxeiner reported 44% of SDH associated with contusion were confirmed to involve lateral head impacts while only 12% of bridging vein subdural hematoma were lateral head impacts. In the current study, 29% of cases with contusion were lateral vehicle impacts compared to 23% of isolated SDH cases which were lateral vehicle impacts.

Overall, the crash conditions most often associated with isolated SDH are different from those associated with SDH combined with contusion. While the rate of both types of subdural increases with age, the age-related increase is greater for isolated SDH, especially among women. For the oldest group of occupants, the rate of isolated SDH is almost twice as high in frontals as in side impacts, while SDH with contusions occur at a higher rate in side impacts than in frontals for all age groups. Isolated SDH occur bilaterally in only 7% of cases, compared to subdural with contusion cases which are bilateral in 41% of cases (Figure 4). Among the isolated subdural cases, the hematoma is contralateral to the vehicle impact side in 69% of cases, while contralateral SDH are documented in only 32% of subdural cases with contusion (Figure 5). These differences between cases with isolated SDH and SDH combined with contusion support that these two injury categories are representative of two different patterns of injury. Assuming that these injury categories correspond approximately to SDH caused by bridging vein failure and SDH caused by contusions or other bleeding sources, the results suggest that the mechanism of injury for these two types of SDH may be quite different.

The types of motion and forces required to produce SDH from bleeding sources on the surface of the brain have not been addressed. Although bleeding from cortical contusions or ruptured cortical vessels into the subdural border cell region would only be possible with rupture of the arachnoid, the loading required to produce this combination of bleeding injury and arachnoid damage has not been explored experimentally, nor have the effects of age on this process been explored. Research on the tolerance of occupants of all ages to SDH would benefit from a better understanding of the mechanism of SDH from sources other than bridging veins.

Older occupants appear to be particularly vulnerable to isolated SDH, which suggests that the increase in SDH rate in older occupants may be more dependent on decreased tolerance to SDH associated with bridging vein failure than to those associated with other bleeding sources. Further research on how tolerance to bridging vein failure changes with age may explain much of the increase in incidence of SDH among older occupants.

CONCLUSIONS

The rate of isolated SDH among crash occupants increases with age, potentially reflecting an increase in the frequency of SDH caused by bridging vein bleeding. This age-related increase is especially prominent among women and in frontal impacts. Determination of age-specific injury tolerance for SDH will require evaluation of the increasing risk of bridging vein failure in older occupants.

SDH accompanied by brain contusions or other potential bleeding sources are also common, especially in side impacts and among occupants younger than 70. These cases potentially represent injuries where subdural bleeding came from sources other than bridging veins. Estimation of the SDH tolerance for all adult occupants will require a better understanding of the mechanism of injury for SDH from these other bleeding sources.

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REFERENCES

Depreitere, B., C. Van Lierde, P. Verschueren, H. Delye, D. Berckmans, I. Verpoest, J. Vander Sloten, G. Van der Perre and J. Goffin. 2005. "Cerebral Bridging Vein Rupture in Humans: An Experimental Evaluation." Iutam Symposium on Impact Biomechanics, Gilchrist, M. D., ed., Springer, pp. 305-12.

Depreitere, B., C. Van Lierde, J.V. Sloten, R. Van Audekercke, G. Van der Perre, C. Plets and J. Goffin. 2006. "Mechanics of Acute Subdural Hematomas Resulting from Bridging Vein Rupture." J Neurosurg, 104(6), pp. 950-6.

Gennarelli, T.A. and L.E. Thibault. 1982. "Biomechanics of Acute Subdural Hematoma." J Trauma, 22(8), pp. 680-6.

Gennarelli, T.A. and D.I. Graham. 2005. "Neuropathology." Textbook of Traumatic Brain Injury, Silver, J. M., T. W. McAllister and S. C. Yudofsky, eds., American Psychiatric Publishing, Inc., Washington, DC.

Graham, D.I. 1996. "Neuropathology of Head Injury." Neurotrauma, Narayan, R. K., J. E. Wilberger and J. T. Povlishock, eds., McGraw Hill, New York, pp. 43-59.

Haines, D.E., H.L. Harkey and O. Al-Mefty. 1993. "The "Subdural" Space: A New Look at an Outdated Concept." Neurosurgery, 32(1), pp. 111-20.

Hanif, S., O. Abodunde, Z. Ali and C. Pidgeon. 2009. "Age Related Outcome in Acute Subdural Haematoma Following Traumatic Head Injury." Irish Medical Journal, 102(8), pp. 255-7.

Holbourn, A.H.S. 1943. "Mechanics of Head Injuries." Lancet, 2, pp. 438-41.

Hukkelhoven, C.W., E.W. Steyerberg, A.J. Rampen, E. Farace, J.D. Habbema, L.F. Marshall, G.D. Murray and A.I. Maas. 2003. "Patient Age and Outcome Following Severe Traumatic Brain Injury: An Analysis of 5600 Patients." J Neurosurg, 99(4), pp. 666-73.

Jamieson, K.G. and J.D. Yelland. 1972. "Surgically Treated Traumatic Subdural Hematomas." J Neurosurg, 37(2), pp. 137-49.

Jane, J.A. and P.C. Francel. 1996. "Age and Outcome of Head Injury." Neurotrauma, Narayan, R. K., J. E. Wilberger and J. T. Povlishock, eds., McGraw-Hill, pp. 793-804.

Kleiven, S. and H. von Holst. 2002. "Consqueences of Reduced Brain Volume Following Impact in Prediction of Subdural Hematoma Evaluated with Numerical Techniques." Traffic Injury Prevention, 3, pp. 303-10.

Lee, M.-C., J.W. Melvin and K. Ueno. 1987. "Finite Element Analysis of Traumatic Subdural Hematoma." 31st Stapp Car Crash Conference, SAE, pp. 67-77.

Lee, M.C. and R.C. Haut. 1989. "Insensitivity of Tensile Failure Properties of Human Bridging Veins to Strain Rate: Implications in Biomechanics of Subdural Hematoma." J Biomech, 22(6-7), pp. 537-42.

Löwenhielm, P. 1974. "Strain Tolerance of the Vv. Cerebri Sup. (Bridging Veins) Calculated from Head-on Collision Tests with Cadavers." Z Rechtsmed, 75(2), pp. 131-44.

Mallory, A. 2010. "Head Injury and Aging: The Importance of Bleeding Injuries." Ann Adv Automot Med, 54, pp. 51-60.

Maxeiner, H. 1997. "Detection of Ruptured Cerebral Bridging Veins at Autopsy." Forensic Sci Int, 89(1-2), pp. 103-10.

Maxeiner, H. 1998. "Subdural Hematomas in Victims with and without Cerebral Contusions - Comparision of Two Types of Head Injuries." Rechtsmedizin, 9(1), pp. 14-20.

Maxeiner, H. and M. Wolff. 2002. "Pure Subdural Hematomas: A Postmortem Analysis of Their Form and Bleeding Points." Neurosurgery, 50(3), pp. 503-9.

Meaney, D.F. 1991. "Biomechanics of Acute Subdural Hematoma in the Subhuman Primate and Man." Ph.D. Dissertation, University of Pennsylvania.

Miller, D.J., I.R. Piper and P.A. Jones. 1996. "Pathophysiology of Head Injury." Neurotrauma, Narayan, R. K., J. E. Wilberger and J. T. Povlishock, eds., McGraw Hill, New York, pp. 61-9.

Miller, J.D. and P.F.X.S. Statham. 2000. "Surgical Management of Traumtic Intracranial Hematomas." Operative Neurosurgical Techniques, Schmidek, H. H., ed., W.B. Saunders Company, Philadelphia, PA, pp. 83-90.

Monson, K.L. 2001. "Mechanical and Failure Properties of Human Cerebral Blood Vessels." Ph.D. Dissertation, University of California, Berkeley.

Ommaya, A.K., F. Faas and P. Yarnell. 1968. "Whiplash Injury and Brain Damage: An Experimental Study." JAMA, 204(4), pp. 285-9.

Perel, P., I. Roberts, O. Bouamra, M. Woodford, J. Mooney and F. Lecky. 2009. "Intracranial Bleeding in Patients with Traumatic Brain Injury: A Prognostic Study." BMC Emergency Medicine, 9(1), p. 15.

Raju, S., D.K. Gupta, V.S. Mehta and A.K. Mahapatra. 2004. "Predictors of Outcome in Acute Subdural Hematoma with Severe Head Injury - a Prospective Study." Indian Journal of Neurotrauma, 1(2), pp. 37-44.

Sawauchi, S. and T. Abe. 2008. "The Effect of Haematoma, Brain Injury, and Secondary Insult on Brain Swelling in Traumatic Acute Subdural Haemorrhage." Acta Neurochir (Wien), 150(6), pp. 531-6; discussion 6.

Seelig, J.M., D.P. Becker, J.D. Miller, R.P. Greenberg, J.D. Ward and S.C. Choi. 1981. "Traumatic Acute Subdural Hematoma: Major Mortality Reduction in Comatose Patients Treated within Four Hours." N Engl J Med, 304(25), pp. 1511-8.

Servadei, F. 1997. "Prognostic Factors in Severely Head Injured Adult Patients with Acute Subdural Haematoma's." Acta Neurochir (Wien), 139(4), pp. 279-85.

Stitzel, J.D., P.D. Kilgo, K.A. Danelson, C.P. Geer, T. Pranikoff and J.W. Meredith. 2008. "Age Thresholds for Increased Mortality of Three Predominant Crash Induced Head Injuries." Annu Proc Assoc Adv Automot Med, 52, pp. 235-44.

Taussky, P., H.R. Widmer, J. Takala and J. Fandino. 2008. "Outcome after Acute Traumatic Subdural and Epidural Haematoma in Switzerland: A Single-Centre Experience." Swiss Med Wkly, 138(19-20), pp. 281-5.

Toyama, Y., T. Kobayashi, Y. Nishiyama, K. Satoh, M. Ohkawa and K. Seki. 2005. "CT for Acute Stage of Closed Head Injury." Radiat Med, 23(5), pp. 309-16.

Yamashima, T. and R.L. Friede. 1984. "Why Do Bridging Veins Rupture into the Virtual Subdural Space?" J Neurol Neurosurg Psychiatry, 47(2), pp. 121-7.

Table A1.
CIREN cases with operative report documentation of bridging vein bleeding adjacent to surgically-evacuated subdural hematoma

Age, Sex & Seat Position	Impact Direction (PDOF)	Crash Severity	Head impact	SDH location	Adjacent Bleeding Source Identified in Operative Report	Other Serious Head Injuries (From AIS-codes with additional detail from diagnostic imaging where available)	AIS-based accompanying injury category as used in NASS CDS analysis
26 y.o. female driver	11 o'clock	BEV= 49 km/h	Roof/side rail with left head	Left panhemispheric and over tentorium	*Bridging vein:* Two lacerated bridging veins near midline	Left cerebral swelling AIS 4 Left subcortical hemorrhage AIS 4 Left subarachnoid hemorrhage AIS 3	Other serious injuries
85 y.o. female right front	12 o'clock	ΔV= 22 km/h	Passenger-side frontal air bag with left face	Left frontotemporal	*Bridging vein:* Torn bridging vein "was cause of hematoma"	**(Fatal)** Cerebral edema AIS 3	Other serious injuries
16 y.o. female right rear	3 o'clock	ΔV= 53 km/h	Left forehead to seatback, right occiput to unknown surface	Left temporal to convexity and over tentorium	*Bridging vein:* Hole in superior sagittal sinus from an evolved bridging vein	Right intracranial vessel laceration AIS 4	Other serious injuries
18 y.o. female driver	2 o'clock	ΔV= 59 km/h	Right door impact documented	Right	*Bridging vein:* Torn parasagittal vein	None	Isolated
46 y.o. female right front	3 o'clock	ΔV= 27 km/h	Right parietal / temporal to door interior or striking vehicle	Right frontal temporal and parietal	*Bridging vein & other:* Torn vein of Labbé, petrosal vein, and sylvian bridging vein	Subarachnoid hemorrhage AIS 3	Other serious injuries
27 y.o. female driver	1 o'clock	ΔV= 32 km/h	Left face and occiput to A-pillar and roof	Left frontal, parietal	*Bridging vein & other:* Bleeding from bridging vein and superficial cortical vein	None	Isolated

Table A2.

CIREN cases with operative report documentation of bleeding adjacent to subdural hematoma from sources *other than bridging veins*

Age, Sex & Seat Position	Impact Direction (PDOF)	Severity	Head impact	SDH location	Adjacent Bleeding Source Identified in Operative Report	Other Serious Head Injuries (From AIS-codes with additional detail from diagnostic imaging where available)	AIS-based accompanying injury category as used in NASS CDS analysis
76 y.o. female driver	2 o'clock, (rotated to 3 o'clock by time of head impact)	BEV= 38 km/h	Right passenger door with right forehead	Bilateral (surgery on left)	Left temporal/parietal intracerebral hematoma	**(Fatal)** Right temporal hinge fracture AIS 4 Right white matter hemorrhage and left temporoparietal intracerebral hematoma AIS 4 Right subarachnoid hemorrhage AIS 3	Other serious injuries
23 y.o. male driver	10 o'clock	ΔV= 28 km/h	B-pillar with left head, seat with right head	Right temporal and along tentorium	Temporal and parietal surface contusions and bleeders	Right epidural hematoma AIS 4 Basilar and right vault fractures AIS 4 Hemorrhagic contusions under SDH AIS 4 Right subarachnoid hemorrhage AIS 3	Contusion
21 y.o. female driver	4 o'clock	ΔV= 26 km/h	B-pillar with right occiput	Left frontal/ temporal	Temporal bleeders and bruises with contusions	Bilateral cerebral swelling AIS 5 Subarachnoid hemorrhage AIS 3 Left frontal-temporal hemorrhagic contusion AIS 4	Contusion
76 y.o. female driver	10 o'clock	ΔV= 12 km/h	B-pillar with left side of head	Left parietal/ occipital	Small arterial bleeder on the brain surface	No other head injuries coded	Isolated
29 y.o. male driver	2 o'clock	ΔV= 44 km/h	A-pillar-mounted handle with right head	Right convexity	Temporoparietal intracerebral hematoma with bleeders	Right parietal skull fracture AIS 4 Right cerebral edema AIS 3 Right intracerebral hematoma not coded	Other serious injuries
49 y.o. female driver	3 o'clock	ΔV= 44 km/h	Tree with right head and face	Left frontal/parietal/ temporal	Underlying hemorrhagic contusion	**(Fatal)** Cerebral hematoma/hemorrhage AIS 4 Fractures basilar skull, right vault and orbit AIS 3 Left subarachnoid hemorrhage AIS 3 Bilateral frontal lobe contusions AIS 3	Contusion