Psychiatry and the Law of Personal Injury

Walter Bromberg

Stuart A. Brody

Follow this and additional works at: https://via.library.depaul.edu/law-review

Recommended Citation

Available at: https://via.library.depaul.edu/law-review/vol21/iss1/3
PSYCHIATRY AND THE LAW OF PERSONAL INJURY†

WALTER BROMBERG*
STUART A. BRODY**

INTRODUCTION

IN RECENT years, the function of psychiatrists, neurologists and psychologists in personal injury cases has become so extensive that attorneys working in this area need a wide acquaintance with new medico-legal viewpoints. Medical aspects of negligence matters involve not only an exact description of the injuries sustained by the client, but also a consideration of the vital subjects of causation and prognosis. In addition to his diagnostic and prognostic evaluation, the medical expert is often asked his opinion as to the proximate cause of a plaintiff's injuries.

In complex cases, the extent of physical, mental and emotional disabilities and their impact on the plaintiff's personality and life pattern become important. Thus, the psychiatric expert may be called upon to supply specific information and carefully considered opinions on all phases of the plaintiff's life.

This article will describe some of the major types of traumatically induced injuries which have a psychological impact on the patient and which are important considerations in seeking recovery. Illustrations from published case material will be used, as well as clinical cases from the files of one of the authors (W.B.) and court decisions ruling on the compensability of certain types of injuries (e.g., impact v. non-impact emotional shock), but the major emphasis will be given to the medical and psychological sequelae of common personal injury cases.

† This article is excerpted from a chapter of a forthcoming work by the authors entitled CURRENT ISSUES IN PSYCHIATRY AND THE LAW.

* DR. BROMBERG, M.D., L.F., A.P.A., is Adjunct Professor of Legal Medicine at University of Pacific, McGeorge School of Law, Sacramento, California.

** DR. BRODY, Ph. D., J.D., is Assistant Dean and Associate Professor of Law at University of Pacific, McGeorge School of Law, Sacramento, California.
THE MEDICAL SPECIALISTS

Medical problems in negligence cases involve many types of specialists. It is important for the attorney to know what the functions of these individuals are, so that he may use these experts’ skills most effectively, saving time and expense for himself and his client.

An attorney handling a personal injury case may find his client requiring the services of an ophthalmologist (specialist in diseases of the eye), orthopedic surgeon (specialist in bone and joint injuries and diseases), otolaryngologist (specialist in diseases of the ear and larynx), or a number of other medical specialists. However, our discussion below will be limited to those most directly involved in diagnosing and treating emotional problems of the injured plaintiff.

The functions of the various specialists in these areas are as follows: The neurologist handles disorders of the brain, spinal cord and peripheral nerves. The neurosurgeon performs similar services, but deals mainly with surgical correction of brain and spinal cord injuries. A psychiatrist handles problems of intellectual dysfunction, emotional disturbances and social deviancy. The psychoanalyst is usually a psychiatrist who has received additional training in the therapeutic techniques of Freud or one of his disciples; he is not often involved in negligence cases, but may be called upon where issues of emotional trauma or distress are involved. The psychologist is not medically trained, but studied instead in a university graduate department of psychology where he earned, in most cases, the Ph.D. degree. He may become involved as an expert where, for example, issues of impaired intelligence due to brain injury require a precise evaluation of plaintiff's intelligence level, or where personality changes in the plaintiff call for the administration and clinical interpretation of various projective techniques such as the Rorschach, TAT, or Draw-a-Person test.

The specialists described above operate from varying theoretical orientations and professional attitudes. Hence, it is important for the attorney to recognize that the physician’s own psychological attitudes in cases involving emotional and nervous disabilities may unwittingly, or even consciously, invade his final judgment as to diagnosis, causation or prognosis. In general, it can be said that the neurologist's perspective is in the direction of organicity, i.e., demon-
strable signs of nervous system injury or disease. The neuro-surgeon is interested more in objective signs of pathology and less in subjective complaints. The psychiatrist's training gives him a humanistic bias toward the patient. His viewpoint includes social, cultural, economic and organic (i.e., neurologic) understanding, as well as the problems of personality. Moreover, the psychiatrist, especially one with psychoanalytic training, relates his observations to his knowledge of unconscious motives and emotions, such as dependency, hostility, psycho-sexual tendencies, etc.

In discussing the physician's attitude towards his patient, it is important to note that he may become involved in what is called "counter-transference." Just as the patient has a transference on an unconscious level from older figures in his environment to his doctor, so the doctor (or the lawyer) may have feelings arising toward his patient (or client) from his own unconscious needs. The doctor, thus, may have an inclination to be the saviour or supporter of emotionally dependent individuals or a severe critic thereof. These attitudes are calibrated within the psychiatrist, if he knows himself, in evaluating a patient's reactions to injury. The psychiatrist should understand not only the conscious material presented to him, but also the basic, unconscious attitudes underlying the patient's complaints.

CLASSIFICATIONS OF NERVOUS SYSTEM INJURIES

The attorney will find that both physical and emotional injuries are involved in many personal injury cases. Often, the overlap between the two makes it difficult to distinguish which complaints are somatically induced and which are psychologically based. However, insofar as it is possible to make a distinction between the two, we shall attempt to do so. Furthermore, while emotional problems can develop from injuries (or near-injuries) to any part of the body, we shall generally limit our discussion to injuries to the central nervous system.

PHYSICALLY-INDUCED NERVOUS SYSTEM INJURIES

The most obvious types of nervous system injuries are skull fractures, brain concussion (with its subsequent postconcussive state), spinal cord damage and peripheral nerve injury. Other common brain...
syndromes include sub-dural or extra-dural hematomas (blood clots), hemorrhages in various areas of the brain, lacerations of brain tissue, and the often denied, but still valid, "neurosis after trauma," or "traumatic neurosis" as it is sometimes called. This last syndrome will be discussed under the section dealing with traumatically-induced mental disorders.

The classification of various mental and physical states following head injury is not entirely agreed upon by all authorities. That classification used by the neurologist and neuro-surgeon differs in some respects from that of neuro-psychiatrists and psychiatrists, chiefly for the reason that the latter are more concerned with the remote mental and emotional effects of such injuries.

The neurologist and neuro-surgeon usually classify head injuries on the basis of pathology as: (1) closed head injuries, including simple concussion, cerebral edema, contusion, and laceration; (2) depressed fracture; and (3) compound fracture. These injuries are further described as follows: (1) closed head injuries—where the injury is not sufficiently serious to open the skull casing, although there may be a linear fracture; these include: (a) simple concussion—the brain encased within the skull receives a "bump" following a blow to the head. There is a momentary stunning effect and possible transitory headache lasting from a few hours to a day; (b) cerebral edema—swelling of the brain tissues, the amount depending upon the degree of injury; (c) contusion—a bruise of the brain tissue itself or the membranes covering the brain (dura mater, arachnoid, pia mater); (d) laceration—a tearing of the brain tissue or surrounding membranes; (2) depressed skull fracture—where a piece of bone is driven into the brain and its coverings; (3) compound skull fracture—where the scalp is broken by a bone fragment.

Corrective surgery is usually required in cases of depressed or compound skull fracture. Any head injury, whether or not accompanied by a fracture, can result in confusion, delirium, coma, or even death, depending on the area of the brain affected and the severity of the blow. Should bleeding occur in the brain tissue, paralysis or other symptoms may occur. When the acute symptoms subside and the patient recovers his consciousness and awareness, he is left with the specific symptoms of headache, dizziness, visual troubles, etc. After the acute phase has passed (in six months generally) and
symptoms still persist, the patient may be said to be suffering from a chronic brain syndrome, indicating the brain damage may be irreversible.

Since the after-effects of head injuries often fall to the psychiatrists or neurologists, their concern with mental or behavioral reactions to trauma is naturally greater. Although workers in this field are not unanimous, the following classification represents a practical overview of the wide range of sequelae of head traumas. Hence, the following classification is chiefly on the basis of reactions in the patient:

1. **Acute brain syndromes:**
   - (a) simple concussion;
   - (b) major concussion;
   - (c) post-concussion syndrome;
   - (d) traumatic coma;
   - (e) traumatic delirium;
   - (f) Korsakoff's syndrome (very uncommon).

2. **Chronic brain syndromes:**
   - (a) with neurotic reaction;
   - (b) with psychotic reaction;
   - (c) with behavioral reaction (personality deterioration);
   - (d) post-concussion syndrome.

3. **Secondary traumatic mental disorders:**
   - (a) "terror" or "traumatic" neuroses;
   - (b) secondary psychoneuroses, including anxiety states and hysteria;
   - (c) "compensation" neuroses.

4. **Functional psychoses** (e.g., schizophrenia, affective psychoses, *i.e.*, manic-depressive states).

5. **Malingering.**

We shall now briefly describe some of the more important of the conditions listed above.

**Acute Brain Syndromes**

The acute brain syndromes are distinguished from chronic brain syndromes by the relatively transitory nature of the symptoms. Injury to the brain is considered reversible in these syndromes and the after-effects of the trauma ordinarily do not persist beyond three to six months.

In simple concussion there is a stunning or momentary loss of consciousness following a blow to the head. The patient has an awareness of what has happened; therefore, he claims no amnesias or similar effects. A mild, transitory headache may follow.

A major concussion may follow a blow to the head, explosion, or whiplash accident. The patient commonly, though not always, is
rendered unconscious for a period of from five minutes to one hour. There may be amnesia for the events just prior to and following the injury. This loss of memory may last for as little as one hour to as long as one or two days. As the patient returns to awareness, he begins to describe headache, dizziness, and other symptoms. If the injury is more serious than described above, the patient may experience prolonged unconsciousness (coma), delirium, or Korsakoff's syndrome. The latter is a sustained period of defective memory with the patient fabricating to fill in the gaps. It is rather uncommon. Hardin describes the clinical picture of concussion succinctly:

About one-half of the patients suffering concussion will recover within a few days, while the remainder will show the sequelae generally called "postconcussion syndrome."

The picture produced by a serious brain injury regularly follows the pattern of recovery outlined by Schilder and Symonds: coma, followed by stupor (deep clouding of consciousness with general restiveness), delirium (moderate clouding of consciousness with disorientation, bewilderment and helplessness), eventuating in a state of Korsakoff's psychosis before recovery.¹

At this point, it might also be well to point out that, in addition to a direct blow to the head, two other forms of injury may cause a concussion. These are the contra-coup concussion and the cranio-cervical syndrome, more commonly known as whiplash.

A contra-coup concussion is one which results in injury to the opposite side of the brain from that which was struck. For example, if a blow occurs to the right temporal area of the head, the left temporal lobe of the brain might strike the internal surface of the skull. Since the skull is a rigid structure, whereas the brain and its surrounding membranes are relatively soft, the brain may be "slapped," so to speak, against the irregular, internal outline of the opposite (left) side of the skull. In these cases, a shearing effect can result as the blow is transmitted to the membranes, blood vessels, or cranial nerves exiting from the brain to the sense organs such as the eyes, ears, and nose inducing specific sensory symptoms as blurred vision, tinnitus, etc.

In the whiplash injury, the head is held back momentarily due to inertia, then snapped forward and backward one or more times. The supporting neck muscles are strained severely, but more sig

nificantly, the brain is "whiplashed" causing a brain concussion within the skull without the head having been struck.

This type of concussion is more common in rear-end auto crashes than is generally acknowledged, even when the driver or passenger was using seat belts. Experimental studies with rhesus monkeys have demonstrated that cerebral concussion, as well as gross hemorrhages and contusions over the surface of the brain, can be produced through whiplash action of the head on the neck alone, without direct impact to the head.\(^2\)

Returning now to the symptoms of concussion, the characteristic signs usually appear within one to five days. The patient experiences a band-like headache, intermittent in character. He may also suffer from any of the following: irritability, lack of ability to concentrate, poor memory, sensitivity to light, watering of the eyes, sensitivity to noises, ringing in the ears, fatigue, loss of appetite, nausea, dizziness (either constant or in sporadic black-outs) and depression with crying spells.

The post-concussion syndrome is of special interest to the attorney because of the large number of claims for recovery from auto accident-related injuries found here. Post-concussion syndrome may be either acute or chronic depending on the length of time the patient is likely to be afflicted with the symptoms. The syndrome may last anywhere from three months to five years, in varying degrees.

As the late effects of concussion merge into a post-concussion syndrome, factors of a psychological nature, other than the actual impact, come into play. The personality of the injured party has much to do with the later clinical picture. As will be noticed, the post-concussion syndrome is similar to the symptoms of neurosis after trauma, (traumatic neurosis) to be discussed later.

Two cases of post-concussion syndrome will now be described. In the first case, a young man of eighteen, with no previous illness of any kind, was injured in a California auto accident. In the second, a fifty-four year-old man was injured in New York. Prior to this accident, his larynx had been removed due to cancer.

**Clinical Case #1.** Mr. Gomes, aged eighteen, was involved in a rear-end auto

---

accident. On February 14, 1968, the car which he was driving apparently overturned. The patient was aware only of a sensation of falling and did not remember whether he struck any part of the car. When the car came to rest, he felt nauseous and had pain in the right side of the head from a laceration. He was treated at the hospital that day. X rays of the skull were negative.

Following the accident, the patient suffered dizziness, especially when he rose from a sitting or lying position. It occurred several times a day, without warning. At times it almost amounted to a "blackout" in which his vision blurred to the point where he could not see. Dizziness cleared up in a few seconds ordinarily. In addition, the patient suffered headaches (frontal in nature) lasting for a day to a month, and nausea. The patient felt "butterflies in his stomach" and had a general feeling of tension which was not present before. He said he was "leery" and "didn't trust it," meaning that he was worried lest the dizziness and blackout spells come when he was in a vulnerable position. He did not feel "sure" of himself.

The course of the symptoms during the following year followed the usual progress of this type of head injury. More than a year after the accident the patient still suffered from occasional dizzy spells and headaches, and often vomited (dry heaves) in the morning.

Two years later, the patient showed improvement as prognosticated. Headaches no longer present: nausea reduced but dizziness occurred about once a week. When he got up quickly, he would black out for a few seconds.

Clinical Case #2. On November 7, 1965, Mr. White was a passenger in a car which was struck broadside. He was aware of having heard the impact, then he saw blood streaming down his face and remembered little until he found himself in the hospital being treated by a surgeon. He estimated that he was unconscious or semi-conscious for about fifteen minutes. He had pain over the forehead, over the right eye, in the right knee and in the back.

The patient complained of: headaches, frontal in type, occurring approximately four times a week and lasting several hours; vertigo, in the morning especially, lasting about five minutes but occurring for briefer periods throughout the day; pain in the neck on the right, especially when turning; pain in the lower back; restless sleep with periods of awakening drenched in perspiration; and nervousness and tension, especially in crowds and crossing the street.

Mental examination showed that the patient was unusually stoical and tended to minimize his symptoms, except for the headache and vertigo. For example, he did not recognize that his episodes of perspiration at night represented nightmares typical of post-traumatic shock.

Not infrequently, one finds a prior history branding such persons as accident-prone, at least from the standpoint of the defendant insurance carriers. There may be psychological reasons why these individuals are involved in multiple accidents, or it may be pure coincidence. A thorough history is all-important in weeding out the various psychological factors operative in a given case. In any event, the effect on the resulting symptoms is noticeable.

In the following case, a young man suffered a brain concussion
with unconsciousness, a convulsive seizure at the time of impact and a positive EEG soon after. The post-concussive syndrome was clear, but this clinical picture took on some features of an hysterical neurosis.

Clinical Case #3. The patient was twenty years old. There was a prior history of injury following an automobile accident on December 24, 1965. He was a passenger at that time. The symptoms were right frontal headaches, trance-like states and "violent behavior," which led to his transfer to a psychiatric hospital. In January of 1966, he suffered a second auto accident accompanied by minor head symptoms. On March 5, 1966, in a third accident, the patient's car was struck from the rear. Immediately thereafter he felt pain in the right side, had a feeling of "flying" and passed out. He woke up in a general hospital where he remained for about nine days. An epileptic fit of some type occurred during this period. The X rays of the skull were negative but the electroencephalogram was reported as "mild to moderate abnormality" without focal signs and with "scattered 5-7 slow waves."

Following this latest accident the patient complained of feelings of light-headedness, dizziness, fainting spells with tremor during which he was partially aware of his surroundings but could not talk or place the conversation he heard. He had one such spell in 1966 and two or three in 1967. He also complained of blurred vision, loss of weight (twelve pounds) and nervousness. During his two hospitalizations, he was observed in two episodes of "spastic shaking with tremors of legs."

The examiners felt that basically they were dealing with a neurotic youth in whom evidences of cerebral concussion were intensified. The tremors of legs were considered hysterical in nature.

Hysterical or other psychological reactions following concussion occur with special vividness among children and adolescents. The following clinical case demonstrates this situation:

Clinical Case #4. A boy of 13, a pupil in the sixth grade of elementary school, from a bi-lingual home, was struck by a motor vehicle, his head hitting the pavement. Although the question of unconsciousness was not clearly settled, it appeared that the patient was stunned but not unconscious. He remembered the policeman who helped him and was able to get up and walk just after the accident.

The presenting symptoms were dizziness and inability to walk, difficulty in moving head due to stiff neck, and difficulty in seeing, described as a blinking spasm. In the ensuing weeks the patient suffered from other symptoms as follows: insomnia with restlessness and talking in his sleep, somnambulism, bed-wetting, a complaining attitude, fatigue, complaints of body-pain and decrease in efficiency in school work.

During the mental examination he was quite uncommunicative. His answers were given slowly. With encouragement, the patient talked more freely as the interview progressed. The initial impression of mental dullness changed as the interview progressed. His mood was generally inhibited and retarded but he complied with spoken instructions from the examiner. The general phobic attitude could be reduced by dealing realistically with the boy. With casual conversation and a relaxed attitude, the patient lost some of his slowness and fearfulness. The personality change described by the father, this obtuseness, apparent dullness and withdrawn attitude, was quickly dissolved by a "therapeutic attitude."
It was concluded, therefore, that there was no permanent personality change manifested in this boy, but a reaction of fearfulness (technically known as a psychological regression), which gave the impression of permanent personality change. This amounted to a traumatic neurosis following concussion.

The following case illustrates some of the other psychological complexities which may accompany head injuries, accentuating or distorting the symptoms of concussion:

Clinical Case #5. The patient, a married woman of twenty-six, was always well and physically vigorous until November, 1965, when she became involved in a motor car accident. She had married at the age of nineteen, had two children, the older of whom was involved in the same accident. There were no previous operations or major illnesses.

In November, 1965, while in her car at a stop light, sitting in the driver's seat, she felt an impact from behind and felt the car pushed forward until it struck another car; she felt herself being rocked back and forth, striking her face on the dashboard. Her four-year old son, sitting immediately behind her, struck the seatback and sustained a fractured skull. After the moment of impact, the patient was aware of her bleeding nose and of her own hysterical screaming as she thought her unconscious son, who was bleeding from the ear and crushed beneath the seatback, was dead. The patient and son were removed to a hospital where both were X-rayed and examined.

Because of her extreme apprehension regarding her son, she was actually unaware of her own symptoms until about a month after the accident. Then the patient began to suffer from headaches in the back of the head, tension or excitement which lasted all day, nervousness, irritability, jittery sensations in her abdomen, depression and restlessness, inability to sleep with a feeling of being unable to accomplish her work or concentrate, burning of the eyes, pain in the mid-back, and sudden sharp pains moving up the back to the shoulders, neck and vertex of the head, then down to the buttocks. At these times her head was motionless and she was almost incapacitated from any movement of the neck and back muscles. This lasted about a week or ten days and subsided under medication. There was also numbness of the back and abdominal area, especially when sleeping, and some numbness of the toes, phobias when in a car or crossing the street, occasional dizziness, and decrease in sexual libido.

Examination showed only moderate tenderness over the posterior neck muscles but no specific tenderness of spinous processes or paravertebral muscles. All movements of the trunk and extremities were performed within normal limits.

This patient had suffered a definite whiplash injury. Because of the serious injury to her four-year old son, however, she repressed her own symptoms until he was out of danger—then the full flood of symptoms assailed her. This reaction is classic for whiplash victims.

The symptoms of concussion and post-concussion, enumerated above, occur in a pattern that deviates only slightly from case to case, although not necessarily represented fully in each patient. Conversely, patients who start with a full gamut of complaints, may lose
some of them gradually even though headache, dizziness, and nervous tension may persist for years.

The degree of unconsciousness immediately following the accident apparently bears little relation to post-concussive symptoms. Neither do the patient's age, previous personality make-up, cultural background or other geographic or social factors. This remarkable specificity of symptoms can be related to the pathology of concussion (minute hemorrhagic areas in the brain tissue, contusion of tissues etc.). One proof of this can be seen in brain concussion cases accompanied by changes in the electroencephalographic waves, i.e., slow waves between four and seven per second. During the first three months after the initial concussion sixty-three percent of post-concussion cases show these positive brain wave tracings. After two years, thirty-eight percent continue to show changes in EEG patterns.\(^3\)

Unlike the physical symptoms, however, neurotic complications following the original concussion do relate to the personality make-up of the patient, a situation which often renders expert judgment difficult.

Although the acute brain trauma and the chronic brain trauma are classified separately here, in reality they often merge. Furthermore, it is often difficult to differentiate organic from mental sequelae, and the separation of neuro-surgical from neuro-psychiatric cases or primary vs. secondary brain trauma syndromes. The following case will illustrate how the organic and psychological factors intertwine:

Clinical Case #6. A woman of thirty-four was admitted to the hospital on August 2, 1965, following an automobile accident. The patient on admission was found to be suffering from a cerebral concussion, a contusion of the brain with an aphasia (difficulty in speaking), a temporary paralysis of the right side of the body and extensive lacerations of the face.

The patient, a passenger sitting next to the door, was injured when a tractor-trailer made a u-turn in front of the car. As a result of the impact, the seat moved forward, the engine was pushed back, and the roof of the car caved in. She sustained a head injury, injuries to both legs, the left hand and right arm.

An electroencephalogram performed ten days after admission indicated a moderate amount of 4-6 cycle per second slow wave activity, with an occasional paroxysmal sharp wave accentuation, indicating abnormality. The official diagnosis was cerebral concussion, contusion of the brain with an aphasia and a temporary right hemiparesis and many lacerations of the face, head, hands, arms and legs.

3. CURRAN, LAW AND MEDICINE 266 (1960).
The past history was negative. Following hospitalization, the patient began to be aware of the following symptoms: headache, some dizziness, tearing and burning eyes with blurring of vision, insomnia, poor appetite, nervousness, irritability, depression and memory loss. In addition, there were pains in the neck, shoulder, and knee, especially on the right side, numbness of the hands and pain in the low back.

The final diagnosis: brain laceration in the left frontal-temporal lobe of the brain in addition brain concussion and multiple lacerations. The evidence of brain injury was the unconsciousness, aphasia, right-sided reflex and motor signs. The concussive symptoms were headache, dizziness, blurred vision, insomnia, nervousness, memory loss, etc. Her husband confirmed the fact that she was depressed, tense, irritable, and sometimes confused. The mental state was characterized by dullness, some traces of aphasia, restricted interests and difficulty in concentration.

The significant point of this case was the demonstrable pathology in the brain. More often it is not so apparent; yet, the patient suffers just as severely as if hemorrhage in the brain, neurologic signs of motor dysfunction or a positive EEG test were present. Moreover, a negative EEG early in the case does not disprove an injury to the brain. In fact, in the ordinary concussion case, the brain wave test is of minor significance. It is also noteworthy that many children frequently show abnormal EEG's without a history of head trauma.

The significance of unconsciousness in terms of the resulting clinical picture also requires comment. It is generally stated that the duration of the unconscious period coincides roughly with the amount of tissue injury. This is true for major degrees of brain injury. Thus, a prolonged coma usually means a serious injury and hence correlates with prolonged symptomatology and disability. But, a brief period of unconsciousness (from a few minutes to a half-hour) may also result in persistent disabling symptoms even though there is less likelihood of brain tissue damage. There may be constant headache, dizziness, etc., for months even though the patient was merely stunned at the time of the accident. Thus, it should be re-emphasized that, except for prolonged coma, the duration of unconsciousness does not determine the presence or absence of troublesome symptoms in the post-concussive state.

**Chronic Brain Syndrome**

Up to this point, we have described some of the more important acute brain syndromes associated with traumatic injuries. By way of contrast, chronic brain syndromes are distinguished from acute brain syndromes in that the former may be related to permanent
brain damage and are usually considered irreversible. A brain disorder which appears reversible, i.e., acute, in the early stages may prove later to have left permanent damage. If so, it will be a persistent organic brain disorder which is then diagnosed as "chronic." For example, the post-concussion syndrome described above as an acute pattern of symptoms could also be considered chronic if the symptoms persist beyond six months or a year.

In addition to the injury-produced, irreversible symptoms of disorientation, faulty memory, impairment of intellectual functions, loss of interest, etc., chronic brain syndromes may also be accompanied by either psychotic, neurotic, or behavioral reactions.

**Secondary Traumatic Mental Disorders**

Secondary traumatic mental disorders involve those patterns of mental, emotional and behavioral symptoms which are related to the patient's involvement in a traumatic accident but which are not directly referable to demonstrable organic pathology. Included here will be traumatic neuroses (often called "terror neuroses"), ego shock and mental anguish, hysteria, malinger and simulation, functional overlay and compensation neurosis.

According to prevailing opinion, the various neurotic reactions following trauma are regarded as "secondary," that is, they were precipitated by a physical injury to the head or body. Thus, panic reactions following an injury are likened to "terror reactions," such as are experienced in sudden major catastrophes like earthquakes or explosions. This is a relatively new concept deriving from work with the military forces during World War II. Those concepts developed from military studies were carried over to civilian problems of a like nature.

Abram Kardiner, a psychoanalyst, studied "shell shock" cases after World War I, extending his work to World War II cases of "combat fatigue." In both groups he found neuroses to occur in response to a sudden threat to ego integrity, through shelling or aerial attacks. The acute-phase symptoms suffered by soldiers under bombardment included all those reactions commonly associated with panic: tachycardia, palpitation, precordial discomfort, nausea, diarrhea, desire to urinate, dyspnea and a feeling of choking or suffocation. The pa-
tient’s pupils were dilated and his face flushed. He perspired excessively and suffered disagreeable sensations, tremulousness, dizziness and often a sense of weakness and of impending death. Most of these symptoms reflected marked changes occurring in the autonomic nervous system secondary to acute anxiety.\(^4\)

The fact that these symptoms added up to the same manifestations of anxiety which are a basic feature in every neurosis led many authors to consider “traumatic neurosis” as similar to hysterical, obsessional, phobic or compulsive neuroses found among persons not subjected to injury. In other words, the reactions spelled “neurosis,” whether or not they were connected with wartime conditions or trauma. This rather rigid view will be modified here. For the purposes of this article, the nervous conditions encountered in civil practice after injury will be considered to stand on their own, so to speak, as traumatic conditions—the proximate emotional results of injury to the head or body.

“Terror” or Traumatic Neurosis

Many neurologists and psychiatrists disbelieve in the “traumatic neurosis” as an entity. Indeed, the *American Psychiatric Association Official Diagnostic Manual* (1968) does not even include this diagnosis. However, a psychiatric glossary published by that organization does define it as follows:

> The term (traumatic neurosis) encompasses combat, occupational and compensation neuroses. These are neurotic reactions that have been attributed to or follow a situational traumatic event, or series of events. Usually the event has some specific and symbolic emotional significance for the patient. The neurosis may be reinforced by secondary gain.\(^5\)

Early views of the mental effects of trauma to the nervous system reach back to Erichsen, a neurologist, who in 1875 described the “Syndrome of Spinal Concussion.” This pattern of symptoms, later known as Erichsen’s Disease, was characterized by nervousness after trauma without signs of actual injury. Erichsen felt the condition was due to molecular changes in the central nervous system. S.V. Clevenger, an authority on nervous and mental diseases, (circa 1889) supported the existence of this syndrome, but Erichsen’s Dis-

---

ease was not believed by the courts and its existence was doubted for many years. Usually such cases were ascribed to hysteria or malingery.

There still persists a suspicion of malingery by attorneys, juries, and courts when faced with nervous conditions following trauma. Furthermore, some physicians still equate traumatic neurosis with hysteria. This bias partially rests on the earlier notion that hysteria reflected an implication of effeminacy among male patients. It was not until the 1870's that Charcot in France and Beard in America recognized that hysteria could occur among men as well as women. The term “traumatic neurosis” was introduced by Oppenheim in Germany in 1889.

For some years the implication was prevalent that neurasthenia (nerve weakness) lay behind post-traumatic conditions. It was chiefly modified through the body-mind approach of Paul Schilder who showed how the body-image, the picture of our bodies by which we unconsciously view ourselves, is altered at the time of an injury to the head. All persons invest the head and nervous system with considerable emotion. Therefore, any injury to the head disrupts the unity of our body-image giving rise to fear, panic and anxiety, hence the frequency of hypochondriacal symptoms in post-traumatic cases. Beyond that, Schilder pointed out that a feeling of “social injustice” arises which invades the mind of the patient, tending to intensify the symptoms caused by an accident. He feels that he has been unfairly singled out and asks, “Why me?”

Concepts relating to the psychology of head injuries rose in significance with the increasing frequency of automobile accidents in this country and combat experiences in World War II. By the late 1930's, it was agreed by most experts that “post-traumatic concussion,” or “post-traumatic neurosis,” constituted a valid medical condition. For example, in 1939, Walter Schaller, an experienced neurologist, lumped all cases of head-injury sequelae under the phrase “post-traumatic concussion” so as “to distinguish traumatic encephalopathy, concussive neurosis, and post-traumatic concussion from post-traumatic psychoneurosis.” He reasoned that the “concussive

state” (now called post-concussive syndrome) was due to small, petchial hemorrhages from capillaries in the brain tissues, found in the cerebral white matter and mid-brain, whereas the “post-traumatic neurosis” showed no organic findings.7

These considerations brought into view the purely psychological or personality make-up of the injured person. It became important whether an injury might “light up” or aggravate certain psychological reactions in the patient based on an unstable or “neurotic” background. Putting the situation in legal terms, Smith and Solomon asked, “Assuming there was an impact produced by the defendant, to what extent shall the law protect the idiosyncratic or excessively vulnerable person in allowing him damages?”8

The psychology of traumatic neurosis can be explained as the defenses of the ego combating the psychological effects of injury. The ego is conceived of as the executive organ of the mind; it receives impressions, coordinates and directs them. One of the ego’s important tasks is to control emotional excitation. In traumatic neurosis, this control is deficient or weakened, resulting in emotional instability and anxiety expressed through physical symptoms, such as muscular tremors, and emotional ones, such as phobias.

In traumatic neurosis, the ego is hurt or shocked by the trauma, or the ego interprets the accident to be the threat of total annihilation. Instead of repressing or blocking off one group of functions, which might result, for example, in blindness or paralysis of one arm, the ego loses its power to control its integrative functions and panic ensues.

The blow or shock to the ego which results in disturbed control does not necessarily bear any relation to the degree of actual injury sustained. The injuring force can be directly applied to any part of the body or it can be distant from the patient, as in a blast accident. In any event, the ego’s efforts to overcome the shock, i.e., to regain mastery of this threat to its existence, determines some of the clinical features of the patient’s neurosis. Thus, obsessive preoccupation with the accident, nightmares repeating the accident (sometimes in dis-

7. Schaller, After Effects of Head Trauma, 113 A.M.A. 1779 (1939).
torted form), intense insecurity, dizziness, loss of confidence and poor concentration represent the ineffectiveness of the ego in controlling emotional excitation. As the ego becomes stronger, or as the traumatic event recedes in time, the obsessional preoccupation with the accident, the phobias against driving and the numerous complaints become less intense.

There are other relevant psychological aspects of traumatic neurosis that also require comment. The patient with traumatic neurosis spreads his feelings beyond his body to the environment. Thus, his symptoms are colored by his attitude toward doctors, insurance carriers, employers and society in general. The patient often projects his hostility to the driver of the vehicle which injured him or to the insurance company behind the negligent driver. Accompanying this hostility (which may occasionally be well-founded) is a dependence on the physician or lawyer handling his case. The traumatic patient is either cloying and insistent, or belligerent and disgusted. He addresses himself to the community because of his prolonged symptoms, making it clear that he is suffering through no fault of his own. One result of these feelings is that the patient represses knowledge of his own negligence in the accident, thus requiring careful sifting of facts by the attorney or medical expert during the exploratory interview.

These social extensions of psychological problems within the traumatized patient require careful consideration when judging the causation or precipitation of the neurosis. Such social extensions readily give rise to the notion that the patient is a fake, is malingering, or is interested only in monetary gain.

There is also the important question of the effect of the case's settlement on the traumatic neurosis or post-concussive state. The patient's various hostilities and dependence reactions alluded to above may make the patient seem more "greenback" minded during the pre-trial and trial period than he actually is. However, experience has shown that the persistence of symptoms has more to do with the underlying pathology than the amount of money received. The attorney must remember that he is dealing with a client whose psyche has been injured to a greater or lesser extent, a psyche which still has to deal with the problems of life with a less-intact ego and with fewer mental resources than were originally present.
Hysteria, Malingery, and Simulation

Dramatic reactions after trauma, either as neurosis or psychosis, raise the question of malingery and hysteria. This is a murky area for the reason that historically, medical examiners have been partial to finding malingery and/or hysteria where the results of trauma were not in accordance with strict anatomical expectations. If a doctor found “no physical evidence” of disease, he labeled the symptoms “hysteric” or “malingered.” A negative attitude on the part of examiners often determined the diagnosis. This negative attitude belonged to the era when neurologists handled cases now belonging in the realm of psychiatry. Thus Strauss and Savitsky, in a pioneer study of head trauma, (1934) commented on earlier attitudes, “The medical literature on the neurosis following trauma is tainted by a polemical undercurrent and an unwarranted hostility and antagonism toward the neurotic.”

This referred to the day when drastic methods were used to rule out malingery, such as allowing a blind man to walk into a wall to test whether he was feigning blindness. Although this rigid attitude has softened since that time, still the “mind set” of the person making the examination is almost as influential in determining malingery as is his analysis of the symptoms presented. The defendant or his agents will wish to prove malingery in neurotic symptoms; the plaintiff or his agents wish to demonstrate that the symptoms are valid. In the absence of objective signs, the psychological “set” of the examiner is obviously significant.

A distinction exists between hysteria and malingery: Hysteria represents a pathological process of the mind wherein painful or unacceptable ideas and emotions are excluded from consciousness. In hysteria, an emotionally charged idea is split off from consciousness and converted into a physical symptom, hence the term “conversion hysteria.” Examples of this mechanism, which is unknown to the patient, are blindness (to shut out a painful or distressing sight), paralysis (to make use of an arm or leg impossible), and staggering gait (without organic cerebral injury). It must be emphasized that in cases of functional loss of a part of the body, the patient does not consciously cause the symptom; it operates beyond his awareness through the unconscious mechanism of repression.

In malingery, a symptom is feigned or simulated. Thus, a malingering patient will act confused, mix up his statements to indicate poor comprehension, or will hold an arm in a bizarre manner to indicate paralysis. To the experienced observer, malingered symptoms tend to appear childish and exaggerated, even contrived. A malingered symptom then is a studied attempt to mimic the condition claimed.

Some competent authorities feel that there is an element of simulation in every neurosis. Thus, Dr. Schaller makes the point:

Every post-traumatic neurosis is not malingery but a sub-conscious simulator . . . Every hysteric is a simulator [representing] a milder implication of motive and conduct [than found in] the malingerer.\(^\text{10}\)

It is to be noted that Dr. Schaller presented this view in 1929. Since then, a more moderate approach has been taken. In his classical text Noyes writes:

Some hysterical phenomena are on the borderline between psychoneurotic reactions and simulation and therefore come close to malingering. Just where, as to awareness, the line between simulation and hysteria should be drawn is therefore often arbitrary.\(^\text{11}\)

Examination of the dynamics of simulation and neurosis leads to an understanding of the underlying emotional process. Malingery is the first step in denial of a reality the patient (or criminal offender) is unwilling to face. Indeed, denial is the first line of defense in any human action for which the perpetrator wishes to shed blame. After denial comes lying in words or behavior through minimizing the truth or exaggerating some aspects of it. The next stage is the concrete assumption of this denial through a bodily disorder—paralysis or other dysfunction—commonly called malingery. Later, this assumption of a malfunctioning organ can be hardened into a fixed neurotic symptom, thus betraying the subtle movement from denial to repression through the unconscious processes of the human ego.

The following case, while just outside the limits of malingery, still illustrates the mental mechanisms discussed above:

Clinical Case #7. The patient, born in Virginia, was able to write and read to a limited degree. His Army experience lasted only six months, most of this time being spent under medical observation for "stomach trouble." In March, 1960, the patient was injured while a passenger in a bus involved in

\(^{10}\) *Supra* note 7.

a rear-end collision. He immediately felt pain in the neck posteriorly radiating down the back to the left leg. He was not unconscious, being able to walk to the ambulance and talk to the officer and driver. Examination at the hospital revealed contusion of the neck and head, the anterior chest and low back. Subsequently, during the next six years, the patient was treated many times by physicians with no improvement of his symptoms. Finally, in January, 1966, he was diagnosed as having a borderline schizophrenia with neurasthenic and conversion manifestations precipitated and increased by the accident of 1960.

The patient complained of pain in the neck and back of head, aches along the spine radiating to the left buttock, thigh and calf, aches in both shoulders, pains across the chest, numbness of the hands, insomnia due to pain, general discomfort and a feeling that his "life was just messed up." He acknowledged that he was distraught and upset, at times agitated, by his lack of improvement.

Neurological examination was entirely negative. The mental examination revealed an attitude consistent with his condition, lack of improvement and his unhappiness because of the "ache in the center of his back."

Reconstruction of this patient's mental condition while in the Army points towards a neurotic condition, most probably conversion hysteria. In any event he had made an adequate adjustment until the injury of March, 1960.

The main problem here was the conversion hysteria following a whiplash and lumbosacral sprain. The excellent muscle tone and absence of spasm in the back, neck and shoulder muscles preclude a continuing myositis. The whiplash effects were long since absent. The deep resentment at his fate, characteristic of those injured "in the rear" (as this patient himself pointed out), accentuated the basic hysteria present. It was not a conscious malingery.

The more usual cases of malingery feature a bald demonstration of neurological dysfunction which is contrary to anatomical possibility. Thus, a patient may indicate he cannot move one finger but is able to move the others which are served by the same nerve or muscle. Or, he may claim loss of sensation which is patently impossible in view of the anatomical nerve supply. A common type of malingery (less often seen nowadays than in the past) is the epileptic "fit." Here, the patient flails one arm, or jerks a part of his body entirely unlike the true epileptic seizure which starts with a clonic spasm of both arms and legs, then advances to tonic jerks which are rhythmic and roughly symmetrical while the patient is totally unconscious. In recent years, this type of gross malingery has become uncommon partly because of the spread of medical information among the general population.

**Functional Overlay**

A frequent addition made to a diagnosis in a medical report is that of "functional overlay," a phrase designed to indicate excessive
complaints over a relatively minor physical finding. The phrase implies that the patient has let his symptoms overwhelm him, permitting them to assume larger proportions than would the medical examiner experienced with such conditions.

In contrast to hysteria and malingery (or simulation) where no pathological condition exists at all, functional overlay indicates there is also some evidence of disturbance in the tissues, perhaps evidence of a muscle spasm, tenderness of a bone or joint, or moderate involvement of a nerve. Technically, the term, "functional overlay," covers the over-valuation of a symptom; it lies in a borderline area between simulation, hysteria and pre-occupation with subjective complaints.

The psychology of over-valuation of a symptom is perilously close to that of a normal reaction, hence it is difficult to establish its compensability. One has to look carefully into the personality and psychological background of the plaintiff in a personal injury case where the examiners claim he has over-valued his symptom. In a sense, all persons over-value their bodies and its deficiencies. Thus, if a person gets a foreign body in the eye, the disabled eye is all-engrossing to the sufferer. So too, an accident victim is automatically forced to devote his complete attention to his injuries until they reduce in intensity. When this reduction does not occur, one can speak of an over-valued complaint. The persistence of this concern may be related to the patient's covert feelings of resentment, frustration at being unable to get rid of his disability (as in many low back pain cases), or feelings of humiliation that he was even injured at all.

This pattern comes to the fore with particular persistence in rear-end collision cases. Here, the patient's paranoid sensitivity is mobilized, especially if the accident occurred without warning. Being attacked from the back (as in case #7) stimulates feelings of betrayal, of undue hurt and unfairness. Where the patient's fears persist far beyond the time anticipated that the post-concussive or whiplash symptoms would usually clear up, we are dealing with an over-valued complaint, or even an activation of an inferiority or other neurotic state, rather than malingery. In fact, most of the phobic reactions following trauma to the head, especially those incurred in automobile accidents, dip into the psychology of the victim, reactivating old fears and anxieties ordinarily hidden from consciousness.
The psychological assault associated with an accidental injury cannot be divorced from the patient's basic fears—which we all share—of unexpected and unanticipated blows, with their implied threat of destruction and death. Indeed, the total thrust of neurosis after trauma has to do with fears of annihilation, whether the fears are controlled to some degree or not. This is the reason for the frequent catastrophic or anxiety dreams which accident victims experience. The dream functions as a safety valve to gradually channel off the fears of destruction unloosed by the trauma itself.

The following case demonstrates some of these psychological elements:

**Clinical Case #8.** A woman of 35, while stopped at a traffic light, was struck from behind by a truck. She was unaware of the impending collision until it happened. The patient suffered from a cranio-cervical syndrome (whiplash) and a mild secondary concussion. Several months later, as the muscle pains, stiffness of the neck, headache, blurred vision and other complaints gradually cleared up, she began to experience severe anxiety attacks when riding in a car. Her particular fear of being struck in the rear of the car, forced her to sit (as passenger) facing the driver so that she could observe both the road before and behind the car. Her phobia became so disturbing that she was unable to ride in a car without sedation and even then with great difficulty.

Careful study of the case revealed that she had a sexual experience early in life in which an attack of rape had resulted in an illegitimate child. There were some threats of anal intercourse at the time. The "rear-end" collision had reactivated her earlier trauma of attack "from the rear."

The patient's psychological reaction to being struck in a rear-end collision does not necessarily dip into the psycho-sexual areas of the patient's mind. More usually, the shock of the impact and its seeming wantonness, as in the case of alcoholic drivers unable to stop their vehicles, induces a strong reaction of anger in the victim. As this reaction fades, a sense of humiliation develops which often, especially in female victims, colors the post-traumatic neurosis symptoms.

**Compensation Neurosis**

The problem of "compensation neurosis," or "greenback neurosis" as it is sometimes facetiously called by defense attorneys, merges with the reactions discussed above when viewed from a psychological standpoint. In these cases, the injuries can be substantial, or they may be trivial; even so, some defendants are loud in their claims for
financial restitution or even retribution. Still, one cannot say that the financial settlement is their only goal. Nor is it true that sudden cures occur in most cases after a financial settlement has been reached. Frequently, after a court settlement is reached the complaints centering around a post-concussion syndrome or a traumatic neurotic condition persist for years.

It is necessary to point out that this view is not universally held among medical experts. Thus, one competent psychiatrist feels, "... it is almost impossible to offer meaningful psychotherapy to a patient suffering from compulsion neurosis prior to settlement of litigation." In spite of this attitude held by some, the question arises whether it is correct to say that "compensation neurosis" exists at all as a clinical entity. At best, the condition is a neurosis with strong dependency features and, at worst, an insurance company's slogan. The situation behind "compensation neurosis" is compounded with complaints from the injury, resentment, and unconscious dependency elements, as was described above under traumatic neurosis. The psychological attitude toward the "company" on the part of the injured client is crucial. As some authors put it, "... the injured patient relates to the Company as a gigantic parent figure at whose hands she could not tolerate frustration . . . ." To return to the thesis that financial settlement does not always settle the patient's problems, one should consider accidents involving older persons where the likelihood of continuing in gainful occupation or in enjoying their "retirement" without disability or disabling symptoms is markedly reduced by the injury.

In this connection, comment should be made on the so-called "secondary gain" from illness. This is a recognized neurotic reaction in which the symptoms suffered convey the advantages of being cared for, "babied," freed from responsibility, or thrust into the center of attention, by virtue of the medical condition. Every person can be shown to experience this secondary gain, usually in minute degree, i.e., every patient from any cause wishes to be regarded as an unfortunate victim of fate, thus extracting sympathy from his or her caretakers, medical attendants and family. In head injury cases, this

tendency is even stronger because of the over-valuation of the head 
(the governing intellect) and because (and this is the important fac-
tor) the injury was inflicted by another person rather than by na-
ture, one's own neglect, poor heredity or an epidemic of some febrile 
ilness.

The secondary gain in head injury patients has the function of 
holding society to blame, in addition to the person or equipment that 
caused the blow; the patient considers himself entirely without fault. 
This attitude, while partly a conscious one, invades the psychology 
of the plaintiff to the point where all symptoms and disabilities are 
intensified. This is particularly true when the injury occurs on a 
common carrier such as a train, bus or plane. In such cases the 
"attacker" is an impersonal corporation operating the vehicle through 
the engineer, driver or pilot. The reaction of anger cannot be easily 
fixed on one person, hence it spreads from the vehicle involved in the 
accident to the corporation behind it. The lack of specific goals for 
feelings of retaliation force themselves back onto the ego of the vic-
tim, a mental mechanism technically called "introjection," wherein 
anger floods the victim, adding to the intensity of the actual physical 
symptoms.

The secondary gain in such cases then revolves around the victim's 
hostile feelings towards the corporation behind the common carrier. 
These feelings simultaneously bolster the symptoms and increase the 
need to make the company pay for pain, disability and the indignity 
for which the victim is blameless. The following is an example of this 
mechanism:

Clinical Case #9. The patient, a New York woman on vacation in California, 
was riding on a Greyhound Bus enroute to Los Angeles in 1962. A truck struck 
the bus, shearing off the front section, and killing two passengers sitting just in 
front of the patient. She was a woman of fifty, previously well-integrated, intelli-
gent and physically and mentally well. She was apparently unconscious for an unde-
termined period. Examinations by several physicians and studies in several hos-
pitals resulted in diagnoses of brain concussion, lacerations and contusions, and 
severe cervical sprain (whiplash). The patient's complaints were numerous—pain 
in temples, numbness and pain in head and neck, head noises, nausea, insomnia, 
nervous and irritable, concentration difficulty, fatigue, dizziness, poor memory, 
photophobia, and hyperacusis.

Examination by competent neurologists agreed on the traumatic neurosis, brain 
concussion and whiplash. A later examiner, six months after the accident, agreed 
with the diagnosis of traumatic neurosis. The patient was given intensive treatment 
with medications and physical therapy for about two years.
Two and a half years later her pain and discomfort worsened. She was troubled with disturbed "balance in hands," muscle jerks at night, startle reactions and nightmares (once and twice a month), and return of "repressed" anger against the bus company. She described her condition as feeling "draggy, with no initiative." She was in constant discomfort. She summed it up by saying "I live in a world, scared of everything." At the last examination, three years after the accident, there was some improvement in neck distress, but persistence of many symptoms. Mental complaints such as tension, shakiness, lack of confidence, impatience, and forgetfulness remained as before, especially since the patient discovered she had been "tailed" and photographed by the Pinkerton Agency, for the Greyhound Bus Company.

Litigation was prolonged, necessitating the patient's travelling from the East to the West Coast on several occasions. Her nervous state improved very little after a judgment in her favor at the trial. Four years after the accident she showed a tense, nervous, and anxious state, classical for post-concussive symptoms even down to the decreased sexual libido, memory decrease, dizziness, photophobia, insomnia, etc. The patient's complaints of changed personality with decreased initiative, lack of confidence, impaired social adjustment, draggy feelings and constant preoccupation with symptoms all appeared valid.

_Ego Shock and Recoverable Mental Anguish_

The problem of anxiety and panic reactions arising under the umbrella of secondary traumatic mental disorders (hysteria or traumatic neurosis) raises the question of compensability of mental anguish suffered by the victim in a negligence case. Many courts have previously refused to allow recovery where the physical consequences complained of resulted solely from the internal operation of mental and emotional stresses, unless there was, coincident in time and place with the stress-producing event, some physical impact which also resulted directly from defendant's negligence. Although this rule has been rejected by a large number of courts, it is still in force in many jurisdictions.

For example, some states continue to follow the doctrine of _Mitchell v. Rochester Ry._, which denied recovery for mental distress without physical contact. In that case, the court expressed the concern that claims for mental distress were too easily faked to permit compensation.

In _Mitchell_, the plaintiff was standing in a cross-walk waiting to board defendant's horse-drawn streetcar. As the team attached to the car drew near, they suddenly veered to the right so that they

came close to plaintiff, although not touching her. When the horses stopped, plaintiff was left standing between the horses' heads. She testified that from the fright and excitement caused by the approach and proximity of the team she became unconscious, later suffering a miscarriage and consequent illness.

In denying recovery, the court stated:

Assuming that the evidence tended to show that the defendant's servant was negligent in the management of the car and horses, and that the plaintiff was free from contributory negligence, the single question presented is whether the plaintiff is entitled to recover for the defendant's negligence which occasioned her fright and alarm, and resulted in the injuries already mentioned . . . . Assuming that fright cannot form the basis of an action, it is obvious that no recovery can be had for injuries resulting therefrom. That the result may be nervous disease, blindness, insanity, or even a miscarriage, in no way changes the principle . . . . If the right of recovery in this class of cases should be once established, it would naturally result in a flood of litigation in cases where the injury complained of may be easily feigned without detection, and where the damages must rest upon mere conjecture or speculation.\textsuperscript{15}

A more recent Pennsylvania case, \textit{Bosley v. Andrews},\textsuperscript{16} also denied recovery to a plaintiff who suffered a heart attack from fright caused by defendant's bull charging at him. The court held that there can be no recovery of damages for injuries resulting from fright or nervous shock or mental or emotional disturbances or distress, unless they are accompanied by physical injury or physical impact.\textsuperscript{17}

The ridiculous extremes to which courts which followed the "impact rule" were put in justifying recovery for mental suffering and anguish is well illustrated in \textit{Christy Bros. Circus v. Turnage}.\textsuperscript{18} In this case, the court held that a valid cause of action was stated by plaintiff, an unmarried woman, who, while a guest at defendant's circus, suffered embarrassment, mortification, and mental pain and suffering when one of the performing horses was backed toward her and evacuated his bowel in her lap to the great amusement of the surrounding crowd.

It is probably the accepted rule in a majority of jurisdictions that where definite and objective physical injury is produced as a result of emotional stress wrongfully caused by defendant, he may be held

\textsuperscript{15} \textit{Id.} at 109, 110, 45 N.E. at 354.

\textsuperscript{16} 393 Pa. 161, 142 A.2d 263 (1958).

\textsuperscript{17} \textit{Id.}

\textsuperscript{18} 38 Ga. App. 581, 144 S.E. 680 (1928).
liable for such consequences notwithstanding the absence of any physical impact on the plaintiff at the time of the mental shock.

The Restatement of Torts provides:

"If the actor's conduct is negligent as violating a duty of care designed to protect another from fright or other emotional disturbance which the actor should recognize as involving an unreasonable risk of bodily harm, the fact that the harm results solely through the internal operation of the fright or other emotional disturbance does not protect the actor from liability, [and] if the actor's conduct is negligent as creating an unreasonable risk of causing bodily harm to another otherwise than by subjecting him to fright, shock, or other similar and immediate emotional disturbance, the fact that such harm results solely from the internal operation of fright or other emotional disturbance does not protect the actor from liability."

The rule allowing recovery for the physical consequences of mental suffering without actual impact has been subjected, in some cases, to the limitation that there can be no recovery except by those who were within the area of physical risk from defendant's negligent act. For example, in Orlo v. Connecticut Co., the court said a jury question was presented where it was shown that defendant's electric wire negligently dropped on a car in which plaintiff, a passenger, sat for several minutes with wires flashing and hissing around the car. Although there were no burns or marks on his body, plaintiff suffered severe nervous shock and severe fright causing him to tremble and shake for about a month and requiring his hospitalization for aggravation of certain pre-existing physical disabilities.

This "area of physical risk" or "zone of danger" rule has probably had its greatest prominence in those cases where recovery for shock or mental anguish is sought by a plaintiff who feared or witnessed actual or threatened harm to another person. However, the courts appear to be expanding the circumstances and conditions for granting recovery for emotional trauma to witnesses of distressing events, even though they were personally not in danger.

The furthest move so far in that direction came from the California Supreme Court in Dillon v. Legg. In this case, a mother saw her infant daughter killed by a car as she crossed an intersection and she brought suit for her own emotional trauma at witnessing the accident. The lower court granted the defendant's motion to dismiss the

19. Restatement (First) of Torts § 436 (1934).
20. 128 Conn. 231, 21 A.2d 402 (1941).
suit on the basis that under prior decisions no cause of action is stated unless a plaintiff's shock has resulted from fear for his own safety. In reversing this decision, the California court stated that the mother's great physical and mental shock and suffering at witnessing this traumatic event was a proper subject for damages since her fear, shock, etc. should have been reasonably foreseeable to the defendant when his negligence was directed to a third person.

The court also set down three factors trial courts should take into account to determine whether a defendant owed a duty to or should reasonably foresee emotional injury to a witness of an injury to a third person:

(1) whether plaintiff was located near the scene of the accident, as contrasted with one who was a distance away from it; (2) whether the shock resulted from a direct emotional impact upon plaintiff from his sensory and contemporaneous observance of the accident, as contrasted with learning of the accident from others after its occurrence; and (3) whether plaintiff and the victim were closely related, as contrasted with a distant relationship or none at all. In light of these factors, the trial court would then determine whether the accident and emotional harm to witnesses was reasonably foreseeable.

Compensable Emotional Disturbance in Industrial Accidents

Workmen's compensation awards for neurological or psychological difficulties resulting from job-related injuries parallel those for non-industrial accidents. The legal difference, of course, is that workmen's compensation statutes reject the common law defenses of contributory negligence, fellow-servant rule, and assumption of the risk, in favor of a general theory of employer's liability without fault.

In recent years, the area of industrial accidents has been extended from the obvious injuries of arms, legs, head and sense organs to the more obscure psychiatric and psychological difficulties, or as they were earlier referred to by the courts, "intangible impairments." There appear to be two main types of "intangible impairments" cases: one involves nervous (neurotic or psychotic) symptoms following actual injuries to the head or any other part of the body; the other includes mental disorders in the absence of impact.
In *Ladner v. Higgins, Inc.*, plaintiff fell about eight feet from a scaffold which in turn struck him on the shoulder. Although there was no permanent bone pathology, the court held the plaintiff was unable to work because of a disabling traumatic neurosis and affirmed the award.

However, in *Miller Rasmussen Ice and Coal v. Industrial Commission*, a Wisconsin court set aside a workmen's compensation award because the plaintiff's physical injury was so slight and his pre-existing neurotic condition was so obvious.

The second group of compensation cases involves no physical impact, but rather considers the psychic stress of plaintiff's ordinary work as the proximate cause of his resulting nervous condition. The case of *Burlington Mills v. Hagood*, voided the impact rule in compensating for a traumatic neurosis. In that case, plaintiff saw a flash of a short-circuited electric motor fifteen feet away and fainted, although a co-worker caught her before she fell. Afterwards, she was unable to work because of a traumatic neurosis. The court said,

We are fully aware that in tort actions we have followed the common-law rule that there can be no recovery for mental anguish unaccompanied by physical injury, and of the reasons for the rule. The rules of the common-law for tort actions, however, do not apply to cases under the Workmen's Compensation Act. Under the Workmen's Compensation Act, the proceeding is not one for damage for a wrong done, but to obtain compensation for a loss sustained by reason of disability. . . . In the instant case, the disability of Mrs. Hagood was occasioned by an injury which may be fairly traced to a risk which arose out of and in the course of her employment. There was a direct causal relation between the electric flash and the irritated condition of her nervous system.

The farthest extention of the doctrine permitting recovery for non-impact-related mental illness caused by stress or emotional pressure in a work situation can be found in *Carter v. General Motors*. In this case the plaintiff, who had been hospitalized on previous occasions, worked on an assembly line job to which he could not adapt. If he followed regular factory procedure in removing burrs from wheel assemblies one at a time, he fell behind the normal pace of the line and was criticized. If he attempted to work on two wheels

---

22. 71 So. 2d 242 (Ct. App. La. 1954).
23. 263 Wis. 538, 57 N.W.2d 736 (1953).
24. 177 Va. 204, 13 S.E.2d 291 (1941).
25. *Id.* at 210-11, 13 S.E.2d at 293.
at once, he sometimes mixed them up and was again criticized—a classic "double bind" situation. After twelve days of this dilemma, plaintiff suffered an emotional collapse (he was diagnosed as schizophrenic, paranoid type), and was hospitalized for one month. Although ruling out a permanent disability award, the court granted plaintiff compensation for his temporary emotional disablement plus medical and hospital care.

Some concern has been expressed that compensation awards may soon become the rule for any employee who suffers some psychological difficulties from work pressures even though the pre-morbid personality is disposed toward emotional disorder. Arguably, the availability of compensation may increase the rate of psychoneurotic reactions. As we have seen above, however, many different external and internal factors may precipitate a psychoneurosis. Little psychiatric evidence is available, however, to show whether or not the promise of financial gain can provide a neurosis-producing motive, although external benefits such as sympathy, attention or compensation may unconsciously prolong the reaction or heighten its intensity.\textsuperscript{27} Two basic reasons are presented by psychiatrists to explain the significance of such factors. First, the external benefits may help the individual to adapt to his psychoneurotic reaction. The receipt of compensation may satisfy dependency or aggressive needs. It may also reduce financial worries resulting from the psychoneurotic reaction, thus reinforcing the effectiveness of the reaction as a conflict-reducing device. Second, many psychiatrists accept the view that such external benefits may constitute advantages which are independent of those associated with the release of psychic tension. A desire to retain these bonuses may unconsciously motivate the individual to prolong the psychoneurotic reaction once it has arisen.

**CONCLUSION**

Because of the many possible psychological ramifications of traumatic injuries, the attorney handling a personal injury case should be sure that a thorough review is made of the emotional, neurological and psychiatric difficulties his client is experiencing. The viewpoint of the examiner should be objective and should consider the deeper emotional consequences of the traumatic event.

\textsuperscript{27} Alexander, Fundamentals of Psychoanalysis 212 (1948).
It is also important to remember that even though objective physical and neurological signs are absent, this does not mean the accident victim is not suffering from a genuine psychiatric disability. The literature is replete with examples of patients whose emotional wounds continue to plague them long after their physical injuries have or should have healed. A personal injury client may require psychiatric treatment in addition to strictly medical care in order to cope with the emotional difficulties arising from his traumatic accident and because of the influence of psychiatric problems on physical well-being.

The attorney, as well as the medical examiner, should treat each case on an individual basis. Every client has his own particular ego strengths, personal weaknesses, cultural background, family situation and psychological resources with which to meet his problems. The symptoms of different persons may sound similar, but justice (and medicine) can be satisfied by nothing less than a thorough study and evaluation of each individual case.