

Chapter 47

Liability of Neurologists

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Malpractice Liability
Nonmalpractice Liability

Liability of the Forensic Neurologist
Conclusion

This chapter provides an overview of the liability issues affecting neurologists. It focuses on current trends in malpractice law. Illustrative management strategies are provided for several common recurring claims involving stroke, epilepsy, and headache. Nonmalpractice liability issues are discussed with particular attention to the unique risks engendered by the expert witness.

MALPRACTICE LIABILITY

Current Trends

Medical malpractice claims in the United States are generally increasing in frequency and severity, with a disproportionately adverse impact on neurology. The data from an insurance consortium review of 3280 neurology claims between 1985 and 2004 paints a disturbing picture:¹ The percentage of neurology claims to total claims continues to increase, the payment ratio (percentage of paid claims to claims closed) remains high, and neurology has the highest average indemnity payment of all specialties including neurosurgery. Moreover, neurology claims are costly to defend.

There are several unique factors inherent to the specialty of neurology that may explain these alarming statistics. First, the unprecedented growth of sophisticated neurodiagnostic tests, the proliferation of powerful neuropharmacological agents, and the advent of more invasive procedures raise the standard of care, increasing the level of accountability and hence likelihood of suit. Second, neurologists confront a diverse array of legal issues beyond the scope of traditional practice involving, *inter alia*, 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, neurology engenders liability beyond the physician–patient relationship to include a host of third parties. Neurologists may face 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 ever-expanding pool of potential claimants. Fourth, the very nature of neurological disease or injury spells a grave outcome for many patients and this is 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.

Neurological Misadventures and Claims Against Neurologists

The most prevalent neurological misadventure is diagnostic error, occurring in one-third of claims.² The most frequent incorrectly diagnosed condition is malignant neoplasm of the brain, followed by intracranial and intraspinal abscess, subarachnoid hemorrhage (SAH), headache, and vertebral fracture.³ Other prevalent misadventures include (in decreasing order of frequency): improperly performed procedure; failure to supervise or monitor case; medication errors; failure to recognize a complication of treatment; procedure performed when not indicated or contraindicated; procedure not performed; delay in performance; and failure to instruct patient.⁴

General Remarks

The provision of medical care that meets or exceeds the prevailing standard may not 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.

Although the root of a malpractice claim is injury or perceived injury, most suits are actually triggered by a breakdown in the physician–patient relationship due to poor communication. A thorough understanding of the relationship, as outlined in this volume, is essential; 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 informed consent detailed in this volume are applicable to all specialties, and are therefore not repeated in this chapter.

Poor documentation is the leading factor in the forced settlement of most malpractice suits. The literature is replete

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with recommendations for ensuring that records are clear, accurate, legible, complete, and timely without alterations or other evidence of spoliation. It is unnecessary to reiterate good record-keeping principles in this chapter, but one legal maxim cannot be overemphasized: "If it is not in the record, it never happened."

Specific Claims

The scope of neurological malpractice liability precludes a compendium of potential claims. Moreover, any such listing would be outdated before publication, as emerging diagnostic and therapeutic options open the door for new claims. It is, however, instructive to consider the most prevalent patient conditions generating suits against neurologists (in decreasing order of frequency): back disorders; cerebrovascular accident; displacement of intervertebral disk; convulsions; headache; epilepsy; migraine; malignant neoplasm of the brain; SAH; and musculoskeletal disorders affecting the neck region.⁵

Intervertebral disk displacement and related spine disorders are not discussed because these claims are predominantly straightforward diagnostic errors, only a small percentage result in an indemnity payment, and the total indemnity is a small fraction of the total paid for all neurology claims. This section outlines several management strategies for the more common claims involving the remaining conditions, arbitrarily grouped together as stroke, epilepsy, and headache, the latter subsuming migraine, brain tumor, and SAH. It is impossible in an introductory chapter to provide a discussion of the widely disparate malpractice suits involving these conditions. Therefore, several particular topics were selected because they are frequently seen by neurologists and nonneurologists alike, affect a large segment of the population, generate recurring claims, and have the potential for devastating outcomes with exceptionally high indemnity payments or judgments.

Stroke

Cerebrovascular disease is the third leading cause of death in the United States with approximately 200,000 fatalities annually; there are more than 700,000 individuals newly diagnosed with stroke every year.⁶ Stroke therapy has changed dramatically over the last decade with the development of specific treatment options (thrombolysis) and prevention strategies (anticoagulation, carotid endarterectomy). These recent advances create a heightened expectation of proper stroke management and, combined with the catastrophic impact of stroke, portend increasing litigation in this area.

Thrombolytic Therapy The administration of tissue plasminogen activator (tPA) within 3 hours of ischemic stroke onset significantly improves functional outcome in selected patients.⁷ The therapeutic window is narrow and strict adherence to the approved protocol inclusion and exclusion criteria is imperative.⁸ tPA thrombolysis represents the neurological standard of care for acute ischemic stroke, despite the fact that a low percentage of eligible patients receive the drug at this time. The hospital, emergency department, and neurology consultant should establish a

dedicated stroke team capable of responding to every acute ischemic stroke patient in a timely fashion and, if indicated, administering tPA. Alternatively, tPA-eligible patients must be promptly transferred to another institution for definitive treatment. Failure of the hospital to provide appropriate facilities and personnel (streamlined emergency room pathways, CT technicians available 24 hours) 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 proven 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. The evidence demonstrates that modification of the criteria, especially the 3-hour time constraint, decreases the benefit of tPA and increases the risk of intracerebral hemorrhage.⁹ Thus it is crucial to determine the time of stroke onset. A common error is to consider the onset time as the time when 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 to be the last time the patient was known to be well (usually the night before), not when the symptoms were first noticed upon waking. The same is true if the patient is unable to communicate the onset time. Additionally, neurologists must be attentive to patients with stroke-related neglect syndromes who cannot reliably observe the time of onset. 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 protocol guidelines. 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 consent may precipitate a malpractice action separate from negligence.¹⁰ Informed consent mandates a frank discussion regarding the benefits and risks of tPA, including the potential for hemorrhage, coma, and death.¹¹ The acute stroke patient may not be able to fully participate in the process due to 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 will recognize an implied consent; there is an assumption that a competent individual would have agreed to the procedure.¹²

Anticoagulant Therapy The use of *heparin* to prevent an impending stroke remains controversial, although evidence

supports immediate anticoagulation for fluctuating basilar artery thrombosis and impending carotid artery occlusion, as well as in certain cases of cardioembolic cerebral infarction.

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 (AF), prosthetic heart valves, and acute myocardial infarction. AF affects 2.5 million Americans and the prevalence increases with age; it increases the risk of stroke 4- to 6-fold across all age groups.¹³ The 5% annual rate of ischemic stroke in untreated AF patients increases with high risk factors such as hypertension, left ventricular dysfunction, transient ischemic attack (TIA), or prior stroke.¹⁴ Anticoagulation with warfarin significantly reduces this risk of stroke, and represents the standard of care for stroke prevention in these patients. In fact, 11 separate guidelines advocate anticoagulation for AF patients with additional risk factors conferring high risk of stroke.¹⁵ These guidelines differ in the classification of risk criteria; however, every guideline 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 amount of monitoring and follow-up required 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 was properly anticoagulated. It is imperative, therefore, to identify patients at risk for stroke in accordance with established clinical guidelines. Accurate diagnosis is essential and neuroimaging to rule out intracranial hemorrhage must be performed 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 due to 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 medication increasing the risk of bleeding should be avoided or used with extreme caution (i.e., 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.

Carotid Endarterectomy and Angioplasty Over one-quarter of recently symptomatic patients with a high-grade carotid stenosis (70% to 99% diameter reduction) will suffer an ipsilateral stroke within 2 years, despite appropriate management of risk factors and antiplatelet therapy.¹⁶ Carotid endarterectomy (CEA) significantly reduces the incidence of cerebral infarction in these patients, and represents the standard of care.¹⁷ There must be careful patient selection (i.e., exclusion of 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 claim is the failure to diagnose TIA or minor stroke, or failure to perform a workup for carotid stenosis, allowing the patient to suffer a recurrent or massive stroke. Every patient with a TIA or stroke should have a carotid doppler or magnetic resonance angiography (MRA) unless surgery is plainly contraindicated. Patients with symptomatic carotid artery stenosis greater than 70% should be offered CEA or carotid angioplasty. 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.¹⁸ Surgery should be offered as soon as possible after a TIA or nondisabling stroke, preferably within 2 weeks of the last symptomatic event.¹⁹ 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 4–6 weeks.²⁰ 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 records.

Epilepsy

Driving Every state restricts issuance of a driver's license to individuals who have suffered a 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 driver's license. The seizure-free interval is variable within individual state jurisdictions, ranging from no fixed duration to one year. The trend is toward a shorter time frame, with consideration of individual factors (seizure due to physician-directed medication changes or a temporary condition, an established pattern of nocturnal seizures). A physician's evaluation must be submitted to the state before a license will be issued.

Neurologists are rightfully concerned about their potential liability when certifying to the state 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 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.²¹ All other states have voluntary reporting statutes. The neurological standard of care for the epileptic patient varies according to the laws and regulations of each state.

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It is incumbent upon neurologists to know the relevant statutes of 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, with a copy of the letter kept in the medical records. The discussion of driving restrictions as well as restriction 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 upon any change in medication, due to the increased risk of breakthrough seizures.

If an epileptic continues driving because the neurologist either 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 patient's estate. Therefore 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.²² 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. However, the level of protection varies among jurisdictions, 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 is far from settled.²³ 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 above.

Teratogenesis There are over 1 million women with epilepsy of childbearing age in the United States; 3–5 births per thousand are to epileptic mothers. Epilepsy is the most common neurological disorder in pregnant women and it raises a number of legal and medical issues. However, the most serious concern 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. Although this higher risk is probably multifactorial with genetic and social components,

AEDs are clearly implicated as human teratogens.²⁴ All conventional AEDs—phenytoin, phenobarbital, carbamazepine, and sodium valproate—share an increased risk of congenital malformations, which commonly include orofacial clefts (cleft lip or cleft palate or both), congenital heart disease, neural tube defects, skeletal abnormalities, and urogenital malformations.²⁵ There are a number of ongoing pregnancy registries collecting data on AED-associated fetal abnormalities, and neurologists must stay current with the results.²⁶ For example, emerging data demonstrates that valproic acid harbors a much greater risk of major fetal malformations than other antiepileptics, and should be avoided in women who may become pregnant.²⁷ The teratogenic potential of the newer AEDs remains unknown, and these drugs should be avoided in 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 in order to minimize liability for these claims.²⁸ 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. Prior to 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 2–5 years, and has a normal neurological examination with no EEG abnormalities, then it may be reasonable to gradually withdraw the drug. The withdrawal must be performed slowly over months, and completed 6 months before conception, since recurrence of seizures 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 will avoid high peak levels, possibly decreasing the potential for teratogenesis. The administration of folic acid in the earliest stages of pregnancy reduces the incidence of neural tube defects and should be given to all women of childbearing potential. Optimal dosage for epileptics is controversial, and data must be extrapolated from studies of nonepileptic women; the author recommends 4.0 mg/day combined with vitamin B₁₂.

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 increases the teratogenic risk. Thus, if an epileptic woman presents after conception on effective monotherapy, the AED should generally not be changed. 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 K₁ 20 mg/day during the last month of pregnancy.

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 16–18 weeks and a level II ultrasound at 18–20 weeks. If indicated, amniocentesis may be offered at 18–20 weeks. The patient should be properly counseled if there is a serious malformation, and provided with the option to terminate the pregnancy.

Headache

Headache is arguably the most common patient complaint in the outpatient neurology setting. It may be of little clinical significance or, paradoxically, herald a catastrophic illness, such as brain tumor, SAH, or meningitis. A complete and accurate diagnosis of the headache patient requires a detailed history coupled with a full neurological and general medical examination. The single most important step in the evaluation is to classify the type of headache and, *pari passu*, ascertain whether it is acute, long-standing, or with recent change. This practical approach will allow 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. Accordingly, headache remains one of the most prevalent conditions in recurring malpractice claims against neurologists. These claims seem to run the gamut: failure to diagnose brain tumor; failure to identify rebound phenomenon; failure to diagnose and treat temporal arteritis; failure to diagnose and treat sphenoid sinusitis; avascular necrosis secondary to steroids; and inappropriate use of triptans or ergotamines.²⁹ There are two areas of particular concern that warrant further discussion: neuroimaging in the patient with headache and the failure to diagnose SAH.

Neuroimaging The role of neuroimaging in the patient with headache and a normal neurological examination remains a controversial area. The American Academy of Neurology (AAN) Practice Guidelines state that “neuroimaging is not usually warranted in patients with migraine and a normal neurological examination,” but should be considered in patients with an abnormal neurological examination or “patients with atypical headache features or headaches that do not fulfill the strict definition of migraine or other primary headache disorder.”³⁰ These parameters presuppose an accurate diagnosis of the patient's headache, which is frequently not the case. For example, it is not uncommon for the neurologist to diagnose a patient with migraine or

chronic headache in the absence of neuroimaging, only to find that subsequent evaluation uncovers a brain tumor.³¹ Arguments that earlier diagnosis would not have materially affected the outcome are generally unsuccessful. There may be absolutely no relationship between the headache and brain tumor, but the trier-of-fact will likely find otherwise if the neurologist failed to order a timely imaging study. Thus, the decision to forgo neuroimaging in a patient with headaches requires a great deal of experience and clinical acumen. For many neurologists, it would simply be prudent to perform an imaging study on every headache patient early in the evaluation. Of course, there is no point in repeating a test if it was already performed, assuming there is no change in the patient's condition. There are no evidence-based recommendations for the relative sensitivity of MRI as compared with CT in the evaluation of migraine or other nonacute headaches. However, MRI is probably a superior choice in most circumstances given its sensitivity and ability to visualize the posterior fossa. Unfortunately, concerns over deselection and negative capitation may deter the neurologist from ordering these studies, and the failure to diagnose brain tumors will probably remain one of the most common malpractice claims.

Subarachnoid Hemorrhage (SAH) The failure to diagnose SAH consistently results in the highest average and highest total indemnity for all claims involving diagnostic error.³² The neurologist must maintain a high index of suspicion for patients presenting with a severe headache of sudden onset (thunderclap headache), neck stiffness or low back pain suggestive of meningeal irritation, focal neurological deficits, cognitive impairment, or a history of premonitory symptoms suggestive of a sentinel bleed or expansion of an aneurysm. A good history of the current headache is essential, because even known migraineurs may suffer a SAH. The patient with a history or exam suggestive of a SAH should have an immediate CT scan of the brain, followed by a lumbar puncture (LP) even if the CT is negative for blood. The LP should include measurement of the opening pressure as well as evaluation for xanthochromia. Based on the clinical history, sequence of events, and time of presentation, as well as CT and LP results, it may be reasonable to proceed with MRA or arteriography.

NONMALPRACTICE LIABILITY

Neurologists must be cognizant of the morass of laws and regulations affecting their practice, which raise the specter of adverse licensing actions, 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 a myriad of *ad personam* (assault, manslaughter, homicide), economic (anti-kickback, self-referral, and antitrust violations; false claims) and regulatory (violation of Americans with Disabilities Act, Health Insurance Portability and Accountability Act, Emergency Medical Treatment and Labor Act) crimes.³³ The relevant

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legal principles governing these diverse areas are substantially the same for all specialties and are detailed elsewhere in this volume. Several examples underscore this broad liability:

- Increasingly, medical malpractice is subject to criminal prosecution. The facts of most emerging cases are *sui generis*, but the underlying theme is criminalization of gross negligence.³⁴
- A “whistleblower” complaint under the False Claims Act *qui tam* provision leads to criminal and civil prosecution of a neurologist performing excessive electrodiagnostic testing procedures.³⁵
- Economic credentialing may take many forms: a hospital surreptitiously discredits the staff neurologist who refused to participate in a fraudulent billing scheme involving neuroimaging studies.³⁶

LIABILITY OF THE FORENSIC NEUROLOGIST

Many neurologists have responded to managed care constraints by expanding their practice to include medical record reviews, independent medical examinations, and expert witness services. These lucrative activities generally do not invoke a physician–patient relationship (thus precluding a medical malpractice claim), but pose unique risks for administrative penalties, civil lawsuits, and criminal prosecution. Of note, malpractice insurance policies may not cover these services without a special rider.

Expert Witness

Anecdotal reports of neurologists advancing specious complaints are legion. One study of malpractice suits against neurologists over a 10-year period documented improper testimony in 37% of cases.³⁷ It is “alarmingly common for accomplished neurologists to hire themselves out for [one-sided testimony].”³⁸ These partisan experts have flourished behind the common law expert witness immunity shield and lack of professional oversight. Today, the pendulum is swinging back toward accountability with increased expert witness liability.³⁹ Friendly expert lawsuits (suits against experts by the party that retained them) are increasing.⁴⁰ The traditional immunity is not absolute,⁴¹ and the majority of states ruling on this issue have carved out exceptions to hold the expert liable for professional negligence. One state Supreme Court explained that an “absence of immunity will . . . protect the litigant from the negligence of an incompetent professional.”⁴² Perhaps this represents 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,⁴³ but that does not necessarily extend to nontestimonial expert activity (discovery of facts, literature search). Nor does it protect the expert from criminal prosecution for improper testimony or misrepresentation of a degree or license. The expert neurologist may also be liable for defamatory communications and negligent or intentional spoliation of evidence.

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.⁴⁴ Some boards have expanded the definition of medical practice to include expert testimony, allowing disciplinary action if warranted.⁴⁵ The AAN adopted *Qualifications and Guidelines for the Physician Expert Witness*,⁴⁶ promulgated a code of professional conduct for legal expert testimony,⁴⁷ and established a formal disciplinary procedure for errant neurologists with potential sanctions ranging from censure to expulsion.⁴⁸ AAN disciplinary actions may trigger the American Board of Psychiatry and Neurology to revoke certification. A recent Seventh Circuit Court of Appeals’ decision validated these types of disciplinary policies and stated in dicta that the American Academy of Neurological Surgeons had a duty to discipline a neurosurgeon for irresponsible testimony.⁴⁹

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 are listed in Box 47-1. It is important to remember that all deposition and trial testimony constitutes a permanent public record, which may be accessed from national repositories at any time.⁵⁰

CONCLUSION

The malpractice climate is increasingly adverse, and non-malpractice liability issues continue intruding into clinical practice. Integrating this chapter with an understanding of the entire volume will provide the neurologist with a guide to meet today’s legal challenges, thereby improving patient care and minimizing malpractice and nonmalpractice liability.

Acknowledgment

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Box 47-1. Guidelines for Expert Testimony

1. Fulfill the AAN qualifications before accepting a case.
2. Review all relevant medical information in the case.
3. Review the standard of care for the time of occurrence.
4. Perform adequate discovery of the facts.
5. Review and understand the relevant literature.
6. Properly assemble and present the case.
7. Avoid losing or destroying any evidence.
8. Provide accurate, impartial, and truthful testimony.
9. Avoid conflicts of interest.
10. Do not discuss the case outside the course of litigation.
11. Compensation must be reasonable, not contingent on outcome.

Endnotes

1. Physician Insurers Association of America, *Risk Management Review (Neurology)*, 2004 Edition.
2. *Id.* at v (Exhibit 5).
3. *Id.*
4. *Id.*
5. *Id.* at vi (Exhibit 6).
6. See generally Centers for Disease Control (www.cdc.gov) and American Heart Association (www.americanheart.org).
7. See, e.g., The NINDS rt-PA Study Group, *Tissue Plasminogen Activator for Acute Ischemic Stroke*, 333 N. Engl. J. Med. 1581–7 (1995); W. Hack et al., *Randomized Double-Blind Placebo-Controlled Trial of Thrombolytic Therapy with Intravenous Alteplase in Acute Ischemic Stroke (ECASS II)*, 352 Lancet 1245–51 (1998); W.M. Clark et al., *Recombinant Tissue-Type Plasminogen Activator (Alteplase) for Ischemic Stroke 3 to 5 Hours after Symptom Onset. The ATLANTIS Study: A Randomized Controlled Trial. Alteplase Thrombolysis for Acute Noninterventive Therapy in Ischemic Stroke*, 282 J.A.M.A. 2019–26 (1999); and J.M. Wardlaw, *Overview of Cochrane Thrombolysis Meta-Analysis*, 57(Supp. 2) Neurology S69–S76 (2001).
8. American Academy of Neurology, *Report of the Quality Standards Subcommittee: Practice Advisory: Thrombolytic Therapy for Acute Ischemic Stroke (Summary Statement)* (1996). See generally M.R. Frankel, *Acute Stroke*, 9(2) Continuum 31–52 (Apr. 2003).
9. See S.L. Hickenbottom and W.G. Barsan, *Acute Ischemic Stroke Therapy*, 19(2) Neuro Clin. 379–97 (2000). See also Frankel, *supra* note 8.
10. See, e.g., *Backlund v. University of Washington*, 975 P. 2d 950 (1999) (negligence and informed consent are alternative theories of liability).
11. A Position Paper of the American Academy of Neurology Ethics and Humanities Subcommittee, *Consent Issues in the Management of Cerebrovascular Diseases*, (1999).
12. See *Canterbury v. Spence*, 464 F. 2d 772 (D.C. Cir. 1972), cert. denied, 408 U.S. 1064 (1974) and its progeny.
13. M.A. Sloan, *Use of Anticoagulant Agents for Stroke Prevention*, 11(4) Continuum 97–127 (Aug. 2005).
14. *Id.* See also R. Llinas and L.R. Caplan, *Evidence-Based Treatment of Patients with Ischemic Cerebrovascular Disease*, 19(1) Neuro Clin. 79–105 (2001).
15. R.G. Hart and R.D. Bailey, *An Assessment of Guidelines for Prevention of Ischemic Stroke*, 59 Neurology 979–82 (2002). See also R. Chan and P. Pullicino, *Cardioembolic Stroke*, 9(2) Continuum 117–30 (Apr. 2003).
16. See, e.g., L.B. Goldstein, *Carotid Endarterectomy for Asymptomatic and Symptomatic Stenosis*, 11(4) Continuum 61–76 (Aug. 2005).
17. AAN Clinical Practice Guidelines, *Assessment: Carotid Endarterectomy—An Evidence-Based Review: Report of the TTA Subcommittee of the AAN*, 65(6) Neurology 794–801 (2005).
18. M.D. Hill, *The High Risk of Stroke Immediately After Transient Ischemic Attack: A Population-Based Study*, 62 Neurology 2015–20 (2004).
19. J.F. Fairhead et al., *Population-Based Study of Delays in Carotid Imaging and Surgery and the Risk of Recurrent Stroke*, 65(3) Neurology 371–75 (2005).
20. *Supra* note 17.
21. The Epilepsy Foundation of America provides a review of the laws in each state (www.efa.org).
22. *Tarasoff v. Regents of the Univ. of Cal.*, 551 P. 2d 334 (Cal. 1976).
23. See, e.g., *Harden v. Dalrymple*, 883 F. Supp. 963 (D. Del. 1995). Cf. *Praesel v. Johnson*, 41 Tex. Super Ct. J. 630 (1998).
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47. §6.4, *American Academy of Neurology Code of Professional Conduct* (2002) (readopted 2005–2006).
48. American Academy of Neurology, *Disciplinary Action Policy*, (revised 26 June 2004) (readopted 2005–2006). See also §§16, 17 *Articles and Bylaws of the American Academy of Neurology* (revised 2001) (readopted 2005–2006).
49. *Austin v. American Association of Neurological Surgeons*, 253 F. 3d 967 (7th Cir. 2001).
50. Some professional organizations maintain copies of depositions and court testimony (e.g., the Defense Research Institute in Chicago, and the Association of Trial Lawyers of America in Washington, D.C.).

