



# NEUROTOXICOLOGY

# Rx Law & Medicine Report

57

VOLUME 1, ISSUE 2  
WINTER 1998

62666 11

Medical Malpractice Damages and the Plaintiff in a Persistent Vegetative State (PVS): What Is the Appropriate Measure of Damages? by <i>Miles J. Zaremski</i> .....	1
Physicians' Perceptions of Managed Care by <i>Robert A. Levine and Alan Lieberson</i> .....	1
— The Clinical Neurological Assessment of Environmental Toxicity by <i>Peter G. Bernad</i> .....	3
An Attorney's Guide to the Medical Record by <i>Elliott B. Oppenheim</i> .....	5
Why do Football Players Learn Ballet? by <i>Fred D. Heckman</i> .....	7

## MEDICAL MALPRACTICE DAMAGES AND THE PLAINTIFF IN A PERSISTENT VEGETATIVE STATE (PVS): WHAT IS THE APPROPRIATE MEASURE OF DAMAGES?

**Miles J. Zaremski**

Any counsel versed in the defense of high-damage medical malpractice lawsuits is no doubt well-acquainted to the typical array of components of a plaintiff's claim for damages. Frequently, plaintiffs ask juries to render independent determinations as to the cost of the injured person's past medical and life care expenses, future medical and life care expenses, lost earning capacity, past pain and suffering, and future pain and suffering. Depending on the jurisdiction of the case, defense counsel may additionally face claims for loss of aid, comfort, society and companionship asserted by spouses, parents, siblings, etc. Because of these elements of a plaintiff's proof, defense counsel will no doubt line up experts to refute liability, and then, should the jury find against the client on liability, defense counsel additionally offers a series of experts to opine that plaintiff's damages experts have simply overstated the injured plaintiff's future medical and life care expenses, future earning potential and past and future pain and suffering.

(Continued on Page 22)

## PHYSICIANS' PERCEPTIONS OF MANAGED CARE

**Robert A. Levine and Alan Lieberson**

In this era of rapid change in health care delivery, with an alphabet soup of organizations (HMOs, PPOs, IPAs, PHOs, MSOs) interacting with providers under new and often ambiguous guidelines, we set out to discover through a questionnaire sent to the Norwalk Hospital medical staff how practicing physicians viewed the rules and regulations of managed care. We were particularly interested in physician views on the legal and ethical implications of various controversial provisions frequently included in managed care contracts as these views might provide information for the Connecticut legislature in developing the proposed Act Concerning Managed Care of 1997.

Unlike surrounding hospitals in Fairfield County, Norwalk has a history of resistance to the expansion of managed care plans (MCPs) into the community, and still maintained a comparatively high percentage of fee-for-service patients at the time the questionnaire was distributed, although this percentage was diminishing rapidly.

Although the response rate was not high, it was evident that the responding staff physicians not only considered many of the contractual provisions addressed in the questionnaire to be unethical, but also believed them to be illegal. To check whether these

(Continued on Page 2)

## THE CLINICAL NEUROLOGICAL ASSESSMENT OF ENVIRONMENTAL TOXICITY

Peter G. Bernad

### *General Toxicity*

Biological toxicity is the ability of a chemical or compound to damage a biological system. The most common routes of exposure in human toxicity include inhalation, dermal absorption and ingestion. Due to the volatile nature of most neurotoxins, the most common exposure route is inhalation. However, deliberate ingestion and accidental immersion in toxins are also known to result in neurologic damage.<sup>1</sup> After inhalation, solvents are rapidly absorbed into the blood stream. Many neurotoxins are highly lipophilic, demonstrating a natural affinity to bind with lipids (fatty tissues). Neurotoxins tend to accumulate in the fatty tissues of animals, which explains why adipose tissue biopsies and pathologic examinations can be useful indicators of toxic exposure. The high solubility of solvents in lipids combined with the high lipid content of the nervous system creates a medium for significant morbidity and potential mortality. The central nervous system (brain and spinal cord) and the peripheral nervous system (peripheral nerves, sensory and motor nerves) are composed in large part of lipid tissues. Toxins will therefore accumulate in the nervous system. This lipophilic tendency of toxins is bolstered by the rich blood supply the circulatory system delivers to the brain, an

additional cause of serious neurologic effects.

### *Overview of Symptoms*

Neurologic symptoms of toxic exposure are specific to each neurotoxin. Solvents, among the most prevalent nervous system toxins, may induce euphoria followed by confusion, tinnitus, disorientation, blurred vision, analgesia, headaches, nystagmus and ataxia. Neurobehavioral problems from solvent exposure include memory loss, decreased attention, altered personality or mood, reduced psychomotor functioning, impaired dexterity and hand-eye coordination, altered reaction time and decreased problem solving ability.

### *Diagnostic Evaluation*

There are four essential elements to diagnosing neurologic injury with toxic exposure etiology:

1. Identification of the event precipitating the onset of symptoms (e.g., a spill);
2. Generation of a hypothesis to explain the relationship between the precipitating event and the subsequent symptoms;
3. The diagnosis must include a description of the precipitating event, optimally with quantification using industrial hygiene data or other natural factors; and
4. The diagnosis must include clinical assessment of the suspected association between exposure and organ or system dysfunction in the patient.

### *Medical/Neurologic Examination*

**Questionnaire.** A comprehensive questionnaire should be given to the patient<sup>2</sup> who should be asked about personal habits, hobbies, and occupational duties as background for the evaluation of potential exposures. Past medical history should be obtained to assess the potential for underlying systemic disease or prior neurologic injuries. The history should also consider socioeconomic factors and potential nutritional deficiencies with possible neurologic implications. An occupational history should be taken to explore the timing of symptoms relative to work exposure and determine whether symptoms or illness have occurred in coworkers. A description of all job titles and types of work performed is helpful and should be obtained whenever possible.

**History.** A complete history should be taken as soon as possible after neurotoxic exposure occurs or is suspected to have occurred. The history should include medical, personal and occupational information, as well as data on symptomatology and particularly observable abnormalities such as tremor, convulsions, vertigo, seizure history, and weakness. It is important to document the main complaint and the onset of the present illness.

**Medication History.** All former and current prescribed and over-the-counter medications should be evaluated. Toxic dementia can be associated with or caused by a large number of legally prescribed medications.

Antipsychotics, hypnotics, sedatives, anticonvulsants, anticholinergics, and various pain relievers can produce encephalopathy. Higher cortical functioning may be impaired, altered, or diminished by benzodiazepine use. Chronic use of anticonvulsants will cause subtle central nervous system effects.

**Personal, Avocational and Social History.** Personal history should include an investigation of the patient's living environment and careful screening for illegal drug use. Occult exposure to carbon monoxide will result in headache and nausea, and drugs such as opiates will also lead to neurotoxic effects. Hobbies, such as modeling, gardening, or painting can also expose the individual to known neurotoxins. A careful examination must include questions examining these potential sources of toxic exposure.

**Physical Examination.** A complete physical examination and neurologic examination should be conducted. Vital signs, weight, and blood pressure should be recorded. The head, neck, eyes, nose and throat should be examined. Special attention should be paid to the skin, lymph nodes, liver and spleen. The neurologic examination should evaluate higher cortical functioning, as well as brain stem functions. Examination of higher intellectual functions includes determining orientation to time, place and person. The neurologic examination should evaluate memory, apraxia, agnosia (visual, auditory and tactile), aphasia, cranial nerves, visual acuity, motor systems, reflexes, sensory systems, and cerebellar functioning (posture, gait, ataxia). Clinicians should also assess the patient's overall mental and behavioral state.

### *Laboratory Diagnostic Procedures*

**Routine Blood Work.** Routine blood work should include a complete blood chemistry profile and urinalysis. There are also some specific analytic tests for particular neurotoxins that the clinician may suspect are involved, including screening for metabolites of solvents in blood or urine. Liver function tests can indicate whether this organ is being stressed by efforts to remove toxins from the circulatory system.

**Structural Imaging.** Many diagnostic techniques are available to evaluate structurally and functionally the central, peripheral and autonomic nervous systems. Structural evaluation of the brain may be indicated in some patients. Structural imaging can be accomplished with computed tomography (CT or CAT), magnetic resonance imaging (MRI), or positron emission tomography (PET). Chronic exposure to solvents is known to result in cerebral atrophy, which can be demonstrated on CT scanning.

**Electroencephalographic Examination.** The functioning of the central and peripheral nervous systems can be evaluated by electromyography (EMG), nerve conduction velocity testing, electronystagmography (ENG), audiologic testing, and balance evaluation. The autonomic nervous system can also be evaluated by pupillometry, electrocardiographic R-R peak interval and sweating evaluation. Neurometric techniques can be quantitative or semi-quantitative. An electroencephalogram provides a classic recording of the brain's elec-

tric activity, and is discussed in greater detail below. Evoked potentials or evoked responses can be recorded using visual, auditory, somatosensory, cognitive, or brain stem stimulation. Semi-quantitative sensory evaluation measures current perception threshold, vibratory perception threshold, and thermal perception threshold.

Electroencephalograms are a functional measurement of the brain's electrical activity. Electroencephalograms are different from traditional magnetic resonance images or computed tomographic images of the brain in that the latter provide morphological information, structural or anatomic details, whereas the former provide information concerning the functional electrical activity of the brain. Computed tomography of a dead person may be viewed as "normal," but such a person's electroencephalogram would be highly abnormal. Electrodes placed in specific positions record the brain's electrical activity and plot this information in wave form on polygraph paper. These polygraphs are read or interpreted by physicians with specialty training, and can offer valuable information regarding the potential organicity of neurologic or psychiatric complaints of subjects.

Quantitative electroencephalography ("qEEG"), which is also known as Brain Electrical Activity Mapping, EEG Brain Mapping, or EEG Topography, involves electroencephalographic frequency analysis, topographic display, and statistical comparisons with a normative database of data received from evaluating a

(Continued on Page 10)

## AN ATTORNEY'S GUIDE TO THE MEDICAL RECORD

**Elliott B. Oppenheim**

### *Introduction*

The ability to spot medical inconsistency is the key to understanding the medical record in forensic litigation. In order to detect departures from the standard of care, one must be able to find these departures: medical inconsistencies signal departures from the standard. While one need not be a physician to spot them, a physician can do it with much more ease and authority than an attorney. Yet, by using the step-by-step approach described in this article, an attorney — any attorney — will be able, at a minimum, to extract a medical record from all medical records, make a notebook of them, and present the record to experts. Here are my secret steps for medical record review based upon over twenty years in medical negligence litigation.

Whether your case is a medical malpractice matter or one where the significance of the medical records remains at the evidentiary periphery, you will best serve your client if you approach the record analysis as if you were an archeologist, with an orderly and objective eye. You must know what should be in the record, what may be missing, what the record really means, and what the record fails to state, leaving much to the wary reader's curiosity. This article primarily relates to medical negligence litigation, but the principles remain the same in all other areas where the physical or mental condition of a patient is at issue.<sup>1</sup>

### *Step 1: Obtain All Medical Records*

Always have the client obtain all medical records created within the period covered by the statute of limitations or from any time when a record may have even remote relevance. In a medical negligence action particularly, have the client do this him or herself, or if with your help, without using your letterhead or authority. Physicians' offices, pharmacies, hospitals, and laboratories generally do not charge patients requesting records for their own use but will charge a dollar or two or more per page when an attorney makes the request, and it is not unusual for them to add administrative fees<sup>2</sup> of \$25 to \$75. When a client obtains his own record, the cost is less, and a claim may be confidentially evaluated without the providers being thereby notified that they are potential defendants. Later on, if litigation commences, you should compare every record the patient obtained with every record subsequently obtained in the discovery process. Discrepancies, alterations, and additions to the records then become embarrassingly obvious to the defendants.

### *Background: The Origin of the Medical Record*

Medical licensure creates for the practitioner a duty to practice medicine according to the standards of the profession.<sup>3</sup> The standard of care includes a duty upon the physician to generate a medical record. Litigating a medical negligence claim, then,

is a battle on standard-of-care turf where the medical record stands as a silent witness to past patient care episodes.<sup>4</sup> None of the sights, sounds, or feelings of any of the persons involved persist in perpetuity; the only reliable record of the incident at issue is the medical record.

When written contemporaneously, these records are highly reflective of what was done on the patient's behalf. They are "kept in the course of the regularly conducted activity"<sup>5</sup> and often constitute the best evidence of departures from the standard of care. During the course of a hospital stay, ten or more persons may make entries into the records in varying capacities. Several years later, when the care is called into question, it may be very hard to find out not only who actually created a particular document but also what factual basis they used when making a given entry. Often, sub-agents have made important entries on the record, memorializing events that they learned about vicariously. For instance, after a cardiac arrest, a senior resident may delegate the note-writing task to the most junior person on the team, a medical student or a first-year resident. That sub-agent enters his inexperienced impressions, but the impressions of the senior resident are forever lost.

Physicians have tried to keep medical records from patients to avoid suits. At one time this approach was upheld<sup>6</sup> in some courts but, fortunately for medical consumers, the generally followed modern rule is different. Records are available as a result of legislative action and court decisions.<sup>7</sup> Records actually belong to the

(Neurology, cont'd from Page 4) population considered "normal" in all aspects and which is age and sex matched to the individual being examined. The three broad uses of qEEG are:

1. *Detection of Organicity:* qEEG can provide objective measures that aid in the search for global or focal abnormality which, if present, may signal an underlying organic process. In qEEG, this search is extended beyond visual EEG inspection by the use of spectral analysis, long-latency event-related potentials, and the significance probability mapping (SPM) process. Applications include, but are not limited to, detecting organic vs. primary illness (e.g., depression vs. dementia), as well as epilepsy, cerebrovascular disease, learning disability, head injury,<sup>3</sup> and headache;
2. *Categorization of Disease or Clinical Condition:* Using discriminant functions (classification rules), patients may be classified as having one or more specific diagnostic conditions on the basis of their qEEG data (i.e. unipolar vs. bipolar depression, mild head injury vs. normal, dementia vs. depression); and
3. *Dipole Source Localization Methodology (DLM) Algorithm:* The goal is to determine the site of origin of epileptiform discharges. Typically, a finite set of comparable spikes is selected and averaged by synchronizing at the time of peak amplitude. The scalp distribution at the averaged spike peak is entered into a DLM algorithm designed for use in a hypothetical three-or-four sphere

simplified head model. The adequacy of the result is often inferred by assessing the residual spatial variance between the original scalp distribution and the scalp distribution calculated by the assumed source at its proposed location.

It should be remembered that qEEG is not a substitute for standard EEG, but a complex methodology that incorporates visual EEG inspection and serves to extend the clinical usefulness of data recorded from the scalp. Criticisms regarding its clinical value (i.e. color maps are deceptive, digital processing lacks sufficient detail, SPMs involve too many statistical tests, qEEG is overly sensitive to artifact) have primarily arisen from its misapplication and misinterpretation stemming from inadequate training and expertise of personnel. Accordingly, proper qEEG application must be performed by clinicians with demonstrated competency in standard EEG and specialized training in qEEG.<sup>4</sup>

There are early animal studies that have reported excessive beta activity in the brains of monkeys and humans following toxic exposure detected on the quantitative electroencephalogram.<sup>5</sup> In a study published in June of 1992 in the journal "Electroencephalographic and Clinical Neurophysiology" by Dr. Jonkman and associates of the Weseinde Hospital, The Hague, The Netherlands, Dr. Jonkman and associates reported on the quantified electroencephalograms of workers occupationally exposed to known solvents and organophosphorus pesticides. Among their findings, Dr.

Jonkman reported increased beta activity when compared to normal controls.<sup>6</sup> Electroencephalographic studies have also been used for the detection of subtle toxic-agent-induced dysfunction, and are an integral part of Russian and European neurotoxicological studies.<sup>7</sup>

### *Differential Diagnosis of Neurotoxic Exposure*

The clinician must rule out other conditions that may mimic the symptoms of toxic exposure. The clinician must be aware of systemic conditions or aspects of the patient's medical history that may produce symptoms similar to solvent-induced toxic encephalopathy. For example, Multiple Sclerosis (MS) and Systemic Lupus Erythematosus (SLE) can produce the central nervous system and peripheral system findings typical of solvent exposure. However, conditions such as these can be ruled out with hematologic, immunologic, or imaging studies such as CT or MRI. These techniques can also rule out infection or tumors that may be the cause of symptoms. A history of head injury should be carefully evaluated, as many patients suffering from postconcussive syndrome may exhibit electroencephalographic and neuropsychologic results that are indicative of organic solvent exposure. Additionally, any history of excessive alcohol intake should be carefully investigated. A personal or family history of psychiatric disease should be excluded as an etiologic factor in the patient's current symptomatology, as should vitamin

or nutritional deficiencies, such as B12 deficiency, which can produce neuropathic or psychotic symptoms.

### *Exposure to Solvents*

#### *Acute and Chronic Effects of Solvent Exposure*

Acute effects of exposure to organic solvents consist of feelings of dizziness, lightheadedness and incoordination, and possibly short-term psychomotor impairment. Removal of the patient from the source of exposure is reported to result in total resolution of symptoms.

Chronic neurological and neuropsychological effects of solvent exposure symptoms include increased fatigue, irritability, depression and loss of interest in daily activities. There have also been reports of cerebral atrophy, deficits in learning and memory, reduction in psychomotor and short-term memory. As the severity of central nervous system (CNS) dysfunction increases, the likelihood of a reversal of symptoms becomes progressively less.<sup>8</sup> The peripheral nervous system may show peripheral neuropathy, which can be examined histologically or using electrophysiologic techniques.

Swedish workers diagnosed with solvent-induced toxic encephalopathy have been known to demonstrate CNS effects on psychometric evaluation up to five years after cessation of exposure. The most common symptoms among these workers were memory disturbances, difficulty in concentration, fatigue, lack of initiative, and mood effects such as irritation and depression.<sup>9</sup> In a

separate epidemiologic study comprising 295 men aged 57 to 75 years, those who had been exposed to mixed solvents over a period of 5 years or more, demonstrated significantly more complaints than in control groups of decreased concentration ability, memory difficulties, and headache in workers who had retired. It is important to note that this study was controlled for over-reporting of symptoms.<sup>10</sup>

#### *Biological Markers*

Surveillance of employees exposed to solvents may require measurements of chemical products in body fluids, usually blood or urine, occasionally breath. Timing of sample collection is essential to this determination. Biological markers are defined by the National Research Council as the measure of variations in biological systems or samples. A change may occur at the molecular level as a sign of exposure to a chemical with no clinical significance. For example, delta-aminolevulinic acid dehydratase enzyme inhibition is a marker for lead exposure with no known deleterious effects.

Markers can indicate potential or existing disease by showing changes in organ function. Certain laboratory tests of organ functions may be altered as a result of disease caused by exposure. Routine function tests, however, are not as a rule sensitive or specific enough to be of value in the detection of early pathology.

The term "risk assessment" usually involves estimates of risks to humans extrapolated from high-level experimental doses used in animal tests. Most tests have been performed with the primary objective of assessing the carcinogenicity of chemicals, and cancer is used as an end point. The paradigms commonly used for the detection of cancer do not adequately apply to neurotoxins. Most hazards assessed in terms of solvent exposures are considered qualitative rather than quantitative in nature.

Screening and surveillance of workers should include a careful occupational and social history, hobbies, and use of cosmetics, detergents and other household items, as well.

#### *Medicolegal Issues*

Medical professionals in the field of neurology are frequently asked to give expert opinions in situations of suspected neurologic injury. This may take the form of evaluating summaries and conclusions of primary treating physicians, or it may occur in depositions or a court of law. Due to the nature of the U.S. legal system, both plaintiff and defense attorneys are entitled to expert neurologic opinion. Frequently the task is to render an opinion concerning patients presenting with mild closed head injury<sup>11</sup> or neurotoxic exposure<sup>12</sup> — patients who are often described as malingerers, hysterics, suffering from psychosomatic or factitious illness, or presenting symptoms developed in the hope of possible secondary gain. It is imperative that the experts maintain a high degree of

(Continued on Page 26)

**(Neurology, cont'd from Page 11)**

professionalism, and study the subjects to the extent that they can rely on accurate and reasonable scientific testimony. In 1997, a group of neurologists and other neuroscientists assembled to form a new organization dedicated to establishing, maintaining, and improving national standards for medicolegal activities among neuroscientists. This organization is the American Neuroscience Forensic Association (ANFA). The circumstances that surround precipitating factors of closed head injury and neurotoxic exposure frequently involve a degree of civil or criminal liability. It is the purpose of the ANFA to provide a forum for the exchange of ideas concerning the medicolegal usefulness of new technology such as qEEG in the investigation of these issues.<sup>13</sup>

Peter G. Bernad, M.D., is a board certified internist and neurologist. In addition to being a practicing physician, Dr. Bernad is a consultant in the field of clinical toxicology. His books, *Closed-Head Injury: A Clinical Source Book* and *Neurotoxicology: A Clinical Sourcebook* are published by LEXIS<sup>®</sup> Law Publishing.

1. H. Tsuruta, *Percutaneous Absorption of Organic Solvents: Comparative Study of the In Vitro Percutaneous Absorption of Chlorinated Solvents in Mice* INDUSTRIAL HEALTH. 1975; 13:227-236.

2. P.G. Bernad & G.S. Saunders, *Evaluation of Patients Exposed to Neurotoxicants. Clinical Toxicology I*. CLINICS IN LABORATORY MEDICINE. 1990; 10(2); 387-402.

3. P.G. BERNAD, *NEURODIAGNOSTIC TESTING IN PATIENTS WITH CLOSED HEAD INJURY*. CLINICAL ELECTROENCEPHALOGRAPHY. 1991; 22(4):203-210.

4. F.H. Duffy, J.R. Hughes, F. Miranda, P.G. Bernad & P. Cook, *Status of Quantitative EEG (QEEG) in Clinical Practice, 1994*. CLINICAL ELECTROENCEPHALOGRAPHY. 1994; 25(4):6-22.

5. F.H. Duffy, J.L. Burchfiel, P.H. Bartels, M. Gaon & V.M. Sim, *Long Term Effects of an Organophosphate upon the Human Electroencephalogram*. TOXICOLOGY AND APPLIED PHARMACOLOGY. 1979 Jan; 47(1):161-76. J.L. Burchfiel, F.H. Duffy & M. Van Sim, *Persistent Effects of Sarin and Dieldrin upon the Primate Electroencephalogram*. TOXICOLOGY AND APPLIED PHARMACOLOGY. 1976 Feb; 35(2):365-79.

6. E.J. Jonkman, et al., *Electroencephalographic Studies in Workers Exposed to Solvents or Pesticides*. ELECTROENCEPHALOGRAPHY AND CLINICAL NEUROPHYSIOLOGY. 1992; 82:438-444.

7. M. Horvath & E. Frantik, *Quantitative Interpretation of Experimental Toxicological Data: The Use of Reference Substances, in 1 ADVERSE EFFECTS OF ENVIRONMENTAL CHEMICALS AND PSYCHOTROPIC DRUGS 11-21 (M. Horvath ed. New York, Elsevier, 1973) and 2 ADVERSE EFFECTS OF ENVIRONMENTAL CHEMICALS AND PSYCHOTROPIC DRUGS: NEUROPHYSIOLOGICAL AND BEHAVIORAL TESTS 334 (M. Horvath & E. Frantik eds. New York, Elsevier, 1976) as per Environmental Health Criteria 60: Principles and Methods for the Assessment of Neurotoxicity Associated with Exposure to Chemicals*. World Health Organization, Geneva 1986, p. 63.

8. Baker and Fine, JOM 28(2): 1986.

9. C. Edling, et al., *Long Term Follow-Up of Workers Exposed to Solvents*, BRITISH JOURNAL OF INDUSTRIAL MEDICINE. 1990; 47:75-82.

10. H.O. Hein, P. Suadicani & F. Gyntelberg, *Mixed Solvent Exposure and Cerebral Symptoms Among Active and Retired Workers. An Epidemiologic Investigation of 3387 Men Aged 53-75 Years*. ACTA NEUROL SCAND. 1990 Feb; 81(2):97-102.

11. P.G. BERNAD, *CLOSED HEAD INJURY: A CLINICAL SOURCEBOOK* (2d Ed. Charlottesville, Virginia, LEXIS<sup>®</sup> Law Publishing, 1998).

12. P.G. BERNAD, *NEUROTOXICOLOGY: A CLINICAL SOURCEBOOK*. (Charlottesville, Virginia, LEXIS<sup>®</sup> Law Publishing, 1998).

13. P.G. BERNAD & R.N. JONES, *Medical Legal Issues for Neuroscience*. CLINICAL ELECTROENCEPHALOGRAPHY. 1992; 23(3):160.

**(Guide, cont'd from Page 6)***Outpatient Treatment Analysis*

Much of modern medical care takes place outside the hospital setting and, therefore, is called outpatient care. This includes all care given in providers' offices, outside laboratories, therapy locations, and emergency clinics. An outpatient facility may include facilities associated in some way with an HMO or a large hospital consortium. If a patient is not intended to stay overnight, the care given is outpatient care. This is important, since there may be different state or federal regulations with respect to inpatient and outpatient care. Unless an outpatient facility is linked to some larger health delivery corporation, a physician's office for instance, none of the forms or charting procedures are standard. Most states have no particular requirements about outpatient records although some states have disciplined physicians on the basis of these records.<sup>17</sup>