Seatbelts contribute to location of lesion in moderate to severe closed head trauma

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Abstract

The relationship between seatbelt use and injury severity, brain lesion location, and functional outcome was investigated in 163 individuals who sustained traumatic brain injuries in motor vehicle collisions. Of this group, 31 were using a seatbelt at the time of the accident and 132 were not. Restrained motor vehicle occupants were significantly more likely to sustain damage to subcortical brain structures than unrestrained occupants. Conversely, unrestrained occupants sustained a greater frequency of posterior brain lesions. In addition, demographic and behavioral variables were significantly related to increased likelihood of seatbelt use. Analyses revealed no significant differences between groups for injury severity variables and functional outcome measures. Seatbelts alter the body's response to forces applied in motor vehicle collisions, creating disparities in lesion sites between restrained and unrestrained motor vehicle occupants. The relationship between seatbelt use and injury severity and functional outcome is discussed. © 2001 National Academy of Neuropsychology. Published by Elsevier Science Ltd.

In the US, 200 per 100,000 people suffer brain injuries each year (Frankowski, 1986); in over half of these cases, motor vehicle collisions are the cause (Kraus & McArthur, 1996). As a result, analyzing how motor vehicle collision factors influence incidence, severity, and outcome of traumatic brain injury (TBI) is of considerable importance. One factor that we investigated is the effect of seatbelt use on brain injury in cases of moderate to severe brain injury.

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The primary purpose in using a seatbelt is to allow the restrained individual to experience the vehicle's deceleration over a distance of several feet, instead of a few inches (Derosa & Larsonneur, 1984). Seatbelts have been shown to be an effective safety device (Derosa & Larsonneur, 1984; Engberg, 1995; Smith-Seemiller et al., 1997). However, the efficacy of seatbelt use in preventing brain injury appears to be limited in some circumstances. While seatbelt use has been associated with lower incidence of brain injury in some studies (Swierzewski et al., 1994), other results have failed to support this finding (Siegel et al., 1993). Investigations have revealed that the benefits of seatbelts are notably reduced during severe collisions (Campbell, 1987; Siegel et al., 1993) and lateral impact collisions (Siegel et al., 1993; Loo et al., 1997). In the former, compartmental intrusion factors have been implicated, while in the latter, seatbelt design limitations may be at fault (Siegel et al., 1993). In a comprehensive review, Engberg (1995) suggested that although the level of protection afforded by seatbelts may have been overstated, they remain effective in reducing the incidence of mild TBI.

Others have investigated the relationship between cognitive performance in TBI survivors and motor vehicle collision factors. Varney and Varney (1995) used detailed accident analyses to calculate how the physical forces applied during a collision (e.g., direction of impact and deceleration) influence the medical and neuropsychological sequelae of TBI. Of particular note in this study was the finding that brain injury and subsequent neuropsychological deficits may occur in the absence of head/obstacle contact. Such whip lash injuries are consistent with the kinds of injury that may be experienced by restrained passengers. In a recent study, Smith-Seemiller et al. (1997) evaluated the neuropsychological performance of restrained and unrestrained occupants. These authors reported greater executive function deficits, suggestive of frontal lobe dysfunction, on neuropsychological testing in their group of unrestrained TBI survivors.

The current investigation was developed to study the relationship between seatbelt use and location of brain injury, injury severity, and functional outcome. In particular, we sought to determine if there were differences in the location of brain injury among restrained and unrestrained occupants. In accordance with the findings of Smith-Seemiller et al. (1997), we hypothesized that failure to use a seatbelt would result in increased frontal lobe injury. Furthermore, we predicted that increased rates of frontal lobe injury in the unrestrained group would result in more compromised functional outcome. Through these analyses, we expected to gain a better understanding of the motor vehicle collision factors that influence rehabilitation and functional outcome.

1. Method

1.1. Participants

Of the 163 individuals who met the inclusionary criteria for this study, 31 were using a seatbelt at the time of accident (19% "restrained") and 132 were not using a seatbelt (81%

Table 1
Demographic and injury severity variables for restrained and unrestrained participants

Variable	Restrained ($n = 31$)	Unrestrained ($n = 132$)	Significance
Gender (% Male)	68	73	$\chi^2(1) = 0.41$
Age (in years; $M \pm SD$)	30.63 ± 13.69	26.16 ± 8.78	$t(161) = 2.23*$
Education (in years)	13.16 ± 2.37	11.88 ± 1.92	$t(161) = 3.20**$
Marital status			
% Married	32	14	
% Single	58	66	$\chi^2(3) = 6.31$
% Divorced/separated	10	20	
ETOH (% positive)	3	28	$\chi^2(1) = 7.99**$
GCS ($M \pm SD$)	6.07 ± 3.05	5.13 ± 1.68	$t(147) = 1.52$
LOC ($M \pm SD$)	4.71 ± 2.12	5.13 ± 1.68	$t(131) = -1.11$
Days acute ($M \pm SD$)	37.23 ± 28.27	45.73 ± 27.76	$t(157) = -1.53$

ETOH = presence or absence of blood alcohol at the time of injury, GCS = Glasgow Coma Scale score in the emergency room, LOC = length of loss consciousness, Days acute = days spent in acute hospital care.
* $p \leq 0.05$.
** $p \leq 0.01$.
*** $p \leq 0.001$.

"unrestrained"). We included brain injury survivors who were injured while domiciled in the state of Pennsylvania and who had applied to a state-funded post-acute rehabilitation program between 1985 and 1996.

The criteria for defining TBI in this study were described by Molitor (1990, pp. 1–2) as "an insult to the brain, not of degenerative or congenital nature, but caused by an external physical force that may produce a diminished or altered state of consciousness, which results in impairments of cognitive abilities or physical functioning." In addition to this definition, all study participants demonstrated at least one of five of the following symptoms associated with TBI (Schatz, 1995): (1) loss of consciousness (LOC) attributable to brain injury, (2) retrograde or posttraumatic amnesia attributable to brain injury, (3) objective neurologic signs of abnormality; (4) diagnosed intracranial lesion(s) obtained on radiologic examination or other neurodiagnostic procedures that are attributable to head injury, and/or (5) observed partial or generalized seizures within 24 h following brain trauma in the absence of history of seizure disorder.

Acute care and rehabilitation records were available for 494 brain injury survivors who met these criteria. From this group of potential participants, individuals were excluded for any of the following reasons: (1) TBI was not sustained as a motor vehicle occupant (243 individuals), (2) chart documentation of restraint use or non-use was not available (66 individuals), or (3) radiologic reports were unavailable (four individuals), or negative or equivocal (18 individuals). The remaining 163 subjects were eligible for this study. Demographic variables are presented in Table 1.

We excluded individuals with unavailable, negative, or equivocal radiologic data because equivocal data or negative data could not be used to indicate the absence of a lesion with a consistent level of confidence. In other words, although positive findings could be considered

to imply the true presence of a lesion in almost all cases (true positives), negative or equivocal findings provided much weaker evidence for the true absence of a lesion. Moreover, in those cases where no radiologic report was made, it was impossible to determine with certainty if this signified the absence or presence of a lesion. It should be noted that five of 18 individuals excluded because of negative or equivocal radiologic data were using seatbelts at the time of the accident.

1.2. Design and procedure

Acute care and rehabilitation records of restrained and unrestrained motor vehicle occupants were compared for demographic variables, injury severity measures, location of brain injury, and functional outcome.

1.3. Injury severity variables

Glasgow Coma Scale (GCS) scores (Teasdale & Jennett, 1974) and duration of LOC was obtained from acute care and rehabilitation hospital reports. In accordance with Katz and Alexander (1994), duration of LOC was recorded as the period of time from the date of injury to the date when the individual was first able to follow command. Because this information was typically garnered from chart records, LOC was coded as an ordinal level scale constructed with seven levels: 1 = less than 20 min, 2 = less than 24 h but greater than 20 min, 3 = less than 1 week but greater than 24 h, 4 = between 1 and 3 weeks, 5 = between 3 and 5 weeks, 6 = between 6 and 8 weeks, and 7 = greater than 8 weeks. Injury severity was also measured as a function of acute care length of stay, calculated as the length of time (in days) between the date of injury and the date of the individual's first discharge from acute care.

1.4. Location of brain injury coding

Computerized tomography (CT) and/or magnetic resonance imaging (MRI) results were culled from radiology reports or discharge summaries from acute care and rehabilitation hospital reports. All incidence of CT or MRI abnormality, including non-hemorrhagic and hemorrhagic infarctions, and epidural and subdural hematomas, were included in this analysis and were considered evidence of brain injury, or "lesions."

Lesions described in radiologic reports were noted for 15 anatomical regions that were further collapsed into six primary areas for analysis. These areas included each of the four cortical lobes, a group of subcortical structures, and a group of brainstem structures. The anatomical components of the subcortical group included the basal ganglia, corpus callosum, internal capsule, thalamus, and hypothalamus. The anatomical components in the brainstem group included the midbrain, pons, and medulla. These three areas correspond, roughly, to the following neurodevelopmental divisions: (1) telencephalon, (2) diencephalon, and (3) mesencephalon, metencephalon, and myelencephalon. Besides

these neurodevelopmental distinctions, these regions were selected, in part, because of the relatively distinct boundaries between these areas and the commonality of brain injuries in each of these regions following motor vehicle accident. Presence of lesion was recorded as a dichotomous variable within each region without regard to the number or size of lesions within that region.

1.5. Outcome variables

Functional outcome was measured at the time of last documented contact with the patient with two separate measures: the Disability Rating Scale (DRS; Rappaport et al., 1982) and the Functional Independence Level (FIL) (Schatz & Chute, 1995). The DRS was chosen because of its reliability and validity in measuring TBI outcome (Gouvier et al., 1987) as well as its sensitivity to survivor recovery across the course of rehabilitation (Rappaport et al., 1982; Hall et al., 1985). In order to account for limitations of the DRS, the FIL scale was also used. The FIL is a 10-point outcome measure based on cognitive and physical disability. The FIL assesses independence by noting the individual's ability to begin, link together, and finish activities of daily living. It also includes the patient's communication skills and judgment. Thus, where the DRS measures only the cognitive components of a particular skill, the FIL scale evaluates both physical disability and cognitive dysfunction. Despite these differences, these two measures are highly correlated; DRS scores account for 49% of the variance in FIL ratings (Schatz & Chute, 1995).

Because functional outcome measures were not consistently recorded in patient charts, both DRS and FIL scores were inferred by one of two raters who evaluated the individual's medical and rehabilitation records to the time of last contact. Raters were not blind to subjects' seatbelt use. However, a reliability study including three raters, one blind to the restraint status of the participants yielded good reliability between raters; the average of the three Spearman correlation coefficients was 0.91 for DRS scores and 0.88 for FIL scores. Our method of obtaining DRS and FIL scores from record review allows for the ascertainment of functional status at specific times during rehabilitation. However, because of this adaptation, DRS scores from our study should not be considered equivalent to other DRS scores reported in the literature.

2. Results

2.1. Demographic variable analysis

Significant between-group differences were noted for the following demographic variables: education, age, marital status, and use of alcohol at the time of the motor vehicle collision. Although restrained and unrestrained occupants were predominantly male, there was no significant effect of gender on frequency of seatbelt use. Results of demographic analyses are presented in Table 1.

2.2. Severity of injury analysis

Statistical analyses yielded no effect of restraint use on any of the injury severity variables. GCS scores between restrained ($M = 6.07$, SD $= 3.05$) and unrestrained ($M = 5.13$, SD $= 2.88$) occupants were similar, $t(147) = 1.52$, $p = 0.13$. Length of LOC was also not significantly different between restrained ($M = 4.71$, SD $= 2.12$) and unrestrained ($M = 5.13$, SD $= 1.68$) groups, $t(131) = -1.11$, $p = 0.27$. Length of acute care hospital stay also demonstrated no significant difference between restrained ($M = 37.23$, SD $= 28.27$) and unrestrained ($M = 45.73$, SD $= 27.76$) occupants, $t(157) = -1.53$, $p = 0.13$. Injury severity variables are also summarized in Table 1.

2.3. Location of injury analysis

Incidence and location of brain lesion were calculated for both restrained and unrestrained motor vehicle occupants. Statistical comparisons were conducted between groups for each of the cortical lobes (i.e., frontal, temporal, parietal, and occipital) as well as the subcortical and brainstem groups.
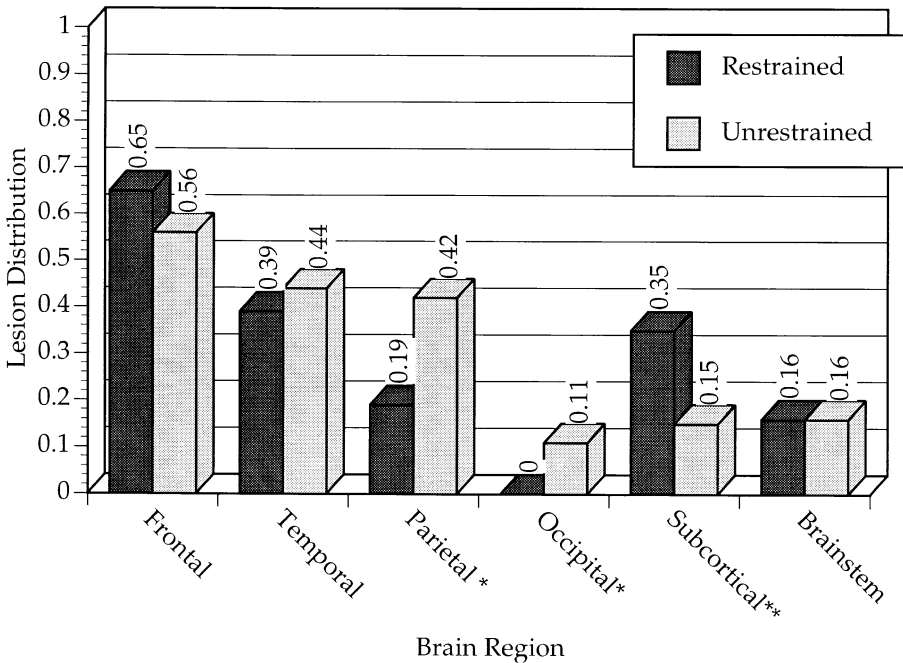

Fig. 1. Lesion distribution among restrained and unrestrained groups. Brain regions indicate lesions apparent within the extent of frontal, temporal, parietal, or occipital cortical areas and subcortical and brainstem regions. Subcortical territory included the basal ganglia, corpus callosum, internal capsule, thalamus, and hypothalamus. Brainstem regions included the midbrain, pons, and medulla. Significant findings are indicated by *($p \leq 0.05$), **($p \leq 0.01$), as noted on the brain region label.

Unrestrained motor vehicle occupants were significantly more likely to sustain damage in posterior regions of the brain. Chi-square analysis revealed significantly higher incidence of lesions in the parietal lobe (42%) when compared to those who were restrained (19%) $[\chi^2(1) = 5.34, p = 0.02]$. Chi-square analysis also revealed significantly higher incidence of lesions in the occipital lobe among unrestrained occupants (19%) when compared to those who were restrained (0%) $[\chi^2(1) = 3.88, p = 0.05]$.

Restrained motor vehicle occupants, conversely, were significantly more likely to sustain damage in the subcortical region than unrestrained occupants (35% and 15%, respectively) $[\chi^2(1) = 6.74, p = 0.01]$.

Restrained and unrestrained individuals were equally likely to sustain observable frontal lobe lesions (65% and 56%, respectively) $[\chi^2(1) = 0.74, p = 0.39]$, temporal lobe lesions (39% and 44%, respectively) $[\chi^2(1) = 0.28, p = 0.60]$, and brainstem lesions (16% and 16%, respectively) $[\chi^2(1) = 0.001, p = 0.98]$. The lesion distribution for restrained and unrestrained motor vehicle occupants is depicted in Fig. 1.

2.4. Functional outcome

Analyses revealed no effect of seatbelt use on outcome measures. On the FIL, the data revealed non-significant differences between restrained ($M = 6.125$, SD = 2.35) and unrestrained ($M = 6.045$, SD = 2.33) TBI survivors at an average of 3 years following the injury, $t(132) = 0.151, p = 0.88$. Data analysis also revealed no significant difference in average record inferred DRS score for restrained occupants ($M = 6.72$, SD = 4.82) and unrestrained occupants ($M = 7.78$, SD = 6.58) at an average of 3 years following the injury, $t(112) = -0.65, p = 0.52$. Thus, the data did not reveal any relationship between seatbelt use and patient outcome.

3. Discussion

Contrary to our initial prediction, analyses of brain lesion location revealed significant between-group differences for posterior and subcortical regions. Occipital and parietal cortical lesions appeared more frequently in unrestrained motor vehicle occupants, while lesions in the basal ganglia, hypothalamus, thalamus, corpus callosum, and internal capsule were more prevalent in restrained motor vehicle occupants. There were no between-group differences in lesion incidence within frontal, temporal, and brain stem regions.

Seatbelts alter the body's response to the forces applied in a motor vehicle collision. One consequence is reduced displacement experienced by restrained occupants and limited opportunity for posterior skull obstacle contact. The greater physical displacement of unrestrained passengers may account for the increased incidence of posterior cortical lesions in this group. These explanations were supported by the current data; no restrained individuals in this study sustained occipital lobe lesions.

A second finding revealed that the frequency of subcortical lesions was greater in restrained passengers. We suspect that restrained occupants are subject to greater inertial

forces focused in the head and neck and may thus have an increased likelihood of sustaining subcortical injury. In support of these findings, McIntosh et al. (1996) emphasized that inertial and impact forces are distinct physical processes that may be responsible for different forms of brain injury. Future investigations should include comprehensive accident analyses, such as those conducted by Siegel et al. (1993) and Varney and Varney (1995). Such efforts will promote a better understanding of the physical forces present at the time of injury in motor vehicle collisions.

Restrained and unrestrained motor vehicle occupants were equally likely to sustain frontal and temporal lobe lesions. During acceleration/deceleration injuries, the anterior and ventral surfaces of the frontal and temporal lobes often collide with the bony ridges in the skull (Whyte & Rosenthal, 1993; Adams et al., 1997). These brain regions appear to be particularly susceptible to bruising regardless of how the head is struck (Whyte & Rosenthal, 1993), perhaps explaining the absence of any between-group differences.

Consistent with Smith-Seemiller et al. (1997), we observed no between-group differences on traditional measures of injury severity (LOC, GCS, length of acute hospital stay). The brain injuries sustained in this investigation, however, appear more severe (mean GCS = 5.3, LOC = 3 to 5 weeks) than in other investigations (Smith-Seemiller et al., 1997; Varney & Varney, 1995).

Analysis of functional outcome scores revealed no significant between-group differences. There are two reasons that likely explain similar outcomes between groups. First, as noted, the participants in this investigation sustained primarily moderate to severe brain injuries; in such cases, the benefit provided by seatbelts may be greatly diminished. Second, observable frontal and temporal lobe lesions occurred in approximately 60% and 40% of the TBI survivors across both restrained and unrestrained groups, respectively. The behavioral and emotional correlates of frontal lobe insult can be particularly debilitating (Kay, 1986) and deficits in memory and attention, related to both frontal and temporal lobe injury, have been associated with high post-injury unemployment rates (Brooks et al., 1987).

Neuropsychological testing has revealed only minimal between-group discrepancies in cognitive performance, with some evidence for greater impairment in frontal lobe functioning among unrestrained individuals (Smith-Seemiller et al., 1997). This finding is somewhat surprising given the absence of differences in frontal and temporal lobe lesion rates between our groups. It is possible that neuropsychological differences between restrained and unrestrained groups, particularly for cognitive domains associated with parietal regions, were not tapped by Smith-Seemiller et al.'s abbreviated neuropsychological evaluation that measured predominantly executive functions and retention of material. Thus, a comprehensive evaluation that assesses subcortical and posterior cortical functions may, indeed, identify cognitive differences between these two groups. If differing neuropsychological profiles are noted, then rehabilitative protocols may be adjusted to suit the relevant cognitive deficits associated with each of these groups.

A number of limitations of the present study are acknowledged. First, the absence of significant differences between groups for some brain regions, such as the frontal lobe, and for functional outcome could be attributed to low statistical power. Because relatively

few subjects in this sample used a seatbelt, it was not possible to use power analysis to specify a priori the number of subjects to be used in this investigation. Instead, we selected a traditional alpha level of 0.05 and conservatively expected a small to medium effect size of 0.20. With these constraints in place, the power of the study approaches the satisfactory range at 0.72 (Cohen, 1988, 1992).

Second, our sample comprises a unique portion of the population of motor vehicle accident survivors. In particular, those individuals in our restrained group represent a small segment of the population of moderately to severely brain injured individuals. It is possible that our restrained group experienced a more extreme level of accident severity than did those in our unrestrained group. Because no between-group differences in injury severity were elicited in our sample, it may be posited that collision factors were necessarily greater in the restrained group. Collision variables such as speed, direction, and type of impact, are not available in most patient records and were not gathered in this investigation. Although these two groups may represent different levels of accident severity, we contend that our sample is representative of restrained and unrestrained individuals who sustain moderate to severe brain injury. Indeed, our sample was drawn from one of the largest TBI databases in Pennsylvania, and we have no reason to suspect that enrollment in the rehabilitation program was influenced by whether or not the individual was using a seatbelt at the time of the accident. It is also unlikely that the distribution of lesions in the restrained group (i.e., more frequent subcortical injuries but fewer posterior injuries), in comparison to the unrestrained group, can be explained solely by differences in accident severity.

It is important to emphasize that seatbelts are an effective safety device, particularly for mild collisions. As noted above, there was a large discrepancy in the number of restrained and unrestrained TBI survivors in this sample, with only 19% of participants using a seatbelt at the time of injury. This frequency of seatbelt use is clearly disproportionate to national seatbelt use averages (68% in 1996; Federal Register, 1997) and suggests that brain injury is most predominant in collisions where restraint systems are not used. Put simply, unrestrained drivers are more likely to obtain moderate to severe TBI. The most likely explanation for this discrepancy is that unrestrained occupants may be more severely injured in a less severe accident. However, other behavioral, social, and demographic factors, including risk taking, age, and alcohol use are also likely to play a prominent role in the occurrence of moderate to severe TBI. This is supported by the demographic data for this investigation; our unrestrained group was younger, less educated, and more likely to be using alcohol at the time of the accident than the restrained group.

In sum, our findings show that seatbelt use differentially affects the anatomical location of brain lesions suffered by automobile occupants who have sustained moderate to severe brain injury. Future investigations that employ measures of collision variables conducted in conjunction with patient follow-up may better delineate the relationship between seatbelts, brain injury, and outcome. Ultimately, with a comprehensive understanding of how seatbelts affect incidence, severity, and lesion location in TBI, a new generation of motor vehicle safety systems may provide even greater protection against brain injury.

References

Adams, R. D., Victor, M., & Ropper, A. H. (1997). *Principles of Neurology* (6th edn.). New York: McGraw-Hill.

Brooks, N., McKinlay, W., Symington, C., Beattie, A., & Campsie, L. (1987). Return to work within the first seven years after head injury. *Brain Inj 1*, 5–19.

Campbell, B. J. (1987). Safety belt injury reduction related to crash severity and front seat position. *J Trauma 27*, 733–739.

Cohen, J. (1988). *Statistical Power Analysis for the Behavioral Sciences* (2nd edn.). San Diego, CA: Academic Press.

Cohen, J. (1992). A power primer. *Psychol Bull 112*, 155–159.

Derosa, D., & Larsonneur, J. F. (1984). Seatbelt improvements, Regie Renault Technical Center-France, Society of Automotive Engineers, 840400, March, 287–298.

Engberg, A. (1995). Severe traumatic brain injury—epidemiology, external causes, prevention, and rehabilitation of mental physical sequelae. *Acta Neurol Scand 92*(164), 127–134.

Federal Register (1997). Presidential initiative for increasing seatbelt use nationwide: recommendations from the Secretary of Transportation. *Pres Doc 62*(75).

Frankowski, R. F. (1986). Descriptive epidemiological studies of head injury in the United States: 1974–1984. *Adv Psychosom Med 16*, 153–172.

Gouvier, W. D., Blanton, P. D., LaPorte, K. K., & Nepomuceno, C. (1987). Reliability and validity of the disability rating scale and the levels of cognitive functioning scale in monitoring recovery from severe head injury. *Arch Phys Med Rehabil 68*, 94–97.

Hall, K., Cope, N., & Rappaport, M. (1985). Glasgow outcome scale and disability rating scale: comparative usefulness following recovery in traumatic head injury. *Arch Phys Med Rehabil 66*, 35–37.

Katz, D. I., & Alexander, M. P. (1994). Traumatic brain injury: predicting course of recovery and outcome for patients admitted to rehabilitation. *Arch Neurol 51*, 661–670.

Kay, T. (1986). Minor head injury: an introduction for professionals. Head Trauma Research Project, New York University Medical Center. National Head Injury Foundation, 1–12.

Kraus, J. F., & McArthur, D. L. (1996). Epidemiologic aspects of brain injury. *Neuroepidemiology 14*, 435–450.

Loo, G. T., Siegel, J. H., Dischinger, P. C., Rixen, D., Burgess, A., Addis, M. O., Quinn, T., McCammon, L., Schmidhauser, C. B., Marsh, P., Hodge, P., & Bents, F. (1997). Airbag protection versus compartment intrusion effect determines the pattern of injuries in multiple trauma motor vehicle crashes. *Trauma: Inj Infect Crit Care 41*(6), 935–950.

McIntosh, T. K., Smith, D. H., Meaney, D. F., Kotapka, M. J., Gennarelli, T. A., & Graham, M. J. (1996). Neuropathological sequelae of traumatic brain injury: relationship to neurochemical and biomechanical mechanisms. *Lab Invest 74*, 315–342.

Molitor, A. J. (1990). Pennsylvania's head injury services. *Health Rep 11*, 1–2.

Rappaport, M., Hall, K. M., Hopkins, K., Belleza, T., & Cope, D. N. (1982). Disability rating scale for severe head trauma: coma to community. *Arch Phys Med Rehabil 63*, 118–123.

Schatz, P. (1995). *Predicting level of independence following moderate and severe traumatic brain injury.* Unpublished doctoral dissertation, Drexel University, Philadelphia.

Schatz, P., & Chute, D. L. (1995). Predicting level of independence following moderate and severe traumatic brain injury. *Arch Clin Neuropsychol 11*, 444–445.

Siegel, J. H., Mason-Gonzalez, S., Dischinger, P., Cushing, B., Read, K., Robinson, R., Smialek, J., Heatfield, B., Hill, W., Bents, F., Jackson, J., Livingston, D., & Clark, C. C. (1993). Safety belt restraints and compartment intrusions in frontal and lateral motor vehicle crashes: mechanisms of injuries, complications, and acute care costs. *J Trauma 5*, 736–758.

Smith-Seemiller, L., Lovell, M. R., Franzen, M. D., Smith, S. S., & Townsend, R. N. (1997). Neuropsychological function in restrained versus unrestrained motor vehicle occupants who suffer closed head injury. *Brain Inj 11*, 743–750.

Swierzewski, M. J., Feliciano, D. V., Lillis, R. P., Illig, K. A., & States, J. D. (1994). Deaths from motor vehicle crashes: patterns of injury in restrained and unrestrained victims. *J Trauma 37*, 404–407.

Teasdale, G., & Jennett, B. (1974). Assessment of coma and impaired consciousness: a practical scale. *Lancet 2*, 81–84.

Varney, N. R., & Varney, R. N. (1995). Brain injury without head injury: some physics of automobile collision with particular reference to brain injuries occurring without physical head trauma. *Appl Neuropsychol 2*, 47–62.

Whyte, J., & Rosenthal, M. (1993). Rehabilitation of the patient with traumatic brain injury. In J. B. DeLisa (Ed.), *Rehabilitation Medicine: Principles and Practice*. (pp. 825–860). Philadelphia: Lippincott.