DePaul Law Review

Volume 18
Issue 2 *Issue 2 & 3 Summer 1969*

Article 17

The Impact of Serious Injuries: Medical Proof for Plaintiff

Lawrence V. Hastings

THE IMPACT OF SERIOUS INJURIES: MEDICAL PROOF FOR PLAINTIFF

LAWRENCE V. HASTINGS*

INTRODUCTION

THE IMPACT of a big or serious negligence case on a jury is dependent on the degree or amount of negligence shown to a jury as well as on the medical proof of the injury. It is important to recognize that if we have the same medical proof and injury in two cases, the case in which the liability appears more shocking or gross will result in a verdict which is more gross—or as has been stated by Harry Gair, one of the greatest trial lawyers of all time, "You've got to shock 'em to sock 'em." So one must shore up as fully as possible not only the medical proof but also the liability evidence. Prove prior notice, knowledge or warning to the defendant of the defect or condition which caused the accident. Show that a reasonable person or corporation would not have permitted such a condition to have existed, and have your expert so testify. In closing argument, point out that due to such utter disregard of the rules of the road or standard usage or custom, your client has suffered a severe and permanent injury which has already, and will in the future, cause him severe economic loss, pain and disability—all of which demonstrates his inability to lead a normal life for the rest of his days. It is only within this frame of reference that the jury will render full justice to your client. So bear down on liability and lead from strength with both your liability and medical witnesses.

* DR. HASTINGS *is Senior Partner in the firm of Green and Hastings in Miami, Florida. He is an Assistant Professor of Medicine at the University of Miami Medical School. He attended the University of Michigan Engineering School, and received his M.D. from Johns Hopkins School of Medicine. He received his LL.B from the University of Miami School of Law. He is a member of the Florida Bar.* DR. HASTINGS *formerly was Senior Assistant Surgeon with the United States Public Health Service. He is a member of the American Medical Association, American College of Legal Medicine, and a Diplomate of the American Board of Legal Medicine. He is a Fellow and Founding Member of the Law-Science Academy Foundation. He is the author of many publications including,* MEDICAL PREPARATION AND PRESENTING OF THE PLAINTIFF'S NEGLIGENCE CASE—TRIAL AND TORT TRENDS; POSITIVE HANDLING OF THE NEGATIVE X-RAY; *Use of Medical Illustrations in Soft Tissue Injury Cases, Presenting Plaintiff's Medical Proof,* AM. JUR. TRIALS, *Vol. 6; and Discovery and Evaluation of Medical Records,* AM. JUR. TRIALS, *Vol. 15.*

Leading from strength is an adage some of us know from the card game of bridge. It applies equally well in a law suit. For example, if you have several strong medical witnesses and others not as qualified or articulate, use only your best. Don't let your weaker witnesses dilute or water down your position. In most instances, the defendant will not call your uncalled weaker witnesses and thus be bound by their unfavorable testimony while at the same time permitting you the opportunity to lead such witnesses on cross-examination. Nevertheless, if you have decided to lead only with your strength, be sure to have full and adequate conferences with those witnesses whom you do not expect to call, so that your cross-examination will be well prepared in the event your opponent does elect to call them in his behalf.

Now, let us consider a few of the more common courtroom issues of medical proof involved in the larger and more substantial cases.

HEAD INJURIES & EXPERT TESTIMONY

How should your client's case of a head injury be handled where his doctor feels he has or may develop epilepsy? This medical battle of proof can be fraught with much difficulty, but, if successful, it can bring considerable reward to your case. The most frequent courtroom issue is whether the epilepsy is caused by the head trauma, or whether it has a hereditary or congenital basis. The former is generally called traumatic epilepsy and the latter, idiopathic epilepsy. Thus, when epilepsy is alleged by the plaintiff to be a consequence of an injury, the defense may contend that the epilepsy was caused by unknown congenital or hereditary factors, and not by his head trauma or brain injury.

What are the chances of developing epilepsy after a given head trauma and upon what factors does it depend? Simply stated, the possibility of the client developing epilepsy depends on (1) the *area* of the brain that receives the trauma, and (2) the *degree* of trauma or penetration into this area.

The brain has three meninges or membranes, which are tissue coverings. The outermost of these three membranes is known as the dura and is a tough fibrous tissue. The dura is sometimes, but not always, penetrated by a skull fracture. It should be noted, however, that traumatic epilepsy does not require penetration of the dura. According to Dr. D. Denny-Brown, Professor of Neurology at the Harvard

Medical School, even when the dura is not penetrated, the incidence of traumatic epilepsy is still about twenty per cent in severe head injury cases. On the other hand, if the dura has been penetrated, the subsequent liability to epilepsy is almost fifty per cent.[1]

The liability or chance of developing epilepsy also varies according to the particular area of the brain which is involved in the injury. For example, the possibility of developing convulsions is as high as 67.7 per cent if the central parietal region is involved in the injury. As Dr. Denny-Brown has observed: "The liability is greatest when the damage is to the frontal or parietal region of the brain, less in the temporal lobe and least in the occipital region."[2]

If epilepsy is going to occur, is it not reasonable that it should be manifested within a short period after the trauma? While this may appear reasonable at first thought (and such may well be the contention of the defense counsel), it just is not so. As a matter of fact, medical authorities point out that convulsions may occur at any time after an injury and, in fact, are not expected to occur until at least eighteen months or two years after the accident. Dr. Walter E. Dandy, late Professor of Neurosurgery at the Johns Hopkins Medical School, emphasizes the prevalence of such delay in the common cerebral injury:

> Moreover, there is a very definite and important time relationship between the accident and the onset of convulsions. Again excluding depressed fractures from which the convulsions may appear immediately after the accident, convulsions due to cerebral trauma rarely begin within a year and usually not until 18 months or 2 years after the accident.[3]

Dr. D. Denny-Brown states similarly with respect to convulsions:

> The attacks of epilepsy, due to brain injury, begin to appear at an interval of time after the injury, probably only when the scar shortens, as all scars do, *with age*, and exerts traction on the neighboring brain substance. Cases have been recorded in which the attacks did not commence until 15, 16 or 20 years after the injury, yet owing to their focal or Jacksonian character, were clearly related to the damage of the brain.[4]

Dr. Israel S. Wechsler, author of *Clinical Neurology*, has noted that "[w]hile the convulsions may become manifest soon after the injury,

[1] Denny-Brown, *Factors of Importance to Head Injury—A General Survey*, 1 CLINICS 1405, 1416 (1943).

[2] *Id.* at 1417.

[3] Dandy, *The Brain—Epilepsy*, in LEWIS'S PRACTICE OF SURGERY 329 (1942).

[4] *Supra* note 1, at 1418 (emphasis added).

they generally set in a few months or years later."[5] It has been similarly commented that though there is likely to be some premonitory symptoms in the interval, such as vertigo or paroxysmal headache, "traumatic epilepsy may develop as late as ten years or more after severe injury of the head."[6]

What about the treatment of this delayed type of traumatic epilepsy? Is the prognosis worse when it appears late than when it appears early? This indeed seems to be the case.

> Attacks which begin in the first month after injury respond well to treatment and tend to disappear, whereas those with later onset respond less well to treatment and often lead to *mental deterioration.*[7]

Dr. A. Earl Walker, Professor of Neurological Surgery at the Johns Hopkins Medical School, concurs in this observation, stating succintly, "that *a late developing epilepsy has a poorer prognosis* than one which comes on shortly after the head injury."[8]

What important medico-legal facts do we know about traumatic epilepsy in children? Traumatic epilepsy is much more likely to develop after head trauma in small children than in older persons. As Dr. Wechsler phrases it, "the younger the individual the graver the possibilities."[9] Dr. Guttmacher notes that Dr. Penfield, the world famous neurologist, "found the incidence of epilepsy higher after injuries occurring before the age of five than at any other time,"[10] and Dr. Guttmacher continues: "A certain number of unfortunate children develop chronic psychopathic behavior after head injuries, which has proven quite unmodifiable."[11]

This brings up an interesting corollary of the sequelae in children after head injury other than the possible development of epilepsy, namely, the development of behavioral changes in children. We are all aware of the fact that personality and behavior changes may occur after a head injury in adults. Both the likelihood and degree of personality and behavior changes after head injury are far greater in children.

[5] WECHSLER, CLINICAL NEUROLOGY 612 (9th ed. 1963).

[6] Stevenson, *Epilepsy and Gunshot Wounds of the Head*, 54 BRAIN 214, 223 (1931).

[7] *Supra* note 1, at 1418 (emphasis added).

[8] WALKER, POSTTRAUMATIC EPILEPSY 7 (1949) (emphasis added).

[9] *Supra* note 5, at 612.

[10] GUTTMACHER & WELHOFTEN, PSYCHIATRY AND THE LAW 153 (1952).

[11] *Id.*

Plaintiff's counsel often seeks to demonstrate an intellectual change in the child. On the other hand, the defense counsel will frequently seize, as a point in his favor, the fact that the child is progressing in his school work with the same degree of aptitude as before the accident. However, medical authorities point out that it is not the intellect, but the personality and behavior that are most often affected in children. The noted English neurologist, Sir Charles Symonds, observes: "In children the most prominent symptom following head injury is often *behavior disorder* associated with defective moral sense."[12] Dr. Strecker, Chief of Psychiatry at the University of Pennsylvania Medical School, agrees:

> While head trauma is rarely productive of definite psychosis, yet it does have important psychiatric implication. Sometimes in adults pronounced dispositional changes occur in the wake of head injury, and in children it is second only to encephalitis epidemics in conditioning extreme abnormalities of behavior. . . . There may be lying, stealing, setting fires, sexual assaults, homicidal attacks, etc. Treatment involves a long period of persistent and impersonal re-education usually in a setting detached from the home environment.[13]

These comments from medical authorities point out the obvious importance of interviewing the parents to determine whether there have been any changes in the behavior or personality of the child since the accident. The child's classmates, school teachers, and other persons close to the child should also be consulted. If it appears there has been some change in disposition of significant degree, a psychiatric consultation should be arranged.

What about the onset of these severe personality changes in children—are they to be expected shortly after the injury, or can they occur many years after the accident? And if they occur many years after the accident are they not susceptible to a defense counsel's attack that they are not related to the head trauma? Medical authorities support the proposition that the full blown picture may be expected to occur only many, many years after the accident. Drs. Bowman and Blau in *Injuries of the Brain and Spinal Cord and Their Coverings* explain that the visibility of personality change is minimized in the home environment.

> The *onset* of the personality change is noted shortly after convalescence from the

[12] Symonds, *Mental Disorder Following Head Injury*, 30 PROC. ROY. SOC. MED. 1081, 1089 (1937) (emphasis added).

[13] STRECKER, FUNDAMENTALS OF PSYCHIATRY 23, 37 (6th ed. 1963).

acute injury. In most instances, *however*, the case does not come to the attention of the physician, expecially the psychiatrist, until many years later. In Blau's series, the ages on admission to psychiatric hospital were greater, varying from 7 to 14 years. When the child is small and at home, the family seems able to tolerate his misbehavior. When, however, he becomes older and is thrown in greater contact with the outside world, the difficulty becomes accentuated and the family find themselves no longer able to cope with the situation. Puberty accentuates the personality difficulties in many cases as well as increasing the likelihood of misbehavior in the sexual field. The symptomatology of the syndrome may be characterized briefly as a complete reversal of personality. The previously normal child becomes asocial, unmanageable and unyielding to any forms of training. . . . The course of the condition is usually unfavorable, and eventually most severe cases have to be institutionalized.[14]

Is the brain wave or electroencephalogram proof positive as to whether there has been brain damage and as to whether the epilepsy is present or will become present? If the brain wave or electroencephalogram is negative may not the defense counsel seize upon this to assure the jury that there is no risk that epilepsy will develop? Such conclusiveness of electroencephalograms is definitely not supported by the medical literature. It has been stated many times that as a prognosticator the EEG leaves much to be desired. Dr. A. Earl Walker, writes:

Paroxysmal (spiking) abnormalities were present in only about one-fifth of the cases and there was no significant difference between the epileptic and the non-epileptic cases in this regard. It would seem, therefore, that the EEG alone cannot tell or foretell whether a patient with a severe head injury has or will have epilepsy.[15]

Dr. Israel Wechsler, in *Clinical Neurology*, similarly warns, "that normal electroencephalograms may be found in persons who have grand or petit mal convulsions and abnormal ones in persons who are free from either."[16]

Let us consider head trauma in the older person. We know that a hip fracture in an elderly person frequently results in his being bedridden for the remainder of his life. This is caused by poor circulation to the hip which occurs in the aging process. In view of this poor circulation, healing in the aged is frequently retarded, or non-existent,

[14] Bowman & Blau, *Psychotic States Following Head and Brain Injuries in Adults and Children*, in INJURIES OF THE BRAIN AND SPINAL CORD AND THEIR COVERINGS 360, 385-86 (S. Brock ed. 4th ed. 1960).

[15] Walker & Kaufman, *The Electorencephalogram After Head Injury*, 109 J. NERV. MENT. DIS. 383, 394 (1949).

[16] *Supra* note 5, at 581.

when the hip is broken. The slow or retarded healing due to poor circulation in an elderly person is true of almost any bone injury in the elderly. There is a similar effect noted in head trauma in older persons. Cerebral arteriosclerosis or, as the layman puts it, hardening of the arteries in the brain, is a prevalent condition in many older people. As a consequence, a relatively minor blow may cause severe pathology and disability in the elderly. Dr. Israel Wechsler writes that, "[a]n alcoholic or a person who already has cerebral arteriosclerosis may rapidly deteriorate and become demented following a blow to the head."[17] Dr. Alan Moritz, Professor of Pathology at Western Reserve Medical School, further elaborates:

> Old persons and chronic alcoholics are particularly susceptible to head injury and subdural hemorrhage may follow impacts so mild that little or no immediate attention is paid to their occurrence. The bleeding is often so slow that days or even weeks elapse before its occurrence is recognized.[18]

We are also aware that hypertension or high blood pressure is common in the older age person. Hypertension, along with the cerebral arteriosclerosis, makes the effects of the head injury much more severe, and in many, will cause the effects to be progressive. Dr. Donald Munro, Professor of Neurological Surgery at Harvard states:

> A craniocerebral injury causes enough disability when it occurs alone in a healthy individual. When the effects of a pre-existing disease are added, the resulting disability may increase out of all proportion. This is especially true when the circulation (for example: arteriosclerosis and/or hypertension) is involved. Not only is the local damage produced at the time of the injury greater but treatment is less effectual. Worst of all, post-injury disability is greater, and in a large group of such patients is progressive also.[19]

Dr. Munro further points out that cranio-cerebral injury may cause progressive mental deterioration in elderly people with arteriosclerosis.[20] Similarly, Dr. Karl Bowman, Professor of Psychiatry at California Medical School, and Dr. Abram Blau of New York University College of Medicine note that "head trauma may act as a precipitating agent

[17] *Supra* note 5, at 611.

[18] Moritz, *Mechanisms of Head Injury*, 117 ANN. SURG. 562, 570 (1943).

[19] Munro, *The Late Effects of Craniocerebral Injuries*, 117 ANN. SURG. 544, 559 (1943).

[20] "A slowly developing rather generalized type of mental deterioration often starts in elderly people with arteriosclerosis as the result of craniocerebral injury. It is similar to the usual type of senile deterioration and interferes with mental activity, judgment, cooperation and memory. Motor and sensory paralyses, as such, are not common. Treatment is unavailing." MUNRO, CRANIOCEREBRAL INJURIES 234 (1939).

or may exaggerate symptoms in the psychosis associated with cerebral arteriosclerosis."[21] It should also be noted that if the head trauma causes a skull fracture, the fracture in the elderly person is likely never to heal by bony union.[22]

A frequent courtroom controversy involves the diagnostic procedure of the spinal tap. A defendant will frequently contend that if a severe brain injury or hemorrhage has actually occurred, blood should appear in a spinal fluid tap, and if the spinal fluid does not show blood, no injury or hemorrhage exists within the brain. Medical literature on this subject clearly reveals that even in cases of hemorrhage or severe brain damage, blood will not necessarily be found on the spinal puncture.

> The end products of trauma to the brain are precisely the same as those of trauma elsewhere, namely, edema, hemorrhage and destruction of tissue. And just as following trauma elsewhere, the relative proportion of edema and blood varies tremendously. An injury may produce nearly all blood and little or no hemorrhage or any combination of both.[23]

The English neurosurgeon, Sir Charles Symonds, has stated that even with a subarachnoid hemorrhage of the brain, there may on occasion be cases where other signs of brain injury, such as a loss of consciousness, positive Babinski, or a change in temperature and pulse, are the only signs present.[24] Other authorities present similar findings. Dr. G. F. Rowbotham, in his text *Acute Injuries of the Head* says that, "[t]he presence of blood in the cerebrospinal fluid . . . will be found in 75% of cases of severe concussion."[25] Dr. Russell L. Cecil and Dr. Robert F. Loeb, in their *Textbook of Medicine* report:

> The cerebrospinal fluid is usually bloody and under increased pressure in patients with cerebral hemorrhage, *but occasionally the fluid may be clear.* . . . Examination of the cerebrospinal fluid is of value in the differential diagnosis between cerebral thrombosis and subdural hematoma. The finding of a bloody or xanthochromic fluid under an increased pressure renders a subdural hematoma the more likely diagnosis. Occasionally, however, the fluid in a patient with subdural hematoma *will be clear*, with or without an increase in the pressure.[26]

[21] *Supra* note 14, at 401.

[22] *Supra* note 19, at 548.

[23] *Supra* note 3, at 255.

[24] Symonds, *Concussion and Contusion of the Brain and Their Sequelae*, in INJURIES OF THE BRAIN AND SPINAL CORD AND THEIR COVERINGS 82 (S. Brock ed. 4th ed. 1960).

[25] ROWBOTHAM, ACUTE INJURIES OF THE HEAD, THEIR DIAGNOSIS, TREATMENT, COMPLICATIONS AND SEQUELS 141 (4th ed. 1964).

[26] CECIL & LOEB, TEXTBOOK OF MEDICINE 615 (11th ed. 1963).

RUPTURED DISC AND MYELOGRAM

A herniated intervertebral disc often is a particularly serious injury with severely disabling long-range consequences, but the nature of the soft tissue injury involved requires careful presentation of medical evidence in order to convince the jury of the existence of a ruptured disc.[27] Moreover, plaintiff often faces the difficulty of demonstrating to the jury's satisfaction that the absence of a positive myelogram is not significant and does not constitute a "devastating medical defense" as asserted by defendant. We shall now touch lightly upon the value, hazards and shortcomings of the myelogram as a diagnostic procedure in an alleged ruptured disc case.

Where the defendant in a ruptured disc case emphasizes, purely as a matter of trial tactics, the plaintiff's failure to have undergone a myelogram or a spinal tap and requests the court to compel the performance of such tests, the plaintiff's attorney can effectively point out that neither of these tests will necessarily prove or disprove the presence of a ruptured disc. Furthermore, plaintiff's counsel can also show that the current medical opinion is that disc herniations or ruptures can be diagnosed without the aid of such tests. For instance, Dr. Walter E. Dandy cautions that the "overwhelming percentage of vertebral discs can be diagnosed and localized by the history and examination alone, and all accessory diagnostic tests (even lumbar punctures) can and should be avoided."[28]

With regard to the diagnostic effectiveness of the myelogram, it has been asserted that, "only about two-thirds of the positive myelographic studies have been confirmed at operation."[29] This evaluation is sup-

[27] *See generally* Hastings, *Positive Handling of the Negative X-ray (Use of Medical Illustrations in Soft Tissue Injury Cases)*, in PERSONAL INJURY ANNUAL 303 (1961) (emphasis added).

[28] Dandy, *Concealed Ruptured Intervertebral Discs—A Plea for the Elimination of Contrast Mediums in Diagnosis*, 117 J. Am. Med. Ass'n 821, 823 (1941).

[29] SHANDS, HANDBOOK OF ORTHOPAEDIC SURGERY 416 (6th ed. 1963). Other authorities impute a somewhat greater degree of conclusiveness to the myelogram. *See, e.g.*, KEY & CONWELL, FRACTURES, DISLOCATIONS AND SPRAINS 345 (7th ed. 1961), wherein the authors state: "We use the myelogram as a guide in our operative procedure but are not limited by it. If its result is negative and we think that the patient should be operated upon, we operate upon him. If positive, it is valuable corroborative evidence of our diagnosis. We find that it is accurate in about 80% of our cases and that a discrepancy between the myelogram and the operative findings occurs in about 20% of our cases. Usually these discrepancies are minor, but in about 5% of the cases major discrepancies have been noted."

ported by the studies of other authorities. Drs. Roy R. Grinker and Paul C. Bucy in their textbook, *Neurology*, state that "myelography may be completely negative in cases where a herniated intervertebral disc is present, and may in other instances provide falsely positive roentgenograms."[30] Dr. Walter E. Dandy has pointed out that certain discs are concealed and therefore will not show up on a myelogram.

> The disclosure of this concealed type of disc is an additional reason for giving up iodized oil (pantopaque) air or other tests. They can't possibly show the lesion, and with negative evidence the patient may well be deprived of operative cure and passed along as a neurasthenic. . . . The concealed disc bulges so slightly that it would never be disclosed by iodized oil or air injections into the spinal canal and will be found at operation only by a careful inspection beneath the dura.[31]

In addition, counsel can prove that the introduction of a needle into a plaintiff's spine, as is necessary in either a myelogram or a spinal tap, may be attended by some danger to the plaintiff. It is quite generally recognized that an improperly performed lumbar puncture may cause injury to the soft inner core or nucleus pulposus of an intervertebral disc.[32] Also, the radiopaque fluid that is injected in preparing a myelogram may cause arachnoiditis or an inflammation of the tissues about the spinal cord. On this subject, Dr. Ralph Ghormley, Professor of Orthopedic Surgery at the Mayo Clinic, notes: "[t]hat the various intraspinal contrast media used in myelography may play a part in setting up an adhesive arachnoiditis seems likely but it is difficult to determine how often or how extensively this occurs."[33] With characteristic purpose, an article in the *Journal of Bone and Joint Surgery* suggests more restrained use of tests utilizing iodized oil:

> (1) Clinical and laboratory data presented suggest that pantopaque and other io-

[30] GRINKER & BUCY, NEUROLOGY 337 (5th ed. 1943).

[31] *Supra* note 28, at 823. Additional support for the view that a myelogram is rarely necessary, and that a disc may be found despite a negative myelogram is contained in BRAIN, DISEASES OF THE NERVOUS SYSTEM 789 (4th ed. 1951): "A myelogram may demonstrate a filling defect (indicating disc) but it is rarely necessary and a disc may be present in spite of a negative myelogram."

[32] Coggeshall, *Backache*, in COMROE'S ARTHRITIS AND ALLIED CONDITIONS 1018 (4th ed. 1949). When the test is properly performed, the needle should enter between the spinous processes of two adjacent vertebrae and enter only the spinal canal. A diagram demonstrating how a disc can be damaged by a lumbar puncture that passes beyond the spinal canal and enters the intervertebral disc may be found in 32 J. BONE JOINT SURG. 981 (1950).

[33] *Multiple Operations for Protracted Lumbar Intervertebral Disc*, 29 PROC. MAYO CLIN. 546, 549 (1954).

dized oils may contribute to or possibly cause severe reaction within the subarachnoid space.
(2) Verbal communication indicating similar experience among orthopaedists and neurosurgeons has suggested to us that there is need for a more critical evaluation of the properties of pantopaque and other iodized oils and a more conservative approach to their use than is often practiced. The importance of complete removal of the substance is evident.[34]

As a result of these dangers, it has strongly been urged that a myelogram should never be performed for purely diagnostic purposes, and that its use should be confined instead to those cases in which the patient has consented, if the study should prove to be positive, to proceed with the operation involved. This operation, moreover, should be performed on the same day as the myelogram.[35]

CONCLUSION

As I have tried to show in the preceding discussion, the impact of serious injuries upon the jury, and the consequent return of a just and substantial verdict, depends in large measure upon plaintiff's medical proof. Unless counsel is well prepared to win common medical courtroom controversies, including, but not limited to, questions concerning head injuries and ruptured discs, the magnitude of plaintiff's injury will not be reflected in the final award.

[34] Hurteau, Baird, & Sinclair, *Arachnoiditis Following the Use of Iodized Oil*, 36A J. BONE JOINT SURG. 393, 400 (1954). Further complication may be caused by the fact that pantopaque, the radiopaque fluid that is most commonly used in performing myelograms, may shortly after injection find its way into the intracranial cavity. "Whenever even a small amount of ethyl iodophenylundecylate (pantopaque) is allowed to remain in the spinal subarachnoid space, it can easily find its way into the intracranial cavity in the following days or weeks. It is not always possible to remove all the ethyl iodophenylundecylate." *Queries and Minor Notes—To the Editor*, 156 J. AM. MED. ASS'N 1296, 1299 (1954).

[35] In the words of Dr. J. Grafton Love of the Neurosurgical Department of the Mayo Clinic: "Dr. Mixter has warned about the injection of iodized oil, and we have adopted the attitude. We have stopped injecting iodized oil for diagnostic purposes unless the patient has already planned—if the study is positive—to proceed with the operation, preferably the same morning. The injection is made in the operating room, Dr. Camp does his fluoroscopy, then the patient is taken back to the operating room, and a laminectomy is performed. If radio-opaque oil is used to localize the lesion, it should be injected into the subarachnoid space in the lumbar segment and the roentgenologic examination should be carried out on the day of the operation." Love, *Protruded Intervertebral Discs—With a Note Regarding Hypertrophy of Ligamental Flava*, 113 J. AM. MED. ASS'N 2029, 2035 (1939).