

THE COMPLEX WEB OF CAUSATION:

MOTOR VEHICLE ACCIDENTS, COMORBIDITY AND PTSD

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COMORBIDITY, CAUSALITY AND THE CONFLICT OF MODELS

An apparently straightforward event occurs when a man swerves to avoid hitting an oncoming car, loses control of his vehicle on an icy road and runs into a tree. The cause is a dangerous patch of highway, the effect is a car accident. But that is about where the simple chain of causality ends. To understand the immediate and long-term impact of the accident on the victim we have to envision a web of causality that embraces multilayered, proximal and distal mechanisms of influence (Krieger, 1994).

The complex and interactive nature of traumatization is reflected in the ways in which clinicians have attempted to define it. Lenore Terr, after studying children who were involved in a mass kidnapping observed that *“Psychic trauma’ occurs when a sudden, unexpected, overwhelmingly intense emotional blow or a series of blows assaults the person from outside. Traumatic events are external, but they quickly become incorporated into the mind”* (Terr, 1990, p.8). Van der Kolk (1989) notes that *“traumatization occurs when both internal and external resources are inadequate to cope with external threat”* (p.393). Both definitions inextricably connect the internal and the external, the personal with the social, the event with the personal interpretation of the event. Clinicians can be quite comfortable with this kind of causal complexity in dealing with a wounded patient because what actually caused the symptoms is less important than treating those symptoms in service of improving the victim’s level of function. But the issues of co-existing

disorders, pre-existing conditions, and causally related disorders are some of the unhappy points of contact, and often conflict, between men and women of medicine and practitioners of the law.

The requirements of the physician are radically different from those of the lawyer. For him or her, placing blame is largely irrelevant and managing the complex and often tragic experiences of human beings is all that matters. The patient is inevitably connected to his past and everything that has ever happened to him. He is comprised of a vast network of interconnected and reverberating experience, as mind impacts body, body alters mind, past influences present, and all is influenced by biological constitution, psychological interpretations of reality, on-going social interactions and the attribution of meaning acquired through the experience of the individual and the social group. Within the confines of such a reality, how can a physician reasonably answer questions of cause and effect without distorting the perceptions of this reality?

This conflict surfaces with enduring frequency around the issue of motor vehicle accidents, psychological injury, and compensation. In the theater of the courtroom, the rules of the game inform the players that they must embrace and administer notions of culpability and restitution. If someone can be found to be at fault for causing the disability or distress of another person, then the victim has a right to fair compensation. Expert testimony of physicians is required around the issue of causality and courts usually are looking at “proximate cause” or a “natural and probable cause and effect relationship”. But in the law, proximate cause does not necessarily mean proximate in time, and therefore there is much that is open to interpretation (Simon, 1995, p. xxiii). Unlike most other psychiatric labels, a diagnosis of PTSD implies causation and therefore post-traumatic stress disorder has been dubbed the “black hole” of litigation, appearing to present the possibility of easily attributing causality while in practice providing a slippery slope down into a pit of medical and legal confusion.

As our evidence-based understanding accumulates about the complex web of causation that characterizes the aftermath of a traumatic event, physicians and other mental health experts are often unable to give the simplistic cause and effect attributions that the legal system requires. If a person loses an arm in an accident, we can safely say that the severing of the major vessels and tissues connecting the arm to the body caused the amputation and that this severing occurred as a result of the shearing of the car door. But when the question is more subtle and involves not an organ or a body part, but an entire self and that self in a social and relational context, all we can safely discuss is before and after, accompanying and “comorbid” events, vulnerabilities and risk factors, sometimes to the point of absurdity, as far as the legal system is concerned.

In this chapter, I will review what we know thus far about what happens to people after many kinds of traumatic experiences and specifically, after vehicular accidents. We know that exposure to overwhelming stress has multiple effects, that there are risk factors, protective factors, cumulative factors, and possibly constitutional factors that influence how each individual responds to every unique event. But all we can do is speak in generalities. We cannot yet predict how any specific individual will be impacted by any traumatic event. What we can safely say is that exposure to trauma increases the likelihood that victims will suffer from complex and interactive physical, emotional, social, and spiritual/existential problems that ultimately may be diagnosed as medical or psychiatric problems. As we will see throughout this chapter, the subjects of post-traumatic stress, stress-related disorders and comorbid conditions can present a nightmarish situation to the legal system and tax medical experts.

THE COMPLICATED NATURE OF TRAUMA-RELATED DISORDERS

The connection between stress and mental disorder has long been recognized. In the late 70's. Brown and Harris demonstrated that about 60% of persons diagnosed as having a mental disorder had experienced a severely stressful life event in the two weeks preceding the onset of that disorder in comparison to 20% in the comparison group (Brown and Harris, 1978). In the months following a traumatic life event, there is a sixfold greater risk of suicide, a twofold greater risk of depressive disorders, and a slight increase in the risk of developing a schizophrenic disorder, according to Paykel's 1978 review. The connections between depressive disorders, panic disorders, phobic disorders were noted even before there was a diagnosis of "post-traumatic stress disorder" (Horowitz, 1993).

As the field of traumatic stress studies has grown, it has become clear that a variety of factors ultimately effect the outcome of a traumatic event. To obtain a clear picture it is necessary to look at many aspects of the entire system: individual vulnerability and risk factors that exist prior to the traumatic experience; the specific characteristics of the traumatic event; how the individual perceives those characteristics in reference to himself and his own history; the responses of the individual and the social environment during and after the traumatic event including the biological, psychological, social, and philosophical responses (McFarlane and Yehuda, 1996; Shalev, 1996). Shalev has argued convincingly that PTSD represents a "biopsychosocial trap" in which neurobiological processes specific to traumatic stress combine with the acquisition of fear-conditioned responses to trauma-related

stimuli, altered cognitive schemata and social apprehension to produce a vicious cycle of progressive deterioration (Shalev, 1996).

PREVALENCE OF PTSD

In America, where most of the community-wide epidemiological data has been gathered thus far, most individuals will experience a traumatic event sometime in their lifetime. According to population studies of trauma and PTSD, about a quarter of individuals who are exposed to a DSM-IV type stressor go on to develop PTSD, although there is variation depending on the type of stressor. By recent estimates, 5 % of men and 10-12 % of women will suffer from PTSD sometime in their lives, and for victims of traumas such as rape, the rate may be as high as 60 percent to 80 percent. For at least a third of sufferers, PTSD is a persistent condition lasting many years and over 80 % of persons with PTSD suffer from other psychiatric disorders (Solomon and Davidson, 1997). For motor vehicle accidents specifically, Breslau et al.'s (1991) study indicates that about 10% of a sample of young urban adults had suffered a serious road accident and 12% of them went on to develop PTSD. Norris's (1992) study is consistent with this PTSD rate in that of 1000 adults living in the southeast U.S., 25% had experienced a traffic accident and about 12% developed PTSD.

A formal diagnosis of PTSD is not the only diagnosis that can result from a traumatic stressor. Davidson has pointed out that there are at least ten other trauma-related disorders found in DSM-III-R including brief reactive psychosis, dissociative identity disorder, dissociative fugue, dissociative amnesia, conversion disorder, borderline personality disorder, depersonalization disorder, somatization disorder, dream anxiety disorder, and antisocial personality disorder (1993). Additionally, it is clinically well established that survivors of prolonged and repeated trauma develop a syndrome that has been termed "complex post-traumatic stress disorder" (Herman, 1992) or "disorders of extreme stress not otherwise specified – DESNOS - reflecting the multidimensional developmental impact on the entire being of this kind of stress (van der Kolk, 1996).

In 1993, Mayou, Bryant and Duthrie published a study that looked at the psychiatric consequences of road traffic accidents in 188 consecutive road accident victims admitted to an emergency department of the main hospital in Oxford, England. Almost one fifth of the victims suffered from acute stress syndrome with mood disturbance and terrifying memories of the accident. Anxiety and depression usually improved over the twelve months of follow-up, but a tenth of the patients still had mood disorders at one year. But specific post-traumatic symptoms were also common with ten percent of the patients suffering from PTSD and many suffering from phobic travel anxiety that was frequently disabling. According to these researchers, post-traumatic

syndromes were not associated with premorbid neurosis but were strongly associated with intrusive memories of the accident that did not occur in patients who had been unconscious or were amnesic for the accident. The victims' mental state at three months was highly predictive of their mental state at one year.

In the same year, Brom, Kleber and Hofman (1993) published another study with similar findings, reporting that 10% of a Dutch cohort of accident victims suffered from PTSD at one month and six months after the accident. In a Swedish study, 84 persons injured in traffic accidents and with moderate to severe injuries were interviewed approximately two years after the accident. Sixty-eight percent of the victims reported that they still suffered from physical sequelae and fifty-seven percent had been or were still suffering from psychological distress after the accident. Sixty-three percent of the accident victims were on the sick list for at least three months as a result of the accident while fifty-eight percent needed some type of handicap aid for some period of time after the accident. For twenty-nine percent of those employed in professions, the traffic accident led to altered working conditions (Andersson, Dahlback and Allebeck, 1994). Green et al. (1993) reported a PTSD rate of 27% in a small series of hospitalized accident victims in Australia. In a report of a severe bus accident in Australia that killed eleven people, 29 survivors were studied (Watts, 1995). Forty-one percent had PTSD and 52% reported severe intrusive or avoidance phenomena, including 31% who had both. These sequelae were associated with being currently distressed by another event and seeing bodies or witnessing the death of someone. Bryant (1997) reported on a series of 1,100 accident survivors in a one-year period in an emergency room in England. A preliminary analysis of the first six months' data indicated that about one in six of those not injured were still have difficulty a year later with work and/or daily tasks and one in five rated the quality of their life as worse than before the accident.

Blanchard and his colleagues at the University of Albany have been researching the effects of motor vehicle accidents and have published a series of articles. A study published in 1994 found that 46% of victims had PTSD. Another 20% had a subsyndromal version of PTSD characterized by the presence of the reexperiencing symptom cluster plus either the avoidance/numbing cluster or the over-arousal cluster of PTSD. In 1995, they reported on 158 victims who sought medical attention after an accident and were assessed for acute psychiatric and psychosocial consequences as well as pre-existing psychopathology at 1-4 months after the accident. They found that 39.2% met criteria for PTSD by DSM-III-R criteria. Those with PTSD were more subjectively distressed and had more impairment in role function.

DiGallo, Barton and Parry-Jones (1997) looked prospectively at the early psychological consequences of traffic accidents in fifty-seven children and

adolescents aged 5-18 years. The children and their parents were interviewed 2-16 days after the accident and then again 12-15 weeks later. Although post-accident stress symptoms had decreased by the second interview, 14% still suffered from moderate or severe PTSD, 17% from serious traffic-related fears and the parents reported increased mood disturbances in their children. Tyano and colleagues (1996) did a seven-year follow-up of child survivors of a bus-train collision and found that high exposure correlated with the highest levels of somatization, depression, phobic anxiety, psychoticism, and more PTSD symptoms. Symptoms of acute stress, and manifestations of fear immediately after the accident were strongly related to long-term maladjustment.

CAUSATIVE VULNERABILITIES AND RISK FACTORS.

How vulnerable was the victim to the long-term effects of trauma before the vehicular accident occurred? That is a fundamental question that arises in the courtroom, in the physician's office, and in the workplace. The question of whether or not the accident caused or was caused by a comorbid condition is a pressing one, but is often very difficult to resolve.

In the first place, there are many physical and behavioural factors that make it more likely that accidents will occur. Davidson et al.'s (1985) early work on the relationship between family psychopathology and chronic PTSD may indicate some genetic risk factors. True et al (1993) have demonstrated that genetic vulnerability had a significant impact on symptom liability among a cohort of combat veterans who were twins. It is possible that intrinsic or acquired problems with vital neurohormonal and neurostructural systems may contribute to increased risk for the later development of severe post-traumatic symptomatology after an accident. Recent studies of hippocampal volume have demonstrated a decreased left hippocampal volume in combat veterans and in victims of child abuse (Bremner et al, 1997). Many studies demonstrate that exposure to trauma impacts the HPA-axis, a finding now noted in combat veterans and victims of child abuse (Stein et al, 1997). In addition, medical conditions like certain cardiac disorders, uncontrolled diabetes, cerebrovascular insufficiency, and epilepsy can all increase the likelihood that a motor vehicle accident may occur. Illegal drugs and alcohol are notable in their ability to compromise driving skills, but so too do many prescription drugs. People who suffer from personality disorders that are frequently related to childhood exposure to trauma, often engage in behaviors that put their lives at risk, including deliberate self-harm behaviors and suicide gestures.

Studies support a growing number of other risk factors in the development of PTSD. For instance, in the National Comorbidity Study (Bromet et al, 1998), a risk factor for women was having a history of affective disorder while for men it was history of anxiety disorder and having a parent with a mental disorder. Harsh and abusive childhood environments, as well as other kinds of previous traumatic experiences,

increase the risk for PTSD after a later traumatic experience and for many other kinds of comorbid psychological and physical conditions (Bremner et al, 1993; Green et al., 1990a; Kulka et al., 1990; Zaidi and Foy, 1994). Child abuse also increase the risk for many different kinds of personality disorders that then can impact on the way the individual deals with a subsequent traumatic event like an automobile accident. Previous experiences also determine the way an individual interprets their place in the world and how they see themselves in relation to others. The characteristic style and attitudes that people have used in the past to cope with stressful events will influence the way they interpret a recent traumatic event like a vehicular accident (McFarlane and Yehuda, 1996).

A previous history of trauma also may have a cumulative effect that impacts on a recent traumatic event. It is a well-recognized clinical phenomenon that a previous history of trauma appears to exacerbate the effects of a recent trauma, producing the effect of “the straw that broke the camel’s back”. This topic has been evaluated in several studies related to vehicular accidents. Blanchard et al. (1994) reported that 74% of the PTSD group had a previous trauma and 52% had experienced a previous serious motor vehicle accident. In the subsyndromal group, 90% of them had been in a previous serious accident and 80% had an other previous trauma. Combining these, 87% of the PTSD group and 100% of the sub-syndromal group admitted to a history of previous trauma. In the Blanchard et al. 1995 study, the rate of prior PTSD among the MVA victims who developed either full PTSD or subsyndromal PTSD was 22.4% compared to 3.9% in those who were relatively unaffected. Winje (1996) looked at the relationship between previous trauma and the subsequent reactions to a bus accident. Prior exposure to trauma did not affect the scores on the Impact of Events Scale but was associated with a sustained vulnerability to general psychological distress as measured by the SCL-90. DiGallo and Parry-Jones (1996) also point out the importance of further study relating to pre-accident disposition and vulnerability as it relates to post-accident morbidity.

A powerful vulnerability appears to be the presence of pre-existing psychiatric disorders. In a study by Mayou, Bryant and Duthrie (1993) there was an association between post-traumatic emotional disorders and having pre-accident psychological or social problems. There was also an association between emotional problems and continuing medical complications. In the Blanchard et al 1994 study, of the PTSD group, 48% also met criteria for major depressive disorder and of the PTSD group, 39% had experienced a previous major depression. Of those with PTSD and major depression, 64% had a history of previous depression and in 82% of them, this present depressive episode had begun after the accident. The authors conclude that, *“previous depression seems to predispose the formerly depressed individual to develop PTSD when involved in a serious MVA”* (Blanchard et al, 1994, p.289).

In their 1995 study, Blanchard et al. found that of the PTSD group, 53% also met criteria for major depression, with most of that (81.8%) developing after the accident. Fifty percent of the motor vehicle accident patients with PTSD had a

history of previous major depression as compared with 23% of the non-PTSD patients. The MVA-PTSD group had a higher rate of lifetime panic disorder (11.3%) and any anxiety disorder (29%). Personality disorders were also common in this group with 13.3% meeting criteria for at least one disorder compared to 8.6% of controls. Of the victims who were in the midst of a major depression when the accident occurred, 75% developed PTSD, although their symptoms of PTSD were no more severe than those who did not have a history of depression. There was a similar relationship between PTSD and panic disorder. Those victims who developed PTSD were more likely to have a history of a panic disorder (11.3%). The MVA victims were also more likely to have a lifetime history of drug dependence – 13.9% vs. 2.1%.

Chubb and Bisson (1996) looked at how patients who already suffered from chronic mental illness fared after a major coach accident. Fifty percent of them had PTSD and the extent of psychological suffering was the most marked in those who suffered from previous depression and anxiety disorders. This effect, however, does not apparently hold for all populations. Resnick and colleagues (1992) studied crime victims and did not find an association between psychiatric diagnoses before the crime and PTSD after the crime except when the person had a prior depression and then was exposed to a high crime stress.

CHARACTERISTICS OF THE EVENT.

Although the diagnosis of PTSD requires exposure to an event that involved actual or threatened death or serious injury or a threat to the physical integrity of self or others, some studies have shown that “ordinary” stressors can produce PTSD – stressors like marital disruptions, illegal activities of one’s children, death of a loved one, money problems, injury (Burstein, 1985; Helzer et al, 1987; Solomon and Caninco, 1990). Green et al.(1990a), reviewed studies up to that point to evaluate the aspects of a traumatic experience that are the most overwhelming, and therefore traumatogenic for the individual. These included: threat to one’s life and body integrity; severe physical harm or injury; receipt of intentional injury/harm; exposure to the grotesque; witnessing or learning of violence to loved ones; learning of exposure to noxious agent; causing death or severe harm to another. Involvement in serious motor vehicle accidents can expose victims to combinations of these stressors, even if the subject escapes the accident physically unharmed. The suddenness of the event, the unexpected nature of the trauma, the intentions of the perpetrator, how long it lasts, how much damage it does to the self and to others, the degree of life threat, the degree of associated humiliation and shame, in addition to actual injury, will all play a role in determining how the traumatic event will impact on the individual.

Later resilience or pathology may also be predicted by how the person deals with the traumatic event itself. Dissociation is commonly associated with traumatic events and can be a protective coping skill, but leads to fragmentation. There is a

growing body of evidence to support the clinical impression that dissociation in the face of trauma represents different kinds of physiological reactivity at the time of the traumatic event, may occur more often in those who have experienced prior victimization and is a marker of long-term psychopathology (Bremner and Brett,1997; Cloitre, Scarvalone and Difede,1997; Griffin, Resick and Mechanic,1997). Impaired cognition, panic and freezing at the time of the event can also lead to the perception of impaired ability to cope, shame, guilt and a generalized feeling of ineffectiveness.

Lifton has talked about the “death imprint” that is associated with exposure to death and dying at the time of the traumatic event. He describes this as the radical intrusion of an image or feeling of threat or end to life. The degree of anxiety associated with the death imprint has to do with the inability to assimilate the experiences. The image remains, haunting the victim as he or she struggles to master and integrate the experience (Lifton, 1993). Victims of automobile accidents frequently dissociate at the time of the crash and yet the experience of near-death may leave them feeling fragmented and subject to intrusive experiences of the close call. Clinicians who have worked with accident victims have noticed that victims may persist, consciously or unconsciously, in the feeling that they died during the crash and for treatment to be successful the experience of having died must be integrated with the reality of having survived (Bills, 1998).

The way in which the individual copes with the trauma may also impact on how successful subsequent coping is. Self-efficacy, the term used to describe our belief about what we are capable of doing in any given situation, is influenced by experience and is put to the test when we are caught in dangerous situations like the ones presented at the time of a motor vehicle accident (Bandura, 1982).

Disaster researchers have described the personal devastation that confronts the trauma survivor who loses important aspects of his or her community or social support system. Survivors of motor vehicle accidents in which friends or relatives have been killed lose people in their lives who may provide an important sense of well-being and identity. To the extent that people cannot return to work or to their normal activities, their sense of being a part of a community may also be jeopardized.

POST-TRAUMA RESPONSES.

After the trauma, many normal functions may be radically altered. Memory problems are quite common after a trauma, as is persistent hyperarousal, a tendency to avoid stimuli that reminds the person of the traumatic event, and intrusive phenomena. Physiological hyperarousal and intrusions can dramatically impact on normal cognition and dysregulate affect. The victim’s view of him or herself as a competent adult may be drastically altered and attitudes towards self or others may be radically changed. If others have been killed in the trauma, survivors may experience survivor guilt, an apparently paradoxical feeling of self-blame for

having survived when others have not. Lifton (1993) has described a phenomenon he calls “failed enactment”, the extreme sense of helplessness one experiences at the time of a trauma, and a profound sense of ineffectiveness or inadequate coping resulting from the inability to have prevented the trauma from happening.

Complicating this even further is the issue of social support after a trauma. Social support has repeatedly been found to be a critical component of recovery after trauma or the lack of such recovery. In one study, less psychological support post-trauma was predictive of PTSD whereas more severe injury did not predict PTSD (Perry et al., 1992). Andersson, Bunketorp and Allebeck (1997) have reported on the high rate of psychosocial complications after road traffic injuries and poor social support is one of the risk factors for such complications. When Buckley, Blanchard, and Hickling (1996) compared victims of MVA’s who developed delayed onset PTSD during a 1-year follow-up interval, MVA victim controls who did not develop PTSD, and a group who met criteria for acute onset PTSD, the delayed onset participants had poorer social support than the controls prior to and after the MVA. Landsman and colleagues (1990) assessed a group of urban trauma victims 3-39 months after their injuries and found that the levels of psychological distress were predicted less by injury severity than by subjective impact of the accident, injury-related financial and employment problems, and family and social environment. Family environment ratings were significantly worse for subjects with elevated levels of psychiatric symptoms.

Rejection, abandonment, blaming on the part of supportive others may lead to a “second injury” (Symonds, 1982), further compounding the effect of the trauma. Accident victims may have post-accident experiences with rescue workers, or hospital personnel that compound the traumatic insult and the hospital itself may be a traumatizing place. The accompanying “sanctuary trauma” is that which occurs when an individual who has suffered a severe stressor next encounters what was expected to be a supportive and protective environment and discovers only more trauma (Silver, 1986).

Finally, traumatic experience, particularly that suffered at the hands of others, shatters the fundamental assumptions upon which we build our sense of reality and safety. We live our day-to-day lives functioning with certain basic beliefs that we simply take for granted – that we are safe, that the world makes sense, that other people can be trusted, that the past is known and the future is predictable (Janoff-Bulman, 1992). Despite newspapers and television reports to the contrary, we don’t really believe that tragedy is going to strike us until it happens. But interpersonal trauma can strip us of the certain ground beneath our feet so that for the victim, there is no longer any safety, the world does not make sense, people cannot be trusted, parts of the past or forgotten or erased, and the future is neither predictable nor necessarily benevolent. All of these factors will interact in unpredictable ways with intrinsic vulnerabilities to produce the complicated picture of comorbidity. Solomon, Iancu and Tyano (1997) studied the effects of a bus-train

collision on the beliefs and cognitions of 389 young adult survivors, 7 years following the accident. At the time of the accident the survivors had all been adolescents. Exposure to the traumatic accident challenged the survivors' beliefs in a benevolent and a just world. The extent of this change in basic assumptions had a direct relationship to the level of exposure to traumatic stimuli. Changes in these basic cognitive schemata were associated with psychiatric symptoms and problems in functioning.

PSYCHIATRIC COMORBIDITY AND PTSD

There is now an abundance of research indicating that there is significant overlap between PTSD and other psychiatric conditions. Some of those premorbid conditions occur immediately after or seemingly along with the PTSD, as in major depression. Others, as in many of the apparently related personality disorders like borderline personality disorder, appear to be a product of the entwined interaction of post-traumatic symptoms, constitutional factors, existing personality, and family environment all interacting with the process of development. Without such a connecting link, many physicians will fail to recognize that many apparently "far-flung" psychological and physical problems may in fact be related to a past history of a traumatic experience, including vehicular accidents. It is this overlap and complexity that makes the discussion of comorbidity and causality so treacherous.

Green and her colleagues (1992) reviewed studies of Vietnam veterans and over three-quarters of the patients with PTSD also met criteria for at least one other diagnosis, most commonly major depression, substance abuse, and antisocial personality disorder. Even in the most conservative study, those with PTSD were two to four times more likely than those without PTSD to have virtually any other psychiatric disorder, particularly somatization (Solomon and Davidson, 1997). According to one study, somatization was found to be 90 times more likely to occur in those with PTSD than in those without PTSD (Davidson et al., 1991). In a study by Breslau and colleagues (1991), those with PTSD were more than six times as likely to have some other psychiatric disorder. The large epidemiological study of Kessler and colleagues (1995) showed that those with PTSD are almost eight times as likely to have three or more disorders – 88% of men and 79% of women with PTSD had a history of at least one other disorder.

Patients with one or more symptoms of PTSD are more likely than those without any mental disorder to experience poor social support, marital difficulties, and occupational problems, as well as more impairment on income and disability measures than even those with major depressive disorder. The people with PTSD symptoms are also more likely to have a number of chronic illnesses, consistent with many other studies of specific trauma groups. Although these patients have a disproportionate utilisation of the health care system, they are reluctant to seek

mental health treatment, a finding that has been seen in many other studies as well (Solomon and Davidson, 1997).

In the Kessler study mentioned earlier, men with PTSD were six to ten times more likely and women four to five times more likely to have affective disorders than those without PTSD. Similar figures appear with anxiety disorders with men three to seven times more likely and women two to four times more likely to have another anxiety disorder along with their PTSD. People with PTSD were also two to three times more likely to have a substance abuse disorder. In a number of studies showing the relationship between PTSD and substance abuse, between 25% and 58% of those seeking substance abuse treatment also were comorbid for PTSD (Grady, 1997).

Research reports connecting PTSD with other psychiatric conditions is growing exponentially. There is such a large literature on the connection between trauma and dissociative disorders that the latter are generally considered to be trauma-related disorders. Suggestions have even been made that it is not especially helpful to define the clinical significance of a given person's posttraumatic disturbance in terms of meeting the criteria for a specific DSM-IV stress category because the symptoms are determined by such a wide range of factors, far beyond the description of the stressor itself (Briere, 1997).

Orsillo et al. (1996) found that relative to veterans without PTSD, veterans with PTSD had significantly higher rates of current major depression, bipolar disorder, panic disorder, and social phobia, as well as significantly higher rates of lifetime major depression, panic disorder, social phobia, and obsessive-compulsive disorder. Rogers et al. (1996) studied the prevalence and characteristics of somatoform disorders among 654 subjects with anxiety disorders - 5.5% of the subjects had past or current somatoform disorders. The subjects with somatoform disorders were significantly more likely to have histories of PTSD, lending support to the observation that somatoform disorders are frequently comorbid with PTSD, other anxiety disorders and depressive disorders. Gleason (1993) Orsillo et al. (1996), and Saunders et al. (1992) all have noted the connection between PTSD and obsessive compulsive disorder in several different populations. Bower and Stein looked at civilian trauma and reported on the increased incidence of panic disorder among those exposed to traumatic suffocation. In a study done by Falsetti and Resnick (1997), 69% of a population seeking treatment for trauma-related problems had panic attacks. This association between panic disorder, other anxiety disorders and PTSD has also been noted by Orsillo et al. (1996); Fierman, (1993); Vasile et al. (1997) and others.

Another important aspect of comorbidity between PTSD and other psychiatric disorders is the established connection between trauma and personality disorders. Van der Kolk (1996) points out that the combination of chronic dissociation, physical problems for which no medical cause can be found, and a lack of adequate ability to self-regulate can have profound effect on the ways in which personality develops.

Many authors have commented upon the ways early childhood exposure to trauma and loss can skew character formation. The connection between borderline personality disorder and trauma, particularly childhood trauma has been studied by a number of authors (Ellason et al., 1996; Herman, Perry and Van der Kolk, 1989; Perry et al., 1990; Sabo, 1997). What is perhaps even more startling is the recognition that character can be changed even after adulthood. Southwick, Yehuda and Giller (1993) concluded from their study of treatment-seeking Vietnam veterans that war-related PTSD is often accompanied by diffuse, debilitating, and enduring impairments in character that were not present before combat exposure.

MEDICAL COMORBIDITY AND TRAUMA-RELATED DISORDER

Victims of traumatic stress are at risk for many comorbid health problems as well as psychological problems. McFarlane and Yehuda (1996) point out that there are three ways that trauma victims may manifest physical problems: as a direct result of the traumatic experience, as an integral part of the post-traumatic stress disorder, or as a nonspecific response to trauma exposure independent of the PTSD. A great deal more research will need to be conducted before we are able to definitively differentiate among these hypotheses.

Much of our present knowledge regarding the profound connections between environmental events and body physiology derives from the field of “stress studies”. As the field of psychoneuroimmunology expands, there is a growing body of information about the relationship between stress, and the immune system that may have serious implications for the study of traumatic stress as well. So far, we know that even mild stress impacts on the immune system (Bachen et al., 1992; Brosschot et al., 1994). In a review article from 1993, Herbert and Cohen assert that there is evidence for a relationship in humans between stress and decreases in functional immune measures. Stress effects the numbers and percentage of circulating white blood cells, immunoglobulin levels, and antibody titers to herpes viruses (Herbert and Cohen, 1993).

Subsequent analyses suggest that objective stressful events are related to larger immune changes than subjective self-reports of stress, that immune response varies with stressor duration, and that interpersonal events are related to different immune outcomes than non-social events. According to an even more recent review by Cohen and Herbert (1996), there is substantial evidence that factors such as stress, negative affect, clinical depression, social support, and repression/denial can influence both cellular and humoral indicators of immune status and function. And, at least in the case of the less serious infectious diseases (colds, influenza, herpes), there is consistent and convincing evidence of links between stress and negative affect and disease onset and progression.

Mood states, compared to a neutral condition, affect the immune system, with some differences between positive and negative moods (Futterman et al, 1994; Kiecolt-Glaser et al., 1993). In two related studies, one of children in day care and another of children entering kindergarten, the development of respiratory illnesses was found to be related to stressful life events (Boyce et al., 1995). Another group looked at conduct disordered, depressed, and normal adolescents and found out that there was a correlation between significant negative life events and lowered natural killer cell activity (Birmaher, 1994). In primates, there is a large body of evidence that disruptions in social relationships have many immunological sequelae, particularly in the young monkey. There is evidence in infant monkeys that normal maternal care is important for the development and maintenance of normal immune function. The immune response of adult monkeys are also affected by aggression within the group (Coe, 1993).

Social support – or the lack of it – has emerged repeatedly in studies of traumatic stress as an important determinant of morbidity. In another group of studies outside of the traumatic stress field, investigators showed that in humans, social support, under experimental conditions of stress, produced less cardiovascular reactivity than a lack of social support (Lepore et al., 1993). A group of Swedish researchers looked at the established connection between social support and mortality. They differentiated between emotional support from very close persons (attachment) and the support provided by an extended network of social connections (social integration). They found that both factors were lower in middle-aged men with coronary heart disease and that this also predicted new cardiovascular incidents (Orth-Gomer, Rosengren, and Wilhelmsen, 1993). Buckley, Blanchard and Hickling (1996) have noted that victims of automobile accidents who suffered from delayed onset PTSD had poorer social support before and after the accident. Social support may be a connecting point for many post-trauma physical and psychological adjustment problems.

Outside of the “stress” literature, most of the research connecting trauma-related disorders to health problems has used adult victims of child abuse as subjects. Clinical wisdom reports that the rate of medical comorbidity in this population appears to be quite high. This observation is supported by a number of studies.

Salmon and Calderbank (1996) looked at 275 British undergraduates and surveyed their history of sexual and physical abuse in childhood and their health care utilization, somatization, and hypochondriasis as an adult. Separate groups recalled physical and sexual abuses. Physical abuse predominating in males and sexual abuse in females. Both types of abuse were followed by a greater number of hospital admissions and surgical procedures, somatization, and hypochondriasis in adulthood.

Internists specializing in gastrointestinal disorders have been noticing the connection between chronic disorders and a past history of childhood abuse. One study looked

at 239 female patients presenting to a gastroenterology clinic. They found that 66.5% of the women had experienced sexual and/or sexual abuse and that the women with a sexual abuse history had more pain, other somatic symptoms, bed disability days, lifetime surgeries, and functional disabilities than those without sexual abuse. Women with physical abuse also had worse health outcome on most indicators, while rape and life threatening physical abuse seem to have worse health effects than less serious physical violence and milder forms of sexual abuse (Leserman, 1996). Fukudo and colleagues studied irritable bowel syndrome and saw that the IBS patients have an exaggerated responsivity of the gastrointestinal tract to mental stress (Fukudo, 1993).

Walker and colleagues looked at the comorbidity between chronic pelvic pain, irritable bowel syndrome, and a past history of abuse. They found that compared to women with irritable bowel syndrome alone, those with both irritable bowel syndrome and chronic pelvic pain were significantly more likely to have a lifetime history of dysthymic disorder, current and lifetime panic disorder, somatization disorder, childhood sexual abuse and hysterectomy (1996). In a randomized survey of 1599 women, 31.5% of participants reported a diagnosis of gynecologic problems in the past 5 years. Those with problems were more likely to report childhood abuse, violent crime victimization, and spouse abuse (Plichta and Abraham, 1996). Another study looked at the connection between chronic intractable pain and histories of childhood sexual abuse in 112 women sampled from a large university campus health center. Fifty-nine (59) women with chronic back pain were sampled and compared with 53 control subjects obtained simultaneously from the same clinical population. The women with chronic intractable back pain had a significantly higher percentage of childhood sexual abuse experiences than controls (Pecukonis, 1996).

Koss and her associates looked at the long-term physical health consequences of criminal victimization. Among a population of almost 400 adult women, they found that compared with nonvictims, victimized women reported more distress, less well-being, visited the doctor twice as frequently and had outpatient costs that were 2.5 times greater (Koss et al., 1991). They also studied almost 2300 women in a health maintenance organization. They had a 45% response rate to their survey and 57% of them had been victims of crime. Rape incidence was approximately 15 times higher than the National Crime Survey estimates for women. Medical care had been sought by 92% of crime victims during the first year following the crime and by 100% during the second year (Koss et al., 1990).

Amir et al. (1997) investigated fibromyalgia and PTSD. Among the PTSD group, 21% had symptoms of the disorder compared to 0% of the control group. When compared to the PTSD group without fibromyalgia symptoms, these patients reported more pain, lower quality of life, higher functional impairment and suffered more psychological distress. Geisser et al. (1996) examined self-reports of pain, affective disturbance, and disability among pain patients with and without symptoms of PTSD. Patients without PTSD symptoms were further subdivided into

persons with no or few symptoms of PTSD whose pain was accident related (Accident/Low PTSD); and patients whose pain was not accident related and did not have PTSD symptoms (No Accident). Both accident-related pain groups were more likely than No Accident patients to be involved in litigation or receiving compensation. Accident/High PTSD patients displayed higher levels of self-reported pain compared to the other two groups. The Accident/High PTSD group also had the highest levels of affective disturbance. Davidson et al. (1991) found an increased incidence of bronchial asthma, hypertension, peptic ulcer associated with PTSD among a large epidemiological sample.

All of these studies indicate that a past history of trauma can impact on post-accident adjustment and health, particularly when the trauma dates back to childhood exposure. It is also well established that a recent trauma can “trigger” the emergence of symptoms related to a long-buried traumatic experience. Herein lies one of the causality dilemmas. If a woman who has been sexually abused as a child is at elevated risk for chronic pain and then is hurt in a car accident, was it the sexual abuse or the accident that “caused” the chronic pain? And what if she had the pain before the accident but continued to work and then after the accident is immobilized? Would the previous trauma ever have been “unearthed” had the automobile accident not occurred? How much do we attribute liability to the past experience and how much to the present? How much of her determination to “make somebody pay” belongs to the present party at fault for the vehicular damage and how much is she taking advantage of an opportunity to make someone accountable for her lifelong pain? Is she malingering? Does the car accident aggravate a pre-existing condition or does the previous history of abuse represent a predisposition, a subject entirely different from an already present disorder? It is clear from the data thus far presented that there is no simple answer for these questions, that this is the dilemma that the web of causation so clearly illustrates.

COURSE, COMORBIDITY AND MVAS

As is clear, PTSD rarely occurs alone. The rate of psychiatric and medical comorbid conditions is extremely high. In 1997, Mayou, Tyndel and Bryant looked at the psychological outcome of a sample of 111 non-head-injured victims of motor vehicle accident five years after the accident. These victims had been assessed previously in a prospective one-year study. A substantial minority described continuing social, physical and psychological difficulties and a quarter of those studied suffered phobic anxiety about traveling in a car. Little had changed between three months, one year, and five years. The prevalence of PTSD remained approximately ten percent throughout the follow-up period, but most early cases had remitted by five years and a similar number of delayed new onsets had occurred between one year and five years. Physical outcome as well as postaccident intrusive memories and emotional distress predicted the presence of PTSD at five years after the accident.

Poor outcome in those who made legal claims, especially those not settled after five years, appeared to be due to their having more serious physical problems. In another article, Mayou (1997) points out that if we look only at formal diagnoses of PTSD, we miss the high rate of travel anxiety and phobia that often occur after vehicular accidents and that have a great impact on everyday life.

In a more recent article (1997), Blanchard et al. looked at 145 individuals who sought medical attention as a result of the accident. They were assessed initially at 1-4 months after the accident and then followed for six months to see how many of the fifty-five who were diagnosed with PTSD and the forty-three with sub-syndromal PTSD would remit. Fifty-five percent of those who showed PTSD initially and sixty-seven percent of those with sub-syndromal PTSD, had remitted at least in part by 6 months. Five percent of the sub-syndromal types had worsened. The variables that largely appeared to predict remission were the severity of the initial symptoms, the degree of initial physical injury, relative physical recovery by four months, and whether a close family member suffered a trauma during the follow-up period. Earlier, Blanchard and colleagues (1996a) reported on 132 victims of automobile accidents and assessed them at 1-4 months after the accident, 6 months later, and 12 months later. Forty-eight met the full criteria for PTSD and of those, half had remitted at least in part at the 6-month assessment and two-thirds by the year assessment. They noted three variables that accounted for the difference between those who improved and those who did not: initial scores on the irritability and foreshortened future symptoms of PTSD and the initial degree of vulnerability the subjects felt in a motor vehicle after the accident.

Buckley, Blanchard and Hickling (1996) did a prospective study of delayed onset PTSD secondary to motor vehicle accidents. The participants had not met criteria for PTSD one to four months after the accident but had developed PTSD during a one-year follow-up interval. Compared with those who did not develop PTSD as well as to those who met criteria for acute onset PTSD, the delayed onset participants were more symptomatic at the time of the initial interview, had poorer social support prior to and after the accident, and for the month prior to the accident had lower Global Assessment of Functioning scores. In another report from the Blanchard group (1996b), using 158 victims of automobile accidents, 70% of the subjects could be classified as PTSD or not with four variables: major depression, fear of dying in the accident, extent of physical injury, and whether litigation had been initiated.

What do we know from recent research on the relationship between car accidents, psychiatric morbidity, and comorbid conditions? Blaszczyński et al. (1998) recently reviewed the literature on the subject and though cautioning that no consistent profile has yet emerged, there are common reported symptoms. These include: depression, anxiety, irritability, driving phobia, anger, sleep disturbances, headache, and post-traumatic stress disorders.

In 1995, Koch and Taylor pointed out that victims of motor vehicle accidents are frequently left with multiple comorbid psychological and physical disorders including PTSD, depression, pain-related conditions, and phobic avoidance of stimuli associated with the accident. Bryant and Harvey (1995) studied 56 Australian patients twelve months subsequent to hospitalization after a vehicular accident. Of this sample, 41% reported significant levels of psychological impairment. Those reporting psychological disturbances had more pain, higher rates of unemployment and substance abuse, avoidance of road transport and compensation claims. Only 44% of these had sought professional help for their conditions

In a study done by Blanchard and colleagues (1996a) four variables combined to predict 64% of the variance in the degree of post-traumatic stress symptoms at twelve months: the presence of alcohol abuse, an Axis-II disorder at the time of the initial assessment, total scores on the hyperarousal and avoidance symptoms of PTSD present at the initial post-accident assessment. In another study by Kuch and colleagues (1994), 55 survivors of traffic accidents with minimal injury and chronic pain were examined. Over thirty-eight percent had simple phobia with onset after the accident. Of these, 23.8% gave a past history of other phobia compared to none of the nonphobics.

Driving phobia is a major problem for many victims after an accident. The inability to drive can impact on the individual's ability to work, to socialize and to actively participate in family life. In the Blanchard et al. 1995 study, all of the driving phobics clustered in the PTSD subgroup, amounting to 15.3% of the PTSD patients who could have been driving at the time of assessment. Over half of the subjects with PTSD who were still driving avoided all driving that was not essential.

The effects of minor head trauma further complicate any analysis of psychiatric comorbidity. In an interesting study reported by Parker and Rosenblum (1996), accident victims who had suffered minor traumatic brain injury were studied twenty months after the accident. There was a loss of 14 points of the Full Scale IQ from estimated pre-injury baseline IQ without evidence of recovery. Personality dysfunctions included cerebral personality disorder and thirty of the thirty-three patients were given a psychiatric diagnosis. The authors caution that the estimation of brain trauma at the time of the accident may be underestimated for many people. In another report by Parker (1996), 31 out of 33 patients suffered an additional psychiatric disorder and manifested a wide spectrum of disorders – cerebral personality disorder, persistent altered states of consciousness, PTSD, psychodynamic reactions to impairment, complex reactions expressing neurological, somatic and psychological dysfunctions. He points out that emotional disorders, combined with persistent cognitive loss and other neuropsychological symptoms greatly impair the capacity of the individual to adapt after traumatic injury and then impact on every aspect of the person's life. This relationship between head injury sustained at the time of an accident and consequent psychological problems may turn out to be a complicated one. Fenton and colleagues (1993) have reported on 45

consecutively admitted patients who had sustained a mild head injury with a post-traumatic amnesia duration of less than twenty-four hours. Interestingly, these head injury patients had an average of three adverse life events in the year preceding the injury compared with 1.5 for controls. Thirty-nine percent of the group was diagnosed with a psychiatric disorder at six weeks after the injury. For this group, the mean level of chronic social difficulties was four times that for the non-psychiatric cases. In this study, the emergence of and persistence of post-concussion syndrome was associated with social adversity before the accident.

Headache is another common presenting complaint after a motor vehicle accident. Chibnall and Duckro (1994) looked at the prevalence of PTSD among post-traumatic headache sufferers and noted that nearly 30% of the sample were diagnosed with PTSD. Depression and suppressed anger were significantly higher in subjects with PTSD compared to the rest of the sample and those with PTSD were more likely to have a history of headache prior to the accident.

WHAT DOES THIS HIGH COMORBIDITY MEAN?

Solomon and Bleich (1998) have recently proposed four alternative hypotheses to explain the high comorbidity found in PTSD: They reason that: 1) preexisting disorders could constitute a vulnerability to PTSD; 2) other disorders could be subsequent complications of PTSD; 3) the disorders could co-occur because of shared risk factors; and 4) comorbidity could be the result of a measurement artifact. As we have already seen, there is evidence that supports the first three hypotheses. In many studies of various survivor groups, depression is the most common comorbid condition. In the authors' discussion, they note that for about 65% of the patients they reviewed for lifetime diagnosis, both PTSD and depression emerged simultaneously. For 16%, depression was reported as the first disorder and PTSD as the first for 19%. In considering whether comorbidity could be a measurement artifact, Bleich et al. (1997) looked at patients originally diagnosed with lifetime depression and found that 98% would still be diagnosed as such even after the common symptoms were removed and 70% of those with PTSD would still be so diagnosed. They drew the inference that PTSD and depression are distinctive diagnostic categories. They also note that there is some evidence that biological markers for the two disorders may also be different. To address the question as to whether the threshold for diagnosing comorbid depression should be raised because of symptom overlap, Blanchard et al. (1998) examined data from 107 motor vehicle accident victims. Of the 62 who met PTSD criteria 1-4 months after the accident, 33 also met criteria for major depression and in 27 cases the depression occurred after the accident. Their statistical analysis indicated that PTSD and major depression are correlated but independent responses to trauma. The patients suffering from both syndromes were more subjectively distressed, suffered more role impairment, and

remitted less readily over the first six months of follow-up than those with PTSD alone.

There may, of course, be shared risk factors for both disorders including family and genetic vulnerabilities. Davidson et al. (1985) found an increase in affective disorders among untraumatized relatives of PTSD patients. The shared risk may also involve the nature of the stressor. Certainly wartime involvement exposes victims to multiple losses as do motor vehicle accidents.

On the other hand, it may well be that a comorbid condition like substance abuse is related to pre-existing substance abuse and there is some evidence to support that. Abuse of substances certainly increases the risk for traumatic exposure, while it contributes to chronicity after a traumatic event. Many other pre-existing conditions increase the likelihood that PTSD will occur and that the two or several conditions will interact with negative consequences to level of function.

Given the complicated nature of the human response to overwhelming experience it is likely that all four hypotheses will find some confirmation and some disconfirmation. As van der Kolk and colleagues (1996) have pointed out, PTSD and its comorbid conditions should be seen as *“complex, somatic, cognitive, affective and behavioral effects of psychological trauma”* rather than as separate conditions.

COMORBIDITY, PTSD AND COMPENSATION

The issues of litigation, compensation, malingering and secondary gain complicate the entire discussion of comorbidity. Human beings are motivated by both conscious and unconscious needs. Consciously, victims desire compensation for injuries sustained due to the negligence or malice of others and it is this conscious intention that is endorsed and supported by the legal notion of liability and restitution. But there are others who consciously desire to “get something for nothing”, who fraudulently claim injury where there is none, or who exaggerate injury in order to increase their claim – these are the malingerers. Additionally, there are people who unconsciously exaggerate their disability because it satisfies their unconscious needs such as a desire for vengeance against a previous injurious party who they cannot confront or in order to evoke sympathetic concern from others that is unavailable in any other way.

In 1968, Levine wrote about the “automobile accident syndrome” in discussing the increasing appearance of cases in which minor physical findings were accompanied by major subjective complaints. He attributed the basic motivation to be that of establishing a medical basis in order to receive monetary compensation. This argument is consistent with one that stretches back to at least the last century when railway accidents contributed to the notion of “compensation neurosis” implying that the symptoms would be resolved as soon as the litigation was resolved. This

overly simplistic notion has been largely discredited and studies from various survivor groups paint a far more complicated picture. In 1973, Culpan and Taylor published an article on a study in which they attempted to follow-up and classify eighty-two New Zealand accident victims referred by lawyers for psychiatric evaluations. They were able to note the progress of 87% of the original sample. Approximately one-third of the group were disabled by neurotic symptoms which appeared to result from the emotional stress of the accident itself and they classified these patients as suffering from true traumatic neurosis, as it was called at the time. They believed that about half of the group were thought to be unconsciously motivated by the possibility of financial compensation and unlike the first group, they failed to improve or became worse until the time of the settlement of their claim. Six percent of the sample were considered to be outright malingerers with “compensation neurosis” who denied concern about the outcome of their lawsuits.

Smith and Frueh (1996) looked at the relationship between compensation seeking, comorbidity, and the apparent exaggeration of PTSD symptoms among Vietnam combat veterans. They found that there was increased comorbidity, particularly affective disorders, among veterans who exaggerated their symptoms, but there was not a relationship between the symptom exaggerators and those seeking compensation. In another study by Frueh, Smith and Barker (1996), compensation seeking veterans had much higher scores of pathology across a wide range of inventories, but they did not differ in frequency of PTSD diagnoses compared to veterans not seeking compensation.

In 1982, Mendelson reviewed the literature and up to that point 75% of those injured in accidents had failed to return to gainful employment two years after legal settlement. McFarlane investigated the survivors of the Ash Wednesday bush fire disaster in Australia and found similar findings in an eleven year follow-up period (Pitman et al, 1996). In Green et al's (1990b) fourteen-year follow-up of the Buffalo Creek dam collapse of 1972, no significant clinical differences were noted between the litigant and nonlitigant survivor groups. In an Israeli study of combat veterans, veterans who had experienced the most severe traumas and had subsequently developed the most severe symptoms and functional limitations sought compensation. The reported range and severity of symptoms and functional limitations did not diminish with compensation and the social function of both groups before the war was comparable (Solomon et al, 1994). Tarsh and Royston (1985) did a follow-up study of 35 claimants one to seven years after their lawsuits were resolved and compensation was received. All these patients suffered from “accident neurosis” with many somatic symptoms. Few claimants recovered and such recovery as did take place was unrelated to the time of compensation. According to the authors, the legal process and the delays involved caused great distress. Mayou, Bryant and Duthie (1993) studied the differential impact of motor vehicle accidents on victims who were able to sue and those who were not. They found that litigation status did not influence the prevalence of psychiatric disorder,

the course of the disorder, or the chronicity of associated disabilities. They found that severe chronic physical and psychological symptoms were also found in the group that was not going to court. In a study by Packard (1992), fifty accident victims who suffered from what had come to be defined as “permanent posttraumatic headaches” were evaluated at least one year after litigation had been settled. Patients with a pre-accident history of headaches, another injury, or head injury were excluded from the study. Only four patients reported any improvement in their headache pattern after settlement of their case.

Binder, Trimble and McNiel (1991) looked at the course of psychological symptoms after a lawsuit and found that a better psychological outcome was associated with a longer time after resolution of the lawsuit, a shorter time between injury and resolution of the suit, and less severe initial psychological symptoms. The finding that a shorter time between injury and resolution of a lawsuit leads to a better prognosis is interesting in that it supports an observation that is frequently observed clinically – that the process of engagement with the legal system can itself be retraumatizing for the victim (Pitman et al., 1996).

CONCLUSION

Given the existing data, it is impossible to attribute simple cause and effect relationships to the effects of traumatic experience, even in one as seemingly straightforward as a motor vehicle accident. Each person brings individual risk factors and vulnerabilities, including previous life experience, to the accident scene. Each accident then has its unique traumatic aspects that interact with the variable perceptions of every person involved in the accident. The victims then struggle to cope with the traumatic event and its after effects, while attempting to make some kind of sense out of what has happened to them. For a certain proportion of the population, the attempts to cope will be further complicated by the “biopsychosocial trap” that is post-traumatic stress disorder (Shalev, 1996), as well as a number of other psychological and physical conditions that make a healthy adjustment far more difficult. Given such a complex web of associations, the physician must be willing to be aware and address issues of multicausality that affect the body, mind, family and soul of any victim if he or she is to truly committed to the provision of good care. As for questions of liability and compensation, for now the only useful measure we probably have is before-and-after level of function and the means to address this will continue to evolve – and be fought out – in the courtrooms of tomorrow.

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