Neurological Malpractice and Nonmalpractice Liability

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KEYWORDS

- Medical malpractice • Neurologic misadventures
- Neurological liability • Forensic neurology

This article provides an overview of the liability issues affecting neurologists. It focuses on current trends in malpractice law, with illustrative management strategies for several common recurring claims involving selected neurologic conditions. Nonmalpractice liability issues are discussed with particular attention to the unique risks engendered by the expert witness.

MALPRACTICE TRENDS

The overall medical malpractice claims frequency (number of claims filed) in the United States is at a historic low; payouts in constant dollars have plummeted, down 45% since 2000.[1,2] The result, however, is a paradoxically adverse impact on the specialty of neurology. The cumulative data from an insurance consortium review of 3812 neurology claims between 1985 and 2008 paints a disturbing picture[3]: the absolute number of paid neurology claims significantly increased over the past 5 years; the extraordinarily high payment ratio (percentage of paid claims to claims closed) more than doubled in the past 5 years (39.58% in 2007); neurology continues to have the highest average indemnity payment of all specialties including neurosurgery and obstetrics ($614,577 in 2007); and neurology claims, compared with every other specialty group, are the most costly to defend.

Several unique factors inherent to the specialty of neurology may explain these alarming statistics, which are at odds with general malpractice trends. First, the unprecedented growth of sophisticated neurodiagnostic tests, the proliferation of powerful neuropharmacologic agents, and the advent of more invasive procedures

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raise the standard of care, increasing the level of accountability and hence likelihood of suit. Second, neurologists, more so than other specialists, confront a diverse array of legal issues beyond the scope of traditional practice involving brain death, genetic testing, competency issues, neurotoxic insults, and evaluation of the neurologically impaired child. These varied conditions, governed by expanding legal doctrines, evolving regulatory control, and political whims, expose the neurologist to a variety of often novel claims. Third, neurologic liability extends beyond the physician-patient relationship to include a host of third parties. For example, there is tort liability for negligence to a patient that also injures a fetus, child, or spouse. In addition to the duty to warn of imminently dangerous patients, there is now a duty to warn third parties of communicable diseases. Neurologists have a duty to warn patients of medical conditions that may impair driving (epilepsy, sleep disorders, stroke); they may also be required to warn others directly, either by statute or an imposed tort duty to warn of foreseeable harm. The result is an everexpanding pool of potential claimants. Fourth, the very nature of neurologic disease or injury spells a grave outcome for many patients, which is undoubtedly reflected in the indemnity payments. The confluence of these factors may herald a fundamental shift transforming neurology from a low-risk specialty to one plagued by malpractice claims.

NEUROLOGIC MISADVENTURES

Medical misadventure refers to personal injury from either a negligent act or omission, or an adverse outcome of properly rendered care. The most prevalent neurologic misadventure is unquestionably diagnostic error, occurring in one third of all claims and in 45% of paid claims over the past two decades.[4] These errors commonly stem from the failure to perform an adequate history and examination, which is the most prevalent procedure resulting in claims against neurologists.[5] The most frequent incorrectly diagnosed conditions are malignant neoplasm of the brain, followed by headache (HA), intracranial and intraspinal abscess, nontraumatic subarachnoid hemorrhage (SAH), and vertebral fracture.[6] Other prevalent misadventures, in decreasing order of frequency, include improperly performed procedure, failure to supervise or monitor a case, medication errors, failure to recognize a complication of treatment, delay in performance, procedure performed when not indicated or contraindicated, procedure not performed, and failure to instruct or communicate with the patient.[7]

CLAIMS AGAINST NEUROLOGISTS

General Remarks

The provision of medical care meeting or even exceeding the prevailing standard may not effectively shield the neurologist from a lawsuit. A solid physician-patient relationship, valid consent, and proper medical record documentation are essential for successful risk management and malpractice defense.

The root of a malpractice claim is injury or perceived injury; however, most suits are actually triggered by a breakdown in the physician-patient relationship caused by poor communication. A thorough understanding of the relationship is crucial; meeting patient expectations through effective communication significantly reduces the risk of suit.

Informed consent issues are a frequent source of malpractice suits, wholly unrelated to negligence claims. The legal theories of consent detailed in the literature are equally applicable to all specialties, and discussed elsewhere in this issue.

Poor documentation is the leading factor in the forced settlement of most malpractice claims. The literature is replete with recommendations for ensuring that records are clear, accurate, complete, legible, and timely without alterations or other evidence of spoliation. It is redundant to reiterate good record-keeping principles in this article; however, one legal maxim must be emphasized: "If it is not in the record, it never happened."

Specific Claims

The extraordinarily broad scope of neurologic malpractice liability precludes a compendium of potential claims. Even limiting the claims to diagnostic errors is overwhelming. Moreover, such a listing is quickly outdated because emerging diagnostic and therapeutic options open the door for new claims. A more instructive approach is to consider the most prevalent patient conditions generating suits against neurologists. These include, in decreasing order of frequency, back disorders, cerebrovascular accident, convulsions, displacement of intervertebral disk, HA, epilepsy, occlusion and stenosis of cerebral arteries, migraines, nontraumatic SAH, and malignant neoplasm of the brain.[8]

Back disorders and intervertebral disk displacement are not discussed because these claims are generally attributable to straightforward diagnostic errors, few result in an indemnity payment, and the total indemnity is a small percentage of that paid for all neurology claims.[9] This article outlines several management strategies pertaining to the remaining conditions, arbitrarily grouped together as stroke, epilepsy, and HA, the latter subsuming migraine, brain tumor, and SAH. Lack of space precludes discussion of the myriad disparate claims involving these conditions. Several key topics were selected because they affect a large segment of the general population, are frequently seen by neurologists and nonneurologists alike, generate recurring claims, and have the potential for exceptionally high indemnity payments or judgments.

The discussion of each condition is written from a legal perspective, focusing on the origin of frequently encountered malpractice claims as opposed to discussing arcane details of sometimes obscure legal principles. This format requires oversimplification of the medical points, which necessitates omitting many conditions, truncating differential diagnoses, and ignoring various diagnostic and therapeutic options. It focuses solely on malpractice issues, and is not a substitute for conventional medical writings. Nor is it a treatise of neurologic malpractice; indeed, an impossible feat for a single article or even a single volume. This article is simply designed to provide the neurologist with a rudimentary understanding of how lawsuits arise, and generate some discussion on adapting practice patterns to improve patient care and minimize liability risk. References are kept to a minimum and, as much as possible, selected to provide the reader with additional background material for specific topics.

HEADACHE

General Considerations

HAs are ubiquitous, arguably the most common disorder encountered by the practicing physician, and the most common presenting symptom in malpractice claims against neurologists.[10] HA may be of little clinical significance or, paradoxically, herald potentially catastrophic illnesses, such as brain tumor, SAH, or meningitis. A complete and accurate diagnosis of the patient with HA requires a detailed history coupled with a full neurologic and general medical examination, as well as diagnostic testing and neuroimaging in selected cases. The single most important step in the evaluation is to classify the type of HA and ascertain whether it is acute, long-standing, or with

recent change. This practical approach allows the neurologist to determine the need for any diagnostic testing and initiate a proper treatment plan, all with the appropriate degree of urgency. Too often, the inexperienced, poorly trained, or hurried neurologist distorts a patient's history or fails to perform an adequate examination, resulting in the wrong diagnosis. Most malpractice suits stem from the failure to elicit an accurate history. The art of history taking cannot be taught in this article or in any other book; it includes an innate ability to establish a rapport, and instill confidence and trust. The author suggests the following methodology for the sole purpose of demonstrating several pitfalls that may lead to misdiagnosis, and recommends that neurologists formulate their own techniques, which will evolve with time, experience, and continuing education (**Box 1**).

Specific Approach

Evaluating the patient with HA requires a systematic approach to exclude more serious conditions, diagnose the primary HA, and formulate a treatment plan. There are particular aspects of each step that seem to generate recurring claims. This overview is limited to nontraumatic HAs in the adult population, with particular attention to the more common diagnostic and treatment errors.

The first step is to exclude serious conditions causing secondary HAs, which may share many of the same clinical features as a primary HA. The differential diagnosis of HA is exceedingly long, and indications for diagnostic testing must be made on an individual basis. The neurologist performing a history and examination should direct particular attention to warning signs or ''red flags'' suggesting a secondary HA, and proceed with appropriate diagnostic and therapeutic intervention. The author proposes the mnemonic ''SIGNAL'' to account for the most commonly misdiagnosed secondary HAs (**Box 2**).

The second step, after excluding secondary HAs, is to diagnose the primary HA in accordance with International Headache Society criteria.[20] It is beyond the scope of this article to review the various HA syndromes; however, the importance of correctly

Box 1
History taking methodology in HA

- Allow ample time for the consultation. Introduce yourself and invite the patient to sit for an interview before changing into a gown. Advise the patient that you have read the referral letter, but never accept either the patient's or referring physician's diagnosis.
- "Tell me about your HAs." Allow the patient to speak uninterruptedly before asking questions. Then begin open-ended queries to determine the quality, severity, location, duration, and time course of events, as well as precipitating, exacerbating, and relieving factors. It is helpful to ask the patient to describe a particular attack. Determine whether the patient has more than one type of HA. It is essential to separately evaluate each HA type, which may not be possible during the initial consultation because of time constraints. Subsequent appointments should be arranged accordingly.
- Communication skills are critical. Knowing which clues to follow and when to interrupt the patient are fundamental to an accurate history. Failure to understand the patient's terminology often leads to a misdiagnosis. The word "throbbing," for example, may be incorrectly translated into a migraine. The HA specialist must avoid distorting the history to fit a preconceived diagnostic category.
- The scope of the history must be sufficiently broad to address systemic diseases that may be relevant to the HA. Past, family, and social histories provide valuable information about the patient's condition. Before concluding the history, it is often enlightening to solicit the patient's opinion regarding the cause of the HA.

Box 2
Warning signs of secondary HA

1. **S**udden onset (thunderclap) HA. The sudden onset of severe HA mandates immediate and thorough evaluation for potential etiologies, such as SAH, intracerebral hemorrhage, venous or sinus thrombosis, intracranial or extracranial arterial dissection, aneurysmal expansion, pituitary apoplexy, or less common conditions.[11] Of these, SAH warrants further discussion. Aneurysmal hemorrhage accounts for 85% of nontraumatic cases and is the focus of this discussion.[12] It is among the most frequently missed serious causes of HA, and has a mortality rate of 50%.[13] More than half of patients presenting to the emergency room with a sentinel HA and SAH are misdiagnosed.[14] The failure to diagnose SAH consistently results in the highest percentage of paid claims (61.6%), and the highest average and highest total indemnity for all claims involving diagnostic error.[15] The sine qua non of SAH is a sudden HA classically described as the "first" or "worst HA of my life," often associated with nausea or vomiting, and followed by signs of meningeal irritation. Perhaps a better description is that the HA presents with maximal severity at onset. There may be cognitive impairment; focal deficits; or, in up to one half of cases, a history of premonitory symptoms suggestive of a sentinel bleed or aneurysmal expansion.[16] The known migraineur presenting with a sentinel HA may be misdiagnosed as having breakthrough symptoms; a thorough history is essential, because most patients recognize that the HA is different from a typical migraine.[17] The patient with thunderclap HA must have immediate CT of the brain and, if negative, a lumbar puncture to include spectrophotometric evaluation for xanthochromia.[18] The failure to perform a CT is the most common error; further evaluation based on the clinical presentation, and CT and lumbar puncture results, may warrant four-vessel cerebral angiography and neurosurgical consultation for definitive intervention.[19]
2. **I**ncreasing or worsening HA. The patient's HA pattern must be interpreted in light of the overall history. Recent-onset HAs with progression may indicate a tumor, subdural hematoma, or other mass lesion, and focal deficits may be present. A slow-growing mass, however, may not be associated with any neurologic deficits. Chronic primary HAs with progression may represent the development of a new, superimposed HA disorder (primary or secondary), or transformation of the primary disorder. It may be impossible to clinically distinguish the transformed migraine, often precipitated by medication overuse, from a new HA disorder. The presentation of an escalating HA, whether acute or chronic, warrants investigation.
3. **G**eneralized disease with HA. There are a plethora of systemic diseases presenting with acute HA including intracranial (eg, meningitis, encephalitis, sphenoid sinusitis) and generalized (eg, Lyme disease) infections; neoplasm (including paraneoplastic disease and leptomeningeal metastases); vascular conditions; autoimmune disorders; metabolic diseases; and toxic exposures. The diagnosis requires proficient examination with attention to systemic signs serving to guide diagnostic intervention. For example, the older patient with HA and visual symptoms may require temporal artery biopsy for giant cell arteritis.
4. **N**eurological or focal signs with HA. A HA associated with transient or permanent focal deficits other than a typical aura requires further evaluation.
5. **A**ctivity, exertion or cough HA. These HAs are frequently associated with posterior fossa structural abnormalities and warrant MRI to provide a definitive diagnosis.
6. **L**abor, pregnancy or postpartum HA. The new onset of HAs or progression of known primary HAs during pregnancy or postpartum raises the concern of sinus thrombosis, cerebral infarction, carotid dissection, pituitary apoplexy, and preeclampsia. These disorders most commonly occur during the third trimester or postpartum, present with HA, and may be associated with focal signs or seizures.

diagnosing the patient cannot be overstated. It is commonplace for the neurologist to label a patient with a particular HA type during the initial consultation and, despite a poor response to treatment, never consider revisiting the diagnosis. These patients are branded with the wrong diagnosis, and resultant therapy is ineffective as well as potentially harmful. It creates a breeding ground for malpractice claims.

The third step is to treat the primary HA with a comprehensive multimodality approach incorporating pharmacologic intervention predicated on evidence-based guidelines.[21] This approach is frequently ignored by the neurologist content with simply prescribing a medication. Management strategies for acute and chronic HA are detailed in the neurologic literature, and each therapeutic modality is subject to a unique array of claims.[22,23] A significant number of these suits, however, allege medication errors, such as failure to manage rebound phenomena; inappropriate use of medications (triptan prescribed in coronary artery disease); failure to properly monitor medication (liver failure on valproic acid); and failure to recognize side effects (β-blockers aggravating Raynaud phenomena).

The majority of patients with refractory HA have been misdiagnosed or improperly treated because of one of the following errors: incomplete or incorrect diagnosis (undiagnosed secondary HA, misdiagnosed primary HA, or failure to recognize multiple HA types); improper imaging studies (''normal'' CT overlooking posterior fossa lesion); ignoring exacerbating factors or triggers (failure to provide dietary instructions); poor pharmacotherapeutic management (subtherapeutic dosage); and neglecting rebound phenomena, which leads to persistent HAs.

Neuroimaging in the HA Patient

The role of neuroimaging in the adult patient with HA and a normal neurologic examination remains a controversial topic.[24] The American Academy of Neurology (AAN) Practice Guidelines state that ''neuroimaging is not usually warranted in patients with migraine and a normal neurologic examination,'' but should be considered in patients with an abnormal neurologic examination or ''patients with atypical headache features or headaches that do not fulfill the strict definition of migraine or other primary headache disorder.''[25] These parameters presuppose an accurate diagnosis of the patient's HA, which is frequently not the case. The most common diagnostic error in neurology is to label a patient with migraine or other HA disorder in the absence of neuroimaging, only to find that subsequent evaluation uncovers a brain tumor.[26] Arguments that earlier diagnosis would not have materially affected the outcome are generally unsuccessful. There may be absolutely no relationship between the HA and brain tumor, but the trier-of-fact will likely find otherwise if the neurologist failed to order a timely imaging study. The decision to forego neuroimaging in a patient with HAs requires a great deal of experience and clinical acumen. For many neurologists, it is simply prudent to perform an imaging study on every HA patient early in the evaluation. There is no point in repeating a test if it was already performed, assuming no change in the patient's condition. There are no evidence-based recommendations in the United States regarding the relative sensitivity of MRI compared with CT in nonacute HA disorders, although a European Task Force recommends MRI.[27] Most experts agree MRI is the superior choice because of its sensitivity to venous thrombosis, extracranial hematomas, neoplasms, and meningeal disease; and ability to visualize the posterior fossa, cervicomedullary junction, and pituitary region. Unfortunately, neurologists may be deterred from ordering these studies because of onerous preauthorization requests or concerns over deselection, and failure to diagnose brain tumor will likely remain one of the most common malpractice claims.

CEREBROVASCULAR DISEASE

Globally, almost 6 million people die from stroke each year; it is the third leading cause of death in the United States with almost 800,000 strokes annually.[28] Stroke therapy has changed dramatically over the past decade with the development of specific treatment options (thrombolysis, endovascular therapy) and refinement of prevention strategies (anticoagulation, carotid endarterectomy [CEA]). These recent advances, along with improved diagnostic modalities, create a heightened expectation of proper stroke management and, combined with the catastrophic impact of stroke, portend increasing litigation in this area.

Thrombolytic Therapy

Tissue plasminogen activator (tPA) thrombolysis arguably represents the neurologic standard of care for acute ischemic stroke, despite the fact that an extremely low percentage of eligible patients receive the drug at this time. Intravenous administration of tPA within 3 hours of ischemic stroke significantly improves functional outcome in selected patients.[29–34] Recent data suggest modest but significant clinical improvement in patients treated 3 to 4.5 hours after onset of stroke symptoms, resulting in a science advisory for this population.[35,36] The therapeutic window is narrow, and strict adherence to the approved protocol inclusion and exclusion criteria is imperative.[37,38] The hospital, emergency department, radiology team, and neurology and neurosurgery consultants should establish a dedicated stroke center capable of responding to every acute ischemic stroke patient in a timely fashion and, if indicated, administering tPA.[39] Alternatively, tPA-eligible patients must be promptly transferred to another institution for definitive treatment if it can be accomplished within a suitable time frame. Failure of the hospital to provide appropriate facilities and personnel (streamlined emergency room intake, CT technicians continuously available) may create liability for all parties including the neurologist.

The failure to recommend or administer tPA to an eligible patient may constitute negligence, unless it can be proved that tPA would not have made a material difference in the patient's outcome. The neurologist deciding not to use tPA in an acute ischemic stroke should clearly document the reasons for that decision in the medical records. It is equally important for the neurologist to resist pressure from the emergency physician or family to use tPA unless the patient meets all inclusion and exclusion criteria. Modification of the criteria, especially the time constraint, decreases the benefit of tPA and increases the risk of intracerebral hemorrhage.[40] Determination of the time of stroke onset is crucial. It is a common error to label the onset as the time symptoms were first observed rather than the last time the patient was known to be well. For example, if the patient awakens with deficits, then the onset time must be considered the last time the patient was known to be well (usually the night before), not when the symptoms were first noticed on wakening. The same holds true for patients unable to communicate these details. Likewise, patients with stroke-related neglect syndromes cannot reliably observe the onset time. Another frequent error is the administration of anticoagulants or antiplatelet agents during the first 24 hours after tPA administration, which greatly increases the risk of intracerebral hemorrhage. Again, it is imperative to follow the guidelines.[41] There are cases, however, where the neurologist may consider all of the risks and benefits, and decide it is in the patient's best interest to deviate from the protocol. This decision should be discussed with the patient or legal representative and family, and thoroughly documented in the records.

The failure to obtain valid informed consent may precipitate a malpractice action separate from negligence.[42] Informed consent mandates a frank discussion regarding the benefits and risks of tPA, including the potential for hemorrhage, coma, and death.[43] The acute stroke patient may not be able to fully participate in the process because of communication deficits or cognitive impairment. Options should then be discussed with a close family member and documented, but only a legal representative (guardian or person with written power of attorney) can give consent. If the patient is unable to give consent and no legal representative is available, the neurologist may proceed with tPA when it is the most reasonable option. Courts recognize an implied consent; there is an assumption that a competent individual would have agreed to the procedure.[44]

Anticoagulation Therapy

The use of heparin to prevent an impending stroke remains controversial despite the absence of supporting evidence, and immediate anticoagulation is occasionally recommended for fluctuating basilar artery thrombosis, extracranial arterial dissection, and imminent carotid artery occlusion, as well as certain cases of cardioembolic and noncardioembolic cerebral infarction. It is increasingly difficult to defend any complications in these circumstances because the weight of the evidence is against anticoagulation.[45]

Warfarin may be beneficial in the first few months after an ischemic event, but there is no definitive evidence that the benefits of long-term anticoagulation for thrombosis or embolism outweigh the potential risks except in patients with nonvalvular atrial fibrillation, prosthetic heart valves, and acute myocardial infarction.[46] Nonvalvular atrial fibrillation affects 2.5 million Americans and the prevalence increases with age; it increases the risk of stroke fourfold to sixfold across all age groups.[47,48] The annual rate of ischemic stroke in untreated nonvalvular atrial fibrillation patients increases with high-risk factors, such as hypertension, left ventricular dysfunction, transient ischemic attack (TIA), or prior stroke.[49] Anticoagulation with warfarin significantly reduces this risk of stroke, and represents the generally accepted standard of care for stroke prevention in these patients.[50] Multiple separate guidelines and over two dozen randomized trials in the past two decades consistently advocate anticoagulation for nonvalvular atrial fibrillation patients with additional risk factors conferring high risk of stroke.[51] These guidelines differ in the classification of risk criteria; however, every statement labels prior stroke or TIA high risk, and recommends anticoagulation. If warfarin is contraindicated, or the patient is at low risk of stroke, then antiplatelet therapy is the appropriate treatment.

Neurologists may be reluctant to use warfarin because of the required follow-up and monitoring, or they may inappropriately minimize the medication dosage out of undue concern about bleeding. This is a frequent subject of litigation, with the claim that a major stroke would have been prevented if the patient had been properly anticoagulated. It is, therefore, imperative to identify patients at risk for stroke in accordance with established clinical guidelines. Accurate diagnosis is essential, including appropriate neuroimaging before initiating therapy. The reasons for or against anticoagulating a patient at risk should be documented in the medical records. For example, if the increased risk of bleeding caused by gait instability outweighs the potential benefits of anticoagulation, then careful documentation may protect against litigation if the patient suffers a massive embolus. Patient and family education concerning the management of anticoagulation is crucial, and should be clearly documented. Certain medications must be avoided or used with extreme caution because of the increased risk of hemorrhage when combined with warfarin (aspirin, barbiturates, cephalosporins,

sulfa drugs, high-dose penicillin). Establish and follow written procedures for monitoring patients on warfarin, or enlist one of the anticoagulant management services.

CEA and Angioplasty

Over one quarter of recently symptomatic patients with a high-grade carotid stenosis (70%–99% diameter reduction) suffer an ipsilateral stroke within 2 years, despite appropriate management of risk factors and antiplatelet therapy.[52] CEA significantly reduces the incidence of cerebral infarction in these patients and may be considered to represent the standard of care; it is moderately useful for symptomatic patients with 50% to 69% stenosis, not indicated for symptomatic patients with less than 50% stenosis, and individualized decisions are required for the smaller benefit in asymptomatic patients with 60% to 99% stenosis.[53] There must be careful patient selection (ie, attention to patients with a high-grade tandem lesion in the ipsilateral intracranial arteries, or asymptomatic patients with severe contralateral carotid artery stenosis or occlusion), and skill of the surgical team is paramount. The most common malpractice claims are failure to diagnose TIA or minor stroke, and failure to perform an evaluation for carotid stenosis, allowing the patient to suffer a recurrent or massive stroke. Every patient with a TIA or stroke should have appropriate neuroimaging unless surgery is plainly contraindicated. Patients with symptomatic carotid artery stenosis greater than 70% should be offered CEA or carotid angioplasty. Other degrees of stenosis require individualized considerations, which must be well documented. Delay in referring a TIA patient with high-grade stenosis for definitive treatment may also constitute negligence, since a high percentage of strokes occur within 48 hours of the TIA.[54] Surgery should be offered as soon as possible after a TIA or nondisabling stroke, preferably within 2 weeks of the last symptomatic event.[55] Premature surgical intervention following a moderate to severe stroke creates a liability risk for extension or hemorrhagic conversion of the infarction; however, there is insufficient evidence to support or refute delaying CEA for 4 to 6 weeks.[56] Carotid angioplasty is a more recent procedure, and its indications are still evolving. Informed consent issues are critical, and all decisions should be thoroughly documented in the medical records.

EPILEPSY

There are over 2 million epileptics in the United States.[57] Approximately 150,000 adults present annually with a first seizure, with almost half recurring to be classified as epilepsy; the lifetime cumulative risk of a seizure ranges from 8% to 10%, with a 3% chance of developing epilepsy.[58] These disorders present formidable legal challenges because of the variable clinical symptoms, diverse etiopathogenetic mechanisms, and diagnostic and therapeutic complexity in patients who commonly harbor intellectual impairment, cognitive dysfunction, and psychiatric symptoms.[59,60]

Driving

Every state restricts issuance of a driver's license to individuals who have suffered loss of consciousness. The laws differ among the states, but generally require that an individual be seizure free for a period of time before obtaining a license. This seizure-free interval is variable within individual state jurisdictions, ranging from no fixed duration to 1 year. A physician's evaluation must be submitted to the state before a license is issued. Neurologists are rightfully concerned about their potential liability when certifying that a patient with epilepsy is capable of driving. Some states grant immunity to the physician, although the level of immunity varies among the jurisdictions, ranging from ''good faith'' immunity to immunity from suit. In other states, physicians are

not granted statutory immunity from liability for the information they provide to the state or for damages arising out of a seizure-related accident. In states without physician immunity laws, courts may still refuse to impose liability on the neurologist who exercised reasonable care and good faith in reporting to the state.

Six states (California, Delaware, Nevada, New Jersey, Oregon, and Pennsylvania) have express mandatory reporting statutes requiring physicians to report patients with epilepsy (or other disorders associated with a loss of consciousness or impaired ability to drive) to the state.[61] All other states have voluntary reporting statutes. The neurologic standard of care for reporting the epileptic patient varies according to the laws and regulations of each state. It is incumbent on neurologists to know the relevant statutes in their jurisdiction, and have an understanding of the common law trends for any ambiguous issues. The neurologist has a duty to advise patients of the legislation in their particular state, and emphasize the importance of complying with the law. If the state has an explicit self-reporting requirement, patients should be advised in writing to comply, retaining a copy of the letter in the medical records. The discussion of driving restrictions and restrictions on other activities, the effect of discontinuing or reducing dosage of a drug, and possible side effects of medications in relation to driving should be clearly documented in the records. These issues should be reiterated and documented on any change in medication because of the increased risk of breakthrough seizures.

If an epileptic patient continues to drive because the neurologist failed to report where reporting is mandatory, or failed to instruct the patient in a voluntary reporting state, then a seizure-related accident may trigger a malpractice suit by the patient or the patient's estate. It is imperative that the neurologist clearly document patient instructions in the medical records, and keep a copy of any notification sent to the state. It is also advisable to record any factors that may mitigate liability for not filing a report. The patient who drives against medical advice is a special concern for every neurologist, especially in voluntary reporting states. *Tarasoff* reasoning may be applied to the neurologist who advises a patient not to drive, learns the patient continues driving, and fails to take any further action.[62] In this situation, the neurologist should inform the patient in writing about the potential consequences of driving, and consider filing a voluntary report with the appropriate state agency. There may be statutory protection for a voluntary report that is made in good faith and consistent with the prevailing standard of care. The level of protection varies among jurisdictions, however, and it is advisable to consult legal counsel.

Neurologists may be liable to third parties for failing to report a patient or certifying a patient to drive. This is an emerging area of liability, and most decisions turn on whether the neurologist owes a duty to the third party. Courts have ruled in both directions, and the issue remains far from settled.[63] Neurologists should adapt practice patterns to comport with the relevant legal trends in their jurisdiction, but even third-party liability is minimized by effective patient discussions, proper reporting, and thorough documentation, as outlined previously.

Teratogenesis

There are over 0.5 million women with epilepsy of childbearing age in the United States; 3 to 5 births per 1000 are to epileptic women.[64] Epilepsy is the most common neurologic disorder in pregnancy, and it raises a host of legal and medical issues. The most serious concern, however, is the potential for congenital malformations in the offspring of mothers taking antiepileptic drugs (AEDs). These mothers have an up to 7% risk of bearing a child with congenital malformations, threefold higher than nonepileptic mothers.[65] This higher risk is probably multifactorial with genetic and social

components, but AEDs are clearly implicated as human teratogens.[66] All conventional AEDs (phenytoin, phenobarbital, carbamazepine, and valproic acid [VPA]) taken during the first trimester share an increased risk of malformations, which commonly include orofacial clefts, congenital heart disease, neural tube defects, and urogenital malformations.[67] It is not clear if the increased risk is imparted from one or some AEDs; however, VPA harbors a greater risk of major fetal malformations and should be avoided in women who may become pregnant.[68] The teratogenic potential of the newer AEDs remains unknown, and these drugs should be avoided during pregnancy.

Malpractice suits for AED-induced fetal malformations have the potential for extraordinarily large settlements or judgments, and tolling of the statute of limitations is commonplace. The neurologist must address a variety of complex issues in epileptic women who take AEDs during their reproductive years to minimize liability for these claims. The recent guidelines are not particularly helpful because of a paucity of evidence limiting the strength of many findings and recommendations.[69,70] The following suggestions are provided to focus on some of the clinical points that seem more commonly raised in lawsuits. Detailed counseling early in the reproductive years should include a discussion of the increased risk of seizures during pregnancy, importance of medication compliance, necessity of regular follow-up with AED levels, risk of malformations, folic acid and vitamin K supplementation, and the importance of avoiding coteratogens. Before pregnancy, it is important to determine whether AEDs are necessary; for example, if the patient is receiving an anticonvulsant for migraine, depression, or some other disorder, it may be possible to discontinue the drug. Additionally, if the patient with a single type of seizure has been in remission for 2 to 5 years, and has a normal neurologic examination with no EEG abnormalities, then it may be reasonable to gradually withdraw the drug. The taper must be performed slowly over months, and completed 6 months before conception, because seizure recurrence is most likely during this time. If treatment is indicated, every effort should be made to place the patient on monotherapy with the lowest effective dose of the most suitable AED. Frequent daily dosing avoids high peak levels, possibly reducing the potential for teratogenesis. The free (non–protein-bound) AED levels should be monitored at least preconception, at the beginning of each trimester, the last month of pregnancy, and 2 months postpartum. Pregnancy screening should include serum alpha fetoprotein at 16 to 18 weeks and a level II ultrasound at 18 to 20 weeks. If indicated, amniocentesis may be offered at 18 to 20 weeks. The patient should be properly counseled if there is a serious malformation, and provided with the option to terminate the pregnancy. The administration of folic acid in the early stages of pregnancy probably decreases the incidence of neural tube defects and, despite the limited guideline recommendations, should be given to all women of childbearing potential. Optimal dosage for epileptics remains controversial, and data must be extrapolated from nonepileptic women; it is a matter of clinical judgment but should be between 0.4 and 4 mg/d.

It is not uncommon for women with epilepsy to present to the neurologist after becoming pregnant. In general, the risk of uncontrolled epilepsy is greater than the risk of AED-induced teratogenesis, and drug treatment must be continued throughout pregnancy. For several reasons, it is a serious albeit common error to change medications for the sole purpose of reducing teratogenic risk. First, there is a risk of precipitating seizures that may reduce placental blood flow and impair fetal oxygenation. Second, the critical period of organogenesis has usually passed, and discontinuing an AED does not lower the risk of congenital malformations. Third, exposing the fetus

to a second agent during the crossover period is akin to polytherapy and increases the teratogenic risk. If an epileptic woman presents after conception on effective monotherapy, the AED, even if VPA, should generally not be changed. Lastly, hemorrhagic disease of the newborn may occur in neonates exposed to hepatic enzyme inducing AEDs, and requires special attention including maternal administration of oral vitamin K during the last month of pregnancy.

NONMALPRACTICE LIABILITY

Neurologists must be cognizant of the morass of laws and regulations affecting their practice, raising the specter of adverse licensing sanctions, civil penalties, and criminal prosecution. This nonmalpractice liability penumbra generically includes credentialing disputes (professional licensure, hospital privileges, professional organization membership); reimbursement issues (fee disputes, program exclusion, denial of managed care contracts); and myriad ad personam (assault, manslaughter, homicide), economic (antikickback, self-referral, and antitrust violations; false claims), and regulatory (violations of Americans with Disabilities Act, Health Insurance Portability and Accountability Act, Emergency Medical Treatment and Labor Act) crimes.[71] The relevant legal principles governing these diverse areas are substantially the same for all specialties and do not warrant review in this article.[72–78]

FORENSIC NEUROLOGY LIABILITY

Many neurologists have addressed managed care constraints by expanding their practices to incorporate medical record reviews, independent medical examinations, and expert witness services. These lucrative activities generally do not invoke a physician-patient relationship (thereby precluding a malpractice claim), but may lead to administrative penalties, civil lawsuits, and criminal prosecution. In particular, expert witness activities engender unique risks warranting further discussion.[79–81]

Anecdotal reports of neurologists advancing specious complaints are legion.[82] One review of expert witness testimony involving neurologists demonstrated improper testimony and erroneous conclusions regarding malpractice in 37% of cases.[83] It is ''alarmingly common for accomplished neurologists to hire themselves out for [one-sided testimony].''[84] These partisan experts have flourished behind the common law expert witness immunity shield and lack of professional oversight. Today, there is a trend toward accountability with increased expert witness liability.[85] Friendly expert lawsuits (retaining party sues the expert) are increasing.[86] The traditional immunity is not absolute, and most states ruling on this issue have carved out exceptions to hold the expert liable for professional negligence.[87,88] One state Supreme Court explained that an ''absence of immunity will… protect the litigant from the negligence of an incompetent professional.''[89] This may represent an effective means of stemming the proliferation of negligent experts. Courts have also upheld suits against opposing and independent experts. Some jurisdictions continue to favor immunity for testimony, but that does not necessarily extend to nontestimonial expert activity (discovery of facts, literature search).[90] Nor does it protect the expert from criminal prosecution for improper testimony or misrepresentation of a degree or license.[91] The expert neurologist may also be liable for defamatory communications, and face administrative, civil, or criminal charges for negligent or intentional spoliation of evidence.[92]

Expert testimony and related activities are subject to increasing scrutiny by state licensing boards and professional organizations. The American Medical Association considers testimony to be the practice of medicine and subject to peer review, and supports state licensing boards in disciplining physicians who provide fraudulent

testimony or false credentials.[93] Some boards have expanded the definition of medical practice to include testimony, allowing disciplinary action if warranted.[94] The AAN adopted *Qualifications and Guidelines for the Physician Expert Witness*, promulgated a code of professional conduct for legal expert testimony, and established a formal disciplinary procedure for errant neurologists with potential sanctions ranging from censure to expulsion.[95–98] AAN disciplinary actions may trigger the American Board of Psychiatry and Neurology to revoke certification.[99] The Seventh Circuit Court of Appeals validated these forms of discipline, stating in dicta that the American Academy of Neurologic Surgeons had a duty to discipline a neurosurgeon for irresponsible testimony.[100]

This complex, evolving area of law will create a more perilous liability climate for the future expert. The standard of care for expert services varies with the particular facts of each case, but salient guidelines applicable to all circumstances include the following: fulfill the AAN qualifications before accepting a case; review all relevant medical information; review the standard of care for the time of occurrence; perform adequate discovery of facts; review and understand the relevant literature; properly assemble and present the case; avoid losing or destroying any evidence; provide accurate, impartial, and truthful testimony; avoid conflicts of interest; do not discuss the case outside the course of litigation; and ensure compensation is reasonable, not contingent on outcome. It is important to remember that all deposition and trial testimony constitutes a permanent public record, which may be accessed from various national repositories. Some professional organizations maintain copies of depositions and court testimony (eg, the Defense Research Institute, in Chicago; Association of Trial Lawyers of America, in Washington; Collaborative Defense Network for Expert Witness Research; and various medical groups, such as the American Association of Neurological Surgeons).

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