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APoplexy in Law

Kurt Garve*

Defendant was convicted of manslaughter. He, together with companions, had attacked deceased, he holding the victim, while an accomplice struck a blow on the left side of decedent's head, from which he became unconscious.

After the attack the victim regained consciousness, but he was delirious for a short time. While being taken home he complained frequently of pain in his head. He grew weaker, staggered, and had to be given assistance. When he finally arrived home, he exclaimed to his mother: "Oh, mother, my head hurts me. One held me, while the other hit me." He became unconscious again and died the following day.

An autopsy disclosed as cause of death a fracture of the skull and a hemorrhage of the brain. There was a large blood clot in the region of the left temple. A small hole was radiating with star-shaped fragments running from the fracture itself.

The defense claimed that it was prejudicial error to receive in evidence the statement made by decedent to his mother, since it formed no part of the res gestae. On appeal the reviewing court thought otherwise. Conviction was affirmed.¹

Though the statement was made forty minutes after the assault should it have been admitted in evidence as part of the res gestae? What grounds may be brought forward by either prosecution or defense in this regard? These, and other, questions have relation to apoplexy in law, with which problem this paper will deal.

MEDICAL FACTS²—MISCELLANEOUS CASES

Generally, causal connection between apoplexy and violence must be established. Yet, apoplexies are not always results of external force.

The principle upon which strokes rest is that some natural or artificial vulnerating force, of whatever description, has:

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¹State v. Humphrey, 173 Minn. 410, 217 N. W. 373 (1928).
1. Acted from within by raising the blood pressure, as in strain; 2. attacked the victim from without, as where a blow against the head is suffered; 3. reduced the vitality to such an extent that the heart gives way and becomes weakened in its blood tension maintaining apparatus. The blood pressure becomes unduly lowered and apoplexy results.

The morbid process of paralysis is not uniform. One speaks of
1. "Circulation-vessel wall tear" apoplexy or hemorrhage. The causative agency raises the blood pressure beyond the breaking point of the affected blood vessel; 2. "Circulation-stagnation" apoplexy, or thrombosis. The vulnerable force acting upon the heart-pump slows down the blood current to such an extent that the blood stops to move from place to place; 3. "Circulation-blockade" apoplexy, or embolism. Some particle of tissue, or some foreign matter, such as air, enters the brain circulation and closes up the blood vessel cork-like. No blood supply can reach the brain substance involved; 4. "Crushed skull and brain tissue" apoplexy. A violence penetrates the bony skull, its linings, and the brain tissue, and destroys portions thereof and blood vessels therein; 5. "Circulation-corrosion" apoplexy. An infected particle of some mother area, as in blood poisoning or syphilis, from the inside or outside of the blood vessel, eats thru its coats so that paralysis occurs.

There are also combinations of the above-given categories of paralysis:
6. A "double vessel-wall tear" apoplexy occurs, when one tear is followed sooner or later by another one. The first tear is often due to violence, while the other one is due to either another trauma or natural causes; 7. "Blockade upon blockade" apoplexy. There is a succession of corking stoppages; 8. "Stagnation after stagnation" apoplexy. Two or more heart failures follow in succession; 9. "Stagnation after blockade" apoplexy. Here, there is a combination of two different morbid processes, one following the other one; 10. "Blockade after stagnation" apoplexy. The weakened heart first causes a stagnation, thereafter a blockade results.

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3Samoskie v. Philadelphia & Reading Coal & Iron Company, 280 Pa. 203, 124 Atl. 73 (1924)—W. C.
4Yodis v. Philadelphia & Reading Coal & Iron Company, 269 Pa. 586, 113 Atl. 73 (1921)—W. C.
5State v. District Court of Ramsey County, 147 Minn. 10, 179 N. W. 217 (1920)—W. C.
6See n. 3.
7See n. 5.
8United States Fidelity & Guaranty Co. v. Green, 38 Ga. App. 50, 142 S. E. 464 (1928)—W. C.
12See n. 5.
12a. Ibid.
The remote causes of apoplexy are manifold. Degenerative processes of the brain vessels, particularly of the arteries, their hardening, syphilis, lead poisoning, alcoholism, and so on are contributors to apoplectic strokes. Certain heart diseases, kidney trouble, tuberculosis, pneumonia may induce apoplexy. Where none of such ailments are to be found, a congenital weakness in some spot of the arterial system of the brain may be detected upon autopsy. However, the violence may be so strong that even a normal blood vessel is ruptured.  

The proximate causes of a stroke may be found in physical strain, efforts to run fast, other over-exertion, sunstroke, heat stroke, and mental excitement. Vomiting, straining at stool, the efforts of coughing violently, use of strong heart stimulants, starvation and malnutrition, a cold bath, and an "apoplectic habit," and no apparent perceptible cause at all are apt to enter the clinical picture. The proximate causes are colorless per se. The question of proximate causation is, therefore, to be answered in the light of the individual facts of the given case under judicial investigation.

While the course of apoplexy is not uniform, ordinarily there are some premonitions. There are headaches, attacks of dizziness and giddiness, insomnia, sensation of numbness in one arm or one leg. There may be ringing in the ears, noises like bells or whistles heard. Flashes of light before the eyes, a sudden sense of blindness in one eye, passing off in a moment, a subjective sense of bad odor, or an unusual taste, nose bleeds, and similar signs show the irritation of the brain areas and the high blood pressure. But it is also true that such prodromata may never be followed by an apoplectic attack.

Then comes the stroke itself. It is ordinarily of sudden onset. But, there may be no unconsciousness. Where there is unconsciousness, the face is flushed with a somewhat bluish tinge. There is complete relaxation, often with involuntary evacuation of bowels or bladder. The mouth is drawn to one side, with dribbling of saliva. There is snoring breathing. One side of the body is paralyzed as shown by the marked contracture of the arm or of the leg, or of both.

It is characteristic of such apoplectic strokes that the paralyzed side is ordinarily opposite to the location of the morbid area in the brain.

As times goes on the paralysis subsides, consciousness returns, and the patient makes a more or less uneventful recovery. Yet, usually some remnants of the apoplexy remain.

Every possible deviation from the "typical" case is to be observed. The ambiguous nature of the apoplectic symptoms may offer diagnostic difficulties to even the best of physicians. There are irregular types of apoplexy, such as paralytic strokes without loss of consciousness, or attacks of gradual onset.

13In Emmett v. Key System Transit Co., 12 Cal. Ind. Acc. Com. Dec. 194 (1925), a cerebral hemorrhage, resulting in death of a motorman, was held to have been due to a collision. Post-mortem disclosed no morbidity otherwise sufficient to account for the accident.

Apoplexies are apt to recur. Seven or eight attacks may precede death. The symptoms of a second attack, for instance, may be the same as those of the preceding attack, or they may be different. Also, there is no rule by which one can determine the length of time or rest between the intervals. A few months, a year, five years, or even ten years may elapse before a recurrence happens. The average is about two years.

When, finally, the terminal condition of an attack of apoplexy has been reached, there remains a certain mental enfeeblement, with lack of control of emotions. After every new attack these mental deteriorations become more and more pronounced. Yet, here also deviation from this regular type may occur, much depending upon the underlying or remote cause.

Some legal issues in connection with apoplexy may be disposed of at this place. It is common knowledge, of which courts will take judicial notice, that arteriosclerosis is a frequent cause of death. One in an advanced state of such a hardening of the arteries is very dangerously ill. In an insurance case, pertaining to death by accidental means, not contributed to by a disease, an instruction that arteriosclerosis is not a disease has been held to constitute reversible error.

It is also common knowledge that wrestling requires an unusual amount of exertion, thus inducing apoplexy. Medical expert testimony in such a case is unnecessary where death is immediately and directly, or naturally and probably, the result of such an accident.16

Negligence of a telegraph company in failing to inform timely the plaintiff that her father has been stricken with apoplexy so that she could not be present at his burial, does not make defendant company liable for mental suffering occasioned by the suspense in not knowing the condition of the patient during the time plaintiff was delayed. Such mental suffering was not one of the natural and usual results of such negligence within the contemplation of the contracting parties.17

Evidence of a fracture of the skull and of injury to one of plaintiff’s ears, resulting in an impairment of hearing and memory, may be introduced, when the pleadings alleged that plaintiff was injured internally, externally and permanently about the head, body, and the limbs so that he became sore, sick, and disabled, etc.18

Where a workman in an elevator shaft was killed by an iron pipe falling from above, evidence could be introduced showing that a heating contractor’s employees were responsible. The doctrine of res ipsa loquitur may be applied; the case, how-

17Western Union Tel. Co. v. Edmondson, 91 Tex. 206, 42 S. W. 549 (1897), reversing 40 S. W. 622 (1897).
ever, being one for the jury, if there be any evidence from which negligence of the defendant could be inferred. 19

Furthermore, negligence of an attorney may be excused and a new trial be granted after judgment on default, where in consequence of an apoplectic stroke and loss of memory defendant fails to attend trial, though a summons had been served after the attack of the apoplectic stroke. 20

Medical testimony which merely shows a possibility that cerebral apoplexy was caused by the accident does not constitute sufficient proof of causal connection. 21

It has also been held that $10,000 for a fracture of the skull, probably interfering with the victim's life work and possibly causing fits, and inability to work together with great pain, are not excessive damages. 22

Other instances pertain to insurance and workmen's compensation. Thus, totally disabled, within the meaning of workmen's compensation law, is a man suffering from a brain lesion, partial loss of speech, mind, and memory, with an abnormal condition of his left side. 23

Concussion of the brain warrants total disability under an insurance policy, even though insured returned to work after the accident, but complained of headaches and inability to see. A condition resulting in entire inability to work developed gradually. There was a one-sided paralysis, accompanied by speechlessness. 24

As the apoplectic stroke may come on suddenly in a previously apparently healthy person, default in giving notice by reason of the physical incapacity of an accident policy holder may be excused, even though written notice be required as a preceding condition to receiving sick benefits. 25

Likewise, in workmen's compensation cases immediate notice to the employer, though required by law, ought not to be required, and lack of such notice ought not to absolve the master from liability in many cases of apoplexy. In emergencies of such a grave character an employee cannot be expected to go thru the


23Roach v. Oswald Lever Co., 274 Pa. 139, 117 Atl. 785 (1922). But injury and death are distinct from each other: City of Milwaukee v. Roth, 185 Wis. 507, 201 N. W. 251 (1924).


2514 R. C. L. 1333/34, § 504, Perm. Supp. Ed., though there is good authority to the contrary.
red tape of regulations of workmen's compensation acts, while he is in imminent
danger of life, and while there is need for instantaneous care.\textsuperscript{26}

Since apoplexy affects both sexes, such ailment is not a disease peculiar to
the male only because death from paralytic stroke occurs more frequently in men
than in women.

One suffering from a fracture of the skull due to an automobile accident was
entitled to reimbursement by an insurance company. The policy provided for pay-
ments under an "immediate medical and surgical aid as is imperative at the time
of the accident" clause. The insured had been picked up and taken to a doctor's
office, and thereafter taken to a hospital, where an operation had been performed
for the removal of a piece of skull. But under such a clause the patient, once he is
removed from the hospital, is not entitled to reimbursement for further medical
or surgical care rendered.\textsuperscript{27}

Where la grippe is contracted three months after the issuance of a sick benefit
policy, and where there was proof of hardening of the arteries, which could not
have come on suddenly, the insurer nevertheless must pay sick benefits, though the
policy provides for indemnity only in case the disability was caused solely by some
morbidity having come into existence after the date of the policy. There was no
proof that the arteriosclerosis could not have come into being within such three
months.\textsuperscript{28}

In regard to breach of promise to marry, it cannot ordinarily be excused
upon the theory of communicability of disease to the spouse or to the offspring,
except in case of syphilis.\textsuperscript{29} Yet, consummation of marriage may endanger the
life of the ailing party, since sexual intercourse may be attended by such a great
mental excitement that apoplexy may result.\textsuperscript{30}

Where a daughter-in-law was treated by her mother-in-law as a member of
the household for a period of twelve years, the presumption arose that such an
association existed up to the time of the mother's death. The daughter was not en-
titled to compensation for services rendered to the mother, suffering from apoplexy
making her helpless during the last few years of such an association. Even if the
contrary be true, nevertheless another presumption arises that such compensation
has been turned over at stated intervals during the period covered.\textsuperscript{31}

\textsuperscript{26}R. C. L. 825, § 113, Perm. Supp. Ed.
\textsuperscript{27}Laidlaw v. Hartford Accident & Indemnity Co., 254 N. Y. 391, 173 N. E. 557 (1930).
\textsuperscript{28}Southern Surety Co. v. Farrell, 79 Colo. 53, 244 Pac. 475 (1926). rehearing den. (1926).
\textsuperscript{29}See "Modern Clinical Syphilology", Stokes, pp. 1011/2, 1927, W. B. Saunders Company,
Phila. & London. Whether or not syphilis is curable is doubtful.
\textsuperscript{30}R. C. L. 166. § 23, Perm. Supp. Ed.
\textsuperscript{31}Brown v. McCurdy, 278 Pa. 19, 122 Atl. 169 (1923).
COURSE OF APopleXY—MENTAL CAPACITY

The apoplectic stroke has now occurred. The pressure-symptoms in cerebral hemorrhage, or the "blood emptiness" in "stagnation" or "blockade" apoplexies exert their influence upon the mind. In a great many cases it is reasonable to assume that there is temporary lack of mental capacity at this time.

But, thereafter, the outlook depends somewhat upon the condition which caused the stroke. Great caution must be exercised in giving an estimate based solely upon physical recovery. The mental incapacity attendant upon apoplexy is never recovered from, absolutely and entirely. The actual obscuration of ideas during the attack, the inability to comprehend, to think, to judge, and to act, subside only slowly. They will ordinarily be even worse after every new stroke than they were before. Yet, gradually the flow of ideas will become clearer as time passes on. It is possible for a man to make a will "within two weeks" of the attack. Mental capacity, in other words, increases the more remote in time the apoplexy-climax is, and decreases the nearer the attack itself is to be found.

All this does not mean that incapacity must have existed only at the time of the attack, or thereafter, or before, by reason of apoplectic danger. There are diseases in which the paralytic stroke is no more than a mere end-event of morbidities which already, prior to the stroke, may have produced unsoundness of mind, though lucid intervals may have existed.

In "blockade" apoplexy due to blood poisoning, for instance, a period of profound germ-intoxication may have antedated, with spells of delirium and with signs of confusional insanity in the forefront. Auto-intoxication, due to retention of bodily waste-products, as in uremic poisoning, may have incapacitated the patient.

Alcoholism of a chronic nature may impair the mind and may be marked by specific, fixed delusions of conjugal infidelity, affecting the will. The mental changes in general paresis, a syphilitic infection of the central nervous system, are already noticeable in the earlier stages of the disease. The mental decadence becomes more and more pronounced as the ailment progresses towards the end-stages.


83 Loss of consciousness, convulsions, mental symptoms, delirium, maniacal and melancholic states, see Struempell, n. 2., vol. 1, p. 738.

84 See n. 32, under "Chronic Alcoholic Insanity", p. 212. But see Catt. v. Robins, 305 Ill. 76, 137 N. E. 101 (1922), rehearing den. (1922), saying (syll. 6.) that "while it may be a matter of common knowledge from the experiences of men that the excessive and habitual use of intoxicants for a long time impairs the mental capacity of the individual using it, still it cannot be said that this is true in all cases, as a matter of law, and it is not proper to instruct the jury that such use of intoxicants will necessarily have that result."

85 See in n. 32, under "General Paresis, Paretic Dementia, pp. 228 et seq.
Drug addiction or drug administration may contribute to mental unsoundness. Even mere old age may warrant a finding of mental incapacity, since there is a degeneration of the brain masses which show shrinkage to a marked degree.

The mentality-phase of apoplexy in all such cases becomes an incident to the preceding shortcomings of the progressive infirmity. It may, however, be assumed that an apoplectic attack, in addition, would generally mark a certain culmination of the ailment and its mental disintegration.

Simple hardening of the arteries, on the other hand, may offer great difficulties in evaluating the qualitative changes of the mind prior to a stroke even in cases in which the arteriosclerosis is most pronounced in the vessels of the brain. "Blockade" apoplexy, without infection and due to purely traumatic influences, as in fractures of the bones, does not give any clue as to mentality, and, in absence of any pertinent history to the contrary, mental capacity ought to be assumed to exist.

Consequently, it may be said in a very broad sense and as a matter of very loose application that:

1. Contractual and testamentary capacities are subject to existing apoplexies;
2. Other diseases may lay the foundation of, or may cause, mental incapacity, independently of apoplexy;
3. Capacity diminishes with increase of drug medication affecting the mind, such as opiates;
4. A lucid interval must always be reckoned with;
5. Capacity increases the more remote in time the apoplectic attack is, and vice versa;

36 Ibidem under "Cocainism", delusions of marital infidelity. As to morphinism it has been said that addicts are at their best when under the influence of their customary dose, and at their worst when in need thereof. Illinois Medical Journal, vol. 66, No. 3, Sept., 1934, an editorial, quoting Judge McDowell's opinion in U. S. Court of Appeals decision, not cited.
37 Ibidem under "Senile Insanity", pp. 306 et seq.; delusions of persecution, non-realization of the present, and the renewal and survival only of the past. See also Nelson v. Blake, 38 N. J., 173 Atl. 625 (1934), appointment of conservator of estate prior to apoplexy.
38 Struempell, n. 2., under "Arteriosclerotic Brain Disease", general weakness of the mind, loss of memory, of mental energy, acitivity, and interest. And see: Ware et al. v. Morton et al., 288 Mass. 107, 192 N. E. 505 (1934), testator suffering from nervous disease, heart ailment, high-blood pressure, and cerebral arteriosclerosis.
40 Beadle v. McCrabb et al., 199 S. W. 353 (Tex. 1917). But see McManus v. McManus, 179 N. W. 603 (Iowa 1920), not opiate; also Ware v. Morton, 288 Mass. 107, 192 N. E. 505 (1934).
6. The issue of specific delusions may affect the interpretation of morbidity of mind in apoplectic cases;\textsuperscript{42}

7. The issue of undue influence may alter the course of proceedings, since the question of capacity may not be involved directly;\textsuperscript{48}

8. A higher degree of capacity is necessary in cases of legal transactions other than making a will.\textsuperscript{44}

How far may either party to a contest go back in the clinical history of the apoplectic testator in order to prove mental competence or incompetence? According to a California case\textsuperscript{45} primarily the time of actual execution of the will and the actual condition of the testator at that time is controlling. The mental conditions before and after execution are important only in so far as they throw light upon the mental condition at the time of execution of the instrument itself. They act merely as commentaries.

In an Illinois case\textsuperscript{46} of insane delusions affecting testamentary disposition, the standard is set of a reasonable length of time prior to and after the taking place of the transaction. This is based upon the ground that these mental conditions are not temporary ailments, but rather diseases of gradual development and of progressive character, growing worse by degrees.

Why should not heredity and the whole life history of the testator be gone into in order to show insanity? Delusions existing in 1892 may have a direct bearing upon the issue, when it is shown that they returned toward the end of testator's life in 1930 by reason of apoplexy or otherwise.\textsuperscript{47} The same is true in regard to cases of undue influence.\textsuperscript{48}

The true rule, in the opinion of the writer, should be much more extensive concerning the clinical history than at first blush the courts might admit. Much would depend upon whether or not there was a disease with a nucleus of insanity therein. Also, if the disease itself be denied, one might have to prove its existence. Thus, since general paralysis may be the cause of apoplexy and of testamentary incapacity, one might be compelled to go back to the primary inocula-
tion with syphilis. Take the case of chronic alcoholism. How could one prove the degeneration of the brain without showing the length of time within which alcoholic oversaturation of the body occurred?

Where, however, ailment and nucleus of insanity are admitted, the limitation would probably and properly consist in the giving of the clinical course of the ailment from that time on when mental incapacity could have started on its way. To leave in pre-apoplectic facts, together with evidence of the post-apoplectic state of mind, would then be a means of judging with considerable accuracy in what state of mental health the testator was at the time of the making of the will.

Where a transaction, testamentary or otherwise, is undertaken and completed prior to the attack of apoplexy, unsoundness of mind cannot be predicated thereupon. A complex of psychosis, old age, bodily infirmities, delusions, eccentricities, attempted suicide, and hardening of the arteries of the brain, while apt to lead to an apoplectic stroke and thus constituting the "pre-apoplectic period" must be decided upon its own merits. Similarly, chronic alcoholism and a stroke five years after the execution of the will, lays stress upon the alcoholic condition more than upon the stroke. In such a case a decree for proponents may be warranted where the instrument is a holographic will in good form and excellent penmanship, and where the maker thereof had been actively engaged in business and had been a director and auditor of a large banking institution at the time of the execution of the will. He had continued to do so until the stroke occurred. See also Kast v. Turley, Stevens v. Stevens, McManus v. McManus, and Prescott v. Merrick.

Next comes the "premonition" phase. Except for some tendency to use one word in the place of another one, a somewhat defective memory, a confusion of thoughts, or an inability to think without conscious effort, a temporary trouble in speaking, or a marked emotional state due to lack of self-control, there is nothing to indicate mental unsoundness. While these prodromata may extend over several days or weeks, or even months, it is nevertheless doubtful whether or not mental incompetency exists.

The attack itself has been mentioned already. Where the patient recovers consciousness after the attack, the mind is usually very dull. Frequently it takes

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49bidem, medical treatment for chronic syphilis 5 years prior to making of the will.
50In re Buechley's Estate, 278 Pa. 227, 122 Atl. 287 (1923); Rowcliffe v. Belson, 261 Ill. 566, 104 N. E. 268 (1914), case of periodical drinker in earlier years of life.
51McIntosh v. McIntosh, 263 Mass. 315, 160 N. E. 814 (1928).
52In re Buechley's Estate, 278 Pa. 227, 122 Atl. 287 (1923); 283 Pa. 107, 128 Atl. 730 (1925).
53111 Conn. 253, 149 Atl. 673, (1930), also Nelson v. Blake, 173 Atl. 625 (R. I. 1934), syll. 3.
55179 N. W. 603 (Iowa 1920), diabetes, amputation of limb, arteriosclerosis, opiates, apoplexy, conveyance-transaction, judgment for defendant.
5646 N. D. 67, 179 N. W. 693 (1920), rehearing den. (1920). See also Preston v. Preston, 149 Md. 498, 132 Atl. 55 (1926), lost will.
some days before the victim recognizes his surroundings. As the mind becomes clearer, a definite estimate of his mental condition becomes possible. In other cases the mental symptoms become more and more pronounced. Confusion of thought is evident in speech and action. A dementia develops, accompanied by emotional excitement, crying or laughing without any cause.

The intellect, finally, during the post-apoplectic period is marked often by a condition of partial enfeeblement with more or less lack of self-control. Defects of memory, if present, pertain to events occurring about the time of, or subsequent to, the attack itself. The memory may remain confused. Sometimes it is questionable whether a victim of a stroke may ever resume his occupation.57 On the other hand, it is often amazing to notice how accurately a business man can judge of his affairs, and how clearly he can indicate his desires in regard to important matters, while he is paralyzed severely so that he cannot talk, hear, or write.58

Wills, made after a stroke, have experienced different fates according to the set of facts shown in the contest. A testator, 90 years of age, with delusions of being in danger of being kidnapped by his own daughters,59 or another testator with change in temperament and character, due to spinal paralysis, and with the insane delusion that his own daughter is the child of another man, though paternity has never been questioned by him until forty years after the birth of such a child,60 are of unsound mind. And, where a day or two before the preparation of the will and on the very day of the signing of the instrument the testator was lying in an apoplectic stupor and knew but little of what was going on, the will could not be upheld, particularly when the testimony of medical experts for proponents tended to prove mental incapacity.61

On the other hand, having several conferences with the attorney employed to draft the will, the testator taking a sketch of the will home and making changes, shows that the intellect could have been but little impaired, though there was evidence of senile dementia. The testator had been able, after the execution of the will, to transact a large amount of business three years later.62 Finally, apoplexy had little influence upon the testator who had suffered a paralytic stroke four years prior to the making of the will, which will was made under undue influence. Only 24 hours preceded death, which was caused rather by a combination of uremic poisoning, heart trouble, diabetes, kidney trouble, and other ailments.63

57Ibidem.
59Anlicker v. Brethorst, 329 Ill. 11, 160 N. E. 197 (1928), reversed in favor of contestant.
60In re Russell’s Estate, 189 Cal. 759, 210 Pac. 249 (1922).
63In re Solomon’s will, 95 N. J. Eq. 706, 124 Atl. 109 (1924), undue influence.
A higher degree of mental capacity is required in other legal transactions. A man, 86 years of age, with bad eyesight and bad hearing, when left physically enfeebled after a stroke, from which he had otherwise made a fair recovery, 64 a grantor with a syphilitic tumor of the brain, whose mind after a paralysis was somewhat addled, and who sold property for a grossly inadequate consideration, 65 are incapable of ordinary business transactions. Similarly, a former school teacher, with more than average education and business qualifications, when suffering from a stroke, was incompetent. At the time of the transaction he could not read, nor write, had spasms of convulsions, failed to recognize old acquaintances, and was otherwise a helpless imbecile. 66 See also Drake v. Mann, 67 Butters v. Butters, 68 and Prudential Life Ins. Co. of America v. La Chance. 69

Where capacity is under scrutiny as to transactions having taken place between two or more strokes, one would expect a greater impairment of mind. There is a long lapse of time after the first stroke with mental enfeeblement due thereto. The second apoplexy is on its way. Additional strokes add some mental degeneration. Yet, in Sexton's Estate 70 the will was upheld. Testatrix, though benumbed in her mental faculties, had been able to make a list of beneficiaries, and to give instructions to the lawyer drafting her will. In In re Dibble, 71 on the other hand, it was a question for the jury whether or not an illiterate woman, old, infirm, afflicted with eye and ear trouble, hard of hearing for all practical purposes, and incapable of conducting ordinary business affairs, could have made a valid will. But, in Hughes v. Freeley 72 testatrix did not possess testamentary capacity. She was found in a state of coma at the time of the execution of the will.

The conclusions one arrives at are that the post-apoplectic period by itself, whether consisting of only one attack of apoplexy or of several, is not a sufficient indication that no testamentary or business capacity existed in fact.

APOPLEXY AND ACCIDENT — THEORIES IN GENERAL

In "accidental" apoplexy plaintiff's theory of compensability is based upon some industrial fortuity, some negligence, or tortious act by defendant, which proximately caused the paralytic stroke. Where a victim of prior apparently good

67 1 N. J. E. 201, 86 Atl. 261 (1913), gift case.
68 177 N. W. 471 (Iowa, 1920).
69 113 Me. 550, 95 Atl. 223 (1915).
70 759, 751 Pac. 778 (1926). And see Rowcliffe v. Belson, 261 Ill. 566, 104 N. E. 268 (1914).
72 294 Pa. 391, 144 Atl. 277 (1928).
health is attacked by some violence or force so that apoplexy ensues, the chronological order of the events may lead to the inference of causal connection between violence-event and brain disorder.

Defendant concedes that plaintiff's theory is substantially correct. But the defense claims that additional factors must be proved. Due consideration must be given to a number of criteria which ought to be applied in order to show causal connection. Apoplexy attacks may be due to natural causes so that there may be mere time coincidence.

A. VIOLENCE VERSUS NATURAL CAUSE

There is always to be considered the issue of accidental violence versus culmination, independent of the apparent fortuity, by the ailment. Where the stroke has been ushered in by some premonitions just prior to the assault of the alleged accident, the inference arises that there was independent culmination rather than accident. In Singlaub v. Industrial Accident Commission the reviewing court pointed out that deceased had made two mistakes in work, with which he was very familiar, prior to the carrying of a key, an alleged overexertion. Such facts very aptly proved that decedent's mind had been "muddled" and not quite normal just previously to the claimed fortuity.

Similar observations have led to the same conclusion in other decisions. Having no recollection of the events from the time one mounts a ladder until one has fallen therefrom, driving a wagon and falling inertly from its seat with no attempt to save oneself from injury, or to break the force of the fall, or to make an outcry of distress prior to or during, the act of falling, being found unconscious in an elevator with no circumstances to indicate that anything is amiss, such circumstances are compatible more with a climax, independent of intervention by some accidental agency, of a pre-existing infirmity than with any other theory.

On the other hand, it must be remembered that premonitions may never be followed by an apoplectic stroke, that successful medical treatments or a natural

74 87 Cal. App. 324, 262 Pac. 411 (1927)—W. C.
remission, may have kept the victim out of the danger-zone of independent culmination. In Carvey v. W. D. Young & Co., the claimant had had occasional spells of dizziness, but had been "all right" on the day of the accident. In Blair v. Village of Coleraine, the petitioner had been treated for his high-blood pressure "occasionally during a number of months." "The blood pressure had been greatly reduced" so that about two weeks prior to the accident the tension was "about normal for a man of relator's age". Then, again, the medical treatment must have been sufficiently strong to provoke some response, or the remission must have been sufficiently lasting to indicate absence of independent climax of the ailment.

It is not always an easy matter to discern between such prodromata of natural climax periods and apoplexy symptoms during the existence of the violence-act itself. Rational behavior of the victim at the beginning and during a part of the act itself raises the presumption that the victim would have remained rational but for the intervention of the accidental agency. A man stricken with apoplexy would not be "likely to answer so promptly" during the performance of his work. Nor would such a person attempt to lift iron beams projecting over a passageway to be used by him, nor try to save himself from the force of a fall by making efforts to grasp the end of a board while riding a bicycle. Efforts to avoid being crushed by a large ash barrel which had been rolled up grade out of a cellar, or a willingness to enter into a friendly wrestling match, point, therefore, to absence of natural climax premonitions, and to accidental apoplexy. In Townsend Grace Co., Inc., v. Ackerman claimant testified that he noticed dizziness during the fall itself, but not before.

Absence of any vulnerating force is also to be considered as clear and convincing proof of independent culmination. Falling on a dirt floor, with no obstructions thereon, no pillars nor posts nearby, nor anything to indicate the cause of collapse, with no signs of external violence on the body except those.

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78218 Mich. 342, 188 N. W. 392 (1922)—W. C.
79177 Minn. 376, 223 N. W. 284 (1929)—W. C.
80The general rule is that the greater the interval of time elapsed between last spell of natural prodromata and the alleged accidental apoplexy, the less likely it is that there is apoplexy, independent of violence, provided a sufficiently strong vulnerating force can be shown: Gausman v. R. T. Pearson, 284 Pa. 348, 131 Atl. 247 (1925)—W. C.
81So-called "pseudo-prodromata", deriving their existence from some accidental source, such as chemicals, which, when inhaled, cause dizziness, fall, and apoplexy, or apoplexy in some other manner; Powers Storage Co. v. Ind. Com., 340 Ill. 498, 173 N. E. 70 (1930).
83Manning v. Pomerene, 101 Neb. 127, 162 N. W. 492 (1917)—W. C.
85Benjamin v. Kurnick, 5 N. J. Misc. 1095, 139 Atl. 440 (1927)—W. C.
caused by the fall itself, and with no evidence of outcries shows that the apoplexy occurred from natural causes only.\footnote{Hansen v. Turner Const. Co., 224 N. Y. 331, 120 N. E. 693 (1918)—W. C.; Crews v. Moseley Bros., 148 Va. 125, 138 S. E. 494 (1927)—W. C.}

Insufficiency of the vulnerating force belongs to the same general category. In\footnote{284 Pa. 348, 131 Atl. 247 (1925)—W. C.} Gausman v. Pearson\footnote{270 Pa. 476, 113 Atl. 666 (1921)—W. C.} the victim had worked indoors alone at light work, on a typical July day—warm, without excessive heat. He was not subjected to any unusual temperature or to overexertion. In Fink v. Sheldon Axel & Spring Co.\footnote{280 Pa. 203, 124 Atl. 471 (1924)—W. C.} the claimant came into contact with a pair of swinging doors which knocked him down. But absence of marks of external violence and the fact that his glasses, unprotected by rims, were still intact after the occurrence, were deemed to exclude accidental apoplexy.

But sufficiency of vulnerating force exists in the "assistance" cases. There is obvious evidence, from the very necessity of help, that a single man may overexert himself. In Samoskie v. Philadelphia & Reading Coal & Iron Co.\footnote{269 Pa. 586, 113 Atl. 75 (1921)—W. C.} the court on review remarked, referring to two witnesses, that they must have exercised only slight energy in starting to push a loaded mine car, and that, therefore, "deceased found it necessary to use greater force and accordingly exerted himself to a greater degree than his share of work called for." Other "assistance" cases are found in lifting a barrel,\footnote{203 Md. 489, 216 N. W. 241 (1927)—W. C.} and in the combination of lifting and of a blow on the head.\footnote{203 Md. 489, 216 N. W. 241 (1927)—W. C.}

Other criteria of overexertion are doing hard work in a cramped position,\footnote{Hull's Case, 125 Me. 135, 131 Atl. 391 (1925)—W. C. See also State v. District Court of Stearns County, 137 Minn. 318, 165 N. W. 667 (1917)—W. C., overexertion and fall.} and operating in intense heat with men spelling one another in using a bar to move a car.\footnote{Murray v. H. P. Cummings Const. Co., 197 App. Div. 903, 188 N. Y. S. 195 (1921)—W. C.}


\footnote{Yodis v. Philadelphia & Reading Coal & Iron Co., 269 Pa. 586, 113 Atl. 75 (1921)—W. C.}
Finally, there are cases of particular importance in such jurisdictions as Pennsylvania. Violence cannot be excluded entirely. If a like or similar force has been applied to the circulatory system more than once and previously to the apoplectic stroke, but without any symptoms thereof, it may be assumed that the paralysis is occupational rather than accidental. There is mere physical depreciation of the workman's body, an investment by the worker in industry, for the deterioration of which no compensation is granted. The criterion is usualness of work so engaged in and see the cause of apoplexy.

This principle has been applied to miners breaking rocks, to a driller, to a ditch digger, to a steam shovel operator who is exposed to the vibrations of the machine with a gradual breaking down of the blood vessels, and to other occupations. It should also be remembered that the mere working in pairs does not constitute necessarily "assistance", as this term has been interpreted above.

B. AGGRAVATION VERSUS ORIGINATION—SUPERINDUCEMENT

Of great importance in regard to apportionment of compensation damages and to the law of health, life and accident insurance is also the issue of aggravation versus origination—superinducement.

In the great majority of cases apoplexies are in part due to some pre-existing bodily infirmity of a progressive character, the morbidity advance being manifest or under cover, pseudo-latent. The blood vessels are primarily affected by the preaccidental ailment. The vulnerating force acts thru the medium of blood pressure or, there may be congenital weakness. In other instances there are such ailments as heart disease, syphilis, kidney trouble, and so on.

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100Crews v. Moseley Bros., 148 Va. 125, 138 S. E. 494 (1927)—W. C.


103Weissman v. Philadelphia Electric Co., 111 Pa. Superior Ct. 353, 170 Atl. 318 (1934)—W. C., acts preparatory in "assistance" cases, however, cannot be excluded, when the victim acts singly and overexerts himself.
It is also possible that the circulatory tree has been weakened by some violence prior to the accident under adjudication. There is some scar tissue in the vessel wall. There may be, in addition, high blood pressure, or some other disease.

However, not all cases show some basis of "aggravativeness". The vulnerating force may be the cause of an original apoplexy in a previously healthy person as to potentiality of apoplexy, as in "crushed brain tissue" apoplexy.

What are the differences between true aggravation-apoplexies and those which occur in "nonapoplective" healthy persons, suffering from accidental injury? Taking the most complicated set of facts with some external wound in addition to some "apoplectic", preexisting infirmity, the chain of events in aggravation-apoplexy may be divided into the following elements:

1. a vulnerating force; 2. a primary injury due thereto—the original injury—the effects of the vulnerating force; 3. after effects, reasonably immediate in time; 4. after effects, remote in time; 5. an "apoplectic", preaccidental infirmity. But, since the vulnerating force of the original injury in aggravation must by necessity have also influenced the preexisting apoplectic disorder, there must be in addition: 6. strength of penetration by the violence into the apoplexy-producing territory of the apoplectic, preaccidental infirmity, and, when arrived thereat, not only an apoplexy-vulnerative capacity, but also vulnerating apoplexy-effect upon the infirmity, without interruption of the chain of events by some outside, intervening force, followed by 7. apoplexy-producing effects of the vulnerating force upon the preexisting infirmity; 8. after effects, proximate in time; 9. after effects, remote in time.

So much is certain that in every aggravation case the vulnerating force, i.e. its effects or after effects, proximate or remote in time must go thru the preexisting, potential apoplexy ailment, turning it into a postaccidental apoplexy, total or partial.

1041. Not acting thru the medium of circulation. "Crushed brain tissue" apoplexy. II. Acting thru the medium of circulation. The blood vessel, prior to the infliction of injury, is A. not traumatically weakened, a. but diseased, arteriosclerosis. There is high blood pressure; 2. other diseases, heart ailment, syphilis, kidney trouble, and so on. High blood pressure may be present or absent. 3. Congenital weakness of wall of vessel. b. not diseased. No abnormality of any kind exists. B. traumatically weakened by some other fortuity, prior to the calamity under adjudication. There is scar tissue in the wall of the blood vessel and there is a. pathology, 1. hardening of the arteries, preaccidental, also high blood pressure, preexisting; 2. some other disease. High blood pressure, preaccidental, may be present or absent; b. no pathology. There is no high blood pressure.

105See "Compensable Aggravation and Acceleration of Preexisting Infirmities under Workmen's Compensation Act", n. 98. No vulnerating capacity existed in Barton v. Pittsburgh Coal Co., 113 Pa. Superior Ct. 454, 173 Atl. 678 (1934)—W. C., preexisting syphilitic morbidity of arteries, striking of head, causing cap and lamp to fall off, no tangible lump or bruise, nor concussion of head, lamp not broken. See also Dowling v. Public Service Production Company, 6 N. J. Misc. 391.
Since then any or all of the links of the chain of events, member 2 to 4\textsuperscript{106},
may thus pierce the boundary between original injury and apoplectic preexisting
infirmitiy, the apoplexy may occur very much later than one may expect at first
sight.

In superinduced original apoplexy, on the other hand, the chain of events
always and inevitably stops at link 4 of the chain. The territory of the preacci-
dental, apoplectic infirmitiy is never invaded by the vulnerating force, directly or
indirectly. Superinduced apoplexy is, therefore, solely the product of the primary
injury, its effects and after effects. But, here also, the paralysis may become mani-
fest a comparatively long time after the happening of the accidental injury.

It should also be understood that there may be superinducement apoplexy,
though there is some preexisting infirmitiy, nonapoplective or even apoplective.
The differentiation between aggravation and origination may therefore offer great
difficulties in some cases.

Maxon \textit{v.} Swift & Company\textsuperscript{107} is probably one of the best instances of aggra-
vation apoplexy. Deceased, suffering from hardening of the arteries, and from
high blood pressure, fell a distance of about 8 feet. After the accident he had
a lump on his head in its left lower region. He died fifteen months later from an
apoplectic stroke caused by his high blood pressure. An autopsy revealed a recent
hemorrhage in the brain substance. The inner surface of the skull showed a frac-
ture, due to the fall. The former lump, the fracture, and the recent hemorrhage
corresponded to one another. The fall, in all probability, had caused a small
tear in the blood vessel at the time of the accident. This lesion had healed. But

\textsuperscript{106}Complications causing aggravation. But, where deceased was suffering from a bone
disease which thickened the bony substance of the skull, though the disease also softened it, a
fall and resulting head injuries, followed by hemorrhage of the brain, were held not to have been
aggravation apoplexy, but rather superinducement paralysis: Shugart \textit{v.} Metropolitan Life Ins.

\textsuperscript{107}158 Minn. 491, 198 N. W. 133 (1924)—W. C. In Frey \textit{v.} Kerens-Donnewald Coal Co.,
271 Ill. 121, 110 N. E. 824 (1915)—W. C., medical testimony explained that, if plaintiff after a
hemorrhage of the brain and operation due thereto, should be working thereafter every day,
this would increase the chances of having another hemorrhage with apoplexy. An interesting
problem arises in regard to workmen’s compensation in such a case in which there is a prior
injury leaving a weakness in the circulatory tree, and where there follows a stroke, or another
apoplexy, while the victim is working for some other employer after the previous accident. It may
be suggested that the latter master is liable in toto and for all damages ensuing thereafter, provided
there is clearly some violence of accidental character rather than merely occupational in nature. On
the other hand, where apoplexy appears from intrinsic cause, and while the employee is not
engaged in industrial work, the first employer should be held liable since there is no break in the
chain of events of the first and only accident. As to industrial depreciation, however, doubt will
exist, particularly in jurisdictions requiring “accident” for compensation. Who is to be held liable?
Probably in the “accident” jurisdictions the first employer would be still liable, unless the
court finds that there is intervention of a new and independent cause. In the “arising out of the
employment” jurisdictions, however, it is possible that the second employer will be held liable.
In such a case an apportionment between the two employers would be most equitable. And see
Texas Employers’ Ins. Ass’n. \textit{v.} McGrady, 296 S. W. 920 (Tex., 1937)—W. C.
it had left a weak spot in the circulatory tree. This gave way to the hardening of the arteries, which had been in existence prior to the fall. The second hemorrhage occurred. The preexisting infirmity had been hastened to a fatal end by the primary injury. The chain of events was not broken. The coincidence of lump, fracture, and later hemorrhage was convincing enough to hold the employer liable.

As a counterpart, a superinducement case, *State v. District Court of Ramsey County* may be introduced. Here apoplexy developed after three fingers had been cut off. A series of apoplexies occurred. From the medical testimony one gathers that in consequence of lowered vitality "a circulation-blockade" or "circulation-stagnation" apoplexy was the result. There was no history of any preexisting infirmity. The morbid consequences were purely complications of the primary injury. Upon similar principles rests seemingly the apoplexy in *Leffler v. Morton Salt Co.*, from Michigan, an infection-inducement case.

Finally, there may be facts which make a distinction between superinducement and aggravation difficult. Where there is an infected wound together with preexisting kidney trouble, high blood pressure, heart ailment, and asthma, the stroke may be superinduced or, what appears more likely, an aggravation apoplexy due to increased blood pressure, the whole process leading through one or more preaccidental infirmities. The mere fact that the patient was in bed, and that this might have improved his preexisting conditions does not contradict either theory.

**C. CRITERIA OF AGGRAVATION**

Are there any other practical criteria which will otherwise aid in determining whether or not there is an aggravation apoplexy?

Apparent good health prior to the fortuity, medical treatment of sufficient effectiveness to reduce the hazards of a natural climax, natural remissions and recesses of the ailment have some probative value in the assumption that the stroke did not come on by itself.

The positive inference of accident arises where the disease terminates at a date earlier than was to be expected, if no accident had happened. A strong suspicion is cast upon the fortuity, the nearer in time thereto the termination of the ailment occurs. An immediate fatal issue, or one nearly so, is particularly
persuasive of causal connection with the violence.\textsuperscript{115} This same principle applies in modifications, where suddenness and rapidity of the previously slowly advancing ailment may be found to exist.\textsuperscript{116} It is this sudden rapidity toward incapacity which is apt to give the plaintiff the necessary preponderance of proof, when the advance of the disease has been tardy for a long period prior to the accident.

But in this class of cases, with a nonmanifest advance of the ailment toward disability, proof of aggravation cannot rest solely upon time coincidence. If the period of nonmanifestation of the apoplectic predisposition, or of apoplexy, could have ended in paralysis at any time, before or after the accident had occurred, claimant has not made out his case\textsuperscript{117}. But, a period of nonmanifestation which would have lasted longer than the violence and its effects and after effects lasted, justifies an award in favor of plaintiff. In the former situation there is an equal probability of injury without damage; in the latter case the fortuity may well be considered a proximate, contributing, and accelerating cause of the apoplectic disability.\textsuperscript{118}

Sameness of seat of disorder, injury, and apoplexy, as in head injuries, also leads to aggravation inference. The close neighborhood suggests entrance into the territory of the preexisting infirmity, provided the vulnerating force was not too weak.\textsuperscript{119}

Somewhat ambiguous is the evidentiary fact of lack of return to the former state of health, or the considerable retardation thereof. This may lead to the conclusion of aggravation. When a previously healthy person would have returned to good health, but the victim does not come back to his former state of health, the accident must have influenced also some preaccidental disorder leading to apoplexy,\textsuperscript{120} unless the situation is explainable by complications which lie entirely within the domain of the primary injury, thus constituting superinducement after effects thereof.\textsuperscript{121}


\textsuperscript{116}Maxon v. Swift & Company, 158 Minn. 491, 198 N. W. 133 (1924)—W. C.

\textsuperscript{117}Usually the vulnerating force is absent or too weak to cause substantial damage:—Kelly v. Nichols, 199 App. Div. 870, 191 N. Y. S. 445 (1921)—W. C.; and see occupational vulnerating force in: Rocco v. Ellworth Collieries Co., 111 Pa. Superior Ct. 508, 170 Atl. 316 (1934)—W. C.

\textsuperscript{118}See n. 116. Medical testimony in regard to aggravation of syphilis, and also judicial decisions, may differ. There are two schools, one holding that syphilis may become aggravated, the other one denying such an acceleration and claiming that syphilis is a constitutional disease which runs its course independently of injury, except in lesions of the head. See “Accidental Injuries”, Henry Kessler, pp. 480 et seq., Lea & Febiger, Phila., also Hamilton v. Congoleum Nairn, Inc., 6 N. J. Misc. 399, 145 Atl. 540 (1928)—W. C.; St. Louis Nat. Stockyards v. Industrial Com., 329 Ill. 221, 160 N. E. 114 (1928).

\textsuperscript{119}See n. 116.

\textsuperscript{120}Ibidem.

\textsuperscript{121}State v. District Court of Ramsey County, 147 Minn. 10, 179 N. W. 217 (1920)—W. C.
D. THE "TIME ELEMENT."

Regardless of the problems of aggravation, superinducement, or origination of apoplexy due to accident, uniformity in the decisions is missing by reason of many factors. There is dissimilarity of the vulnerating forces. Within the same category of accidents there is difference in strength of vulnerative penetration quantitatively. Certain criteria, however, apply to all cases of violence paralysis.

The "time element", for instance, is an essential test of compensability. Within what period of time did the paralytic stroke manifest itself after the vulnerating force has reached its peak of injury power? When may the victim of an accidental injury be deemed to have recovered so far that no causal connection exists between his accident and his apoplexy appearing thereafter?

No specific medical standards appear to be applicable. Medical testimony, in fact, is rather contradictory. In McAdoo v. Cudahy Packing Co.122 the medical report, adopted by the commission, says that "if the injury is sufficient to produce a serious result, the symptoms are continuous in almost all cases, and manifestations of serious injury are present from the start." In Selaya v. Ruthven & Cerrano123 the "substantial interval between injury and rupture of the blood vessel" made causal connection doubtful in the eyes of the medical men. But, in a Michigan decision124 a doctor states that "a man may fall on the street and suffer a fracture of the skull with hemorrhage, and he may subsequently go to his home and become unconscious from the gradual accumulation of blood in the brain". In Samoskie v. Philadelphia & Reading Coal & Iron Co.125, two physicians held the opinion entertained in the McAdoo case, while other doctors unhesitatingly testified that an interval of from 4 to 15 minutes, or of even a longer time, before outward manifestation of the apoplexy appeared, was "not unusual", but merely signified the original break to be of slight character.

A fair average of medical opinions, then, would seem to point to three outstanding landmarks, which pertain to all cases:

a. a Postulate—that there have been no prodromata just prior to the calamity;

b. a Concession—that the manifestation of signs of apoplexy depends upon the extent of the injury, and upon its location within a more or less vital spot of the body control apparatus within the brain;

c. a Problem—that possibly the victim may have recuperated reasonably definitely from the violence, as far as the paralytic stroke is concerned, whereafter it occurs from some other cause.

125280 Pa. 205, 124 Atl. 471 (1924)—W. C.
An unduly long interval of nonmanifestation raises prima facie suspicion of recuperation. But, a hidden initiatory period of from 4 to 15 minutes, of 3 hours, half a day, a day, and so on may nevertheless justify judgment for plaintiff.

All turns about permanency of recovery. A pain in the stomach appearing immediately after an overexertion, followed three weeks later by a paralysis is compatible with fortuitous violence. There was an interval of time between accident and apoplexy, symptoms thereof appearing in continuity. On the following two days there was weakness and vomiting of blood. Hiccoughs and great weakness came next, lasting for about two weeks. Finally the apoplexy appeared. In Texas Employers' Ins. Ass'n v. McGrady the victim remained in bed for a few weeks, whereafter he was given light work on account of his inability to perform hard manual labor. Immediately after the accident a slight impediment of speech, and other symptoms of apoplexy were noticed. Further attacks appeared later. There is also an insurance case in which a "spaetapoplexie" or "delayed apoplexy" resulted from concussion of the brain. Plaintiff immediately complained of dizziness, headache, blurred vision, and of other eye trouble. There were also mental symptoms. The quality of his work fell off from the date of the injury and grew progressively worse. He failed to note defects in the work under his supervision, and others made reports thereon. The accident occurred May 19, and the complete apoplexy became clinically manifest as such between June 20 to 23.

Furthermore, no recuperation exists, where the physiologically and anatomically normal tissue, though diseased, has been replaced by some "substitute repair" structure of lessened resistance as to the impact of high blood pressure. Here, the latter acts as an intrinsic force making the individual more susceptible to apoplexy by reason of the scar than in the case the trauma had not occurred. An employer was held liable, though the apoplexy happened 18 months after the fortuity had occurred. The scar in the circulatory tree, being due to industry and leaving the man unprotected against the onslaught of the increased blood pressure from natural causes, contributed to a premature death by another stroke. During

126Ibidem.
127See n. 123.
129Kingston-Pocahontas Coal Co. v. Maynard, 209 Ky. 431, 273 S. W. 34 (1925)—W. C.
13196 S. W. 920 (Tex. 1927)—W. C.
133Apoplexy with loss of consciousness occurring some time (but not immediately) after trauma. Cases have been described in which the interval between traumatism and stroke lasted from six days to four weeks. Autopsy showed in the majority of cases a cerebral hemorrhage. Tice, vol 9, p. 580, see n. 2.
these 18 months the victim uninterruptedly suffered from after effect symptoms of the first hemorrhage. 134

In other cases there is, perhaps, an accident necessitating amputation. Infection results. A "corrosion circulation" or "blockade" or "stagnation" apoplexy follows. The only significant mark is that this happens while the patient was still more or less under the influence of germ-toxicity. 135 Or, a "blockade" may follow while the victim is recuperating from a fractured, cut, or crushed bone injury. 136 Syphilis may, when aggravated, 137 lead to apoplectic incapacity a considerable time later. 138 There may also be an aggravation of a preexisting heart disease. 139 Finally, lack of elimination and auto-intoxication may bring about an apoplectic stroke during the course of hospitalization or medico-surgical treatment. See Frey v. Kerens-Donnewald Coal Co., 140 and Orchard Wilhelm Co. v. Petersen. 141

On the other side, recuperation may be assumed to exist, where, as in McAdoo v. Cudaby Packing Co., 142 there was unconsciousness for about half an hour but thereafter no incapacity nor symptoms of stroke appeared until three weeks later. The chain of events was held broken in Chilton v. Louisiana Cent. Lumber Co., 143 where the attending physician and the employee himself considered the injury as not serious, and where location of the paralytic area within the brain did not support the claim. The stroke occurred more than a year later in a man suffering from high blood pressure. In Chicago, R. I. & M. Ry. Co. v. Harton, 144 a fracture of the skull case, the employee, for more than a year after the accident and while apparently in good health, had been working as a farm hand. The injury itself was not dangerous.

As a general rule, causal connection exists between violence and brain injury, when seen from the point of view of the "time element"

1. where there is a period of apparent preaccidental good health, as this term is understood by the judiciary,
2. followed (after an attack of sufficiently strong vulnerative character of apoplexy capacity) by a period of latency—or of apparent good health in re-

134 Maxon v. Swift & Company.—W. C., see n. 116.
136 State v. District Court of Ramsey County, 147 Minn. 10, 179 N. W. 217 (1920)—W. C.
In fractures of the bone circulation blockade may occur from the 4th to the 72nd day after the injury, on the average between the 13th and 20th day, "Practical Surgery", Senn, p. 397, 1902, W. B. Saunders Company, Phila. & London.
137 See n. 118.
139 Cook County v. Industrial Commission, 329 Ill. 79, 158 N. E. 405 (1927).
1390 271 Ill. 121, 110 N. E. 824 (1915)—W. C.
141 127 Neb. 476, 256 N. W. 37 (1924)—W. C.
142 See n. 122.
143 12 La. App. 299, 125 So. 457 (1929)—W. C.
gard to apoplexy—between calamity and forerunners of the paralytic stroke reasonably sufficient to dovetail with, and to account for, the phase of hidden initiatory development,

3. succeeded immediately thereafter by manifestations of brain trouble,

4. and, perhaps, again followed by a secondary "sham-intermission-recovery period", containing some sign of apoplexy, however, and

5. leading to the full clinical picture of a total or partial stroke without inter-
vention of some event indicative of the fact that the recuperative powers of the body in the meantime have reasonably definitely and permanently overcome the morbid influence of the fortuity, its effects, and after effects, proximate or remote in time, upon the apoplexy resisting powers of the body.

The rule applies to the more difficult and irregular cases. Where there is immediate disability or death, this criterion is to be condensed. Other variations are also apt to shorten this yardstick.

E. THE "LOCATION CRITERION."

The matching of the seat of external injury with that of the brain injury is sometimes also a most persuasive criterion of causal connection. The close neighborhood affords a means of tracing the vulnerating wave. A gunshot wound, penetrating thru the osseous skull into the brain substance, is very convincing proof of "crushed brain substance" apoplexy. The starting point of the violence, its track, and the terminal point are generally clearly demonstrable.

A blow against the head, when followed by brain injury on the same side, is also easily conceived to be causally connected with each other. Maxon v. Swift & Company, a Minnesota case, is a good example.

The plausibility of causality decreases with the increase of distance from the brain focus. Striking the small of the back against a projection, for instance, makes paralysis unlikely, in absence of an additional head injury or some other explainable morbidity to the contrary. A California Accident Commission decision represents such a situation.

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145See n. 1. Such a sign may not be detectable until after an autopsy has been performed. The interval may be comparatively long. Take Maxon v. Swift & Company, n. 116, and leave all subjective and objective signs out, except the lump, fracture and the second rupture as after effect and in the opinion of the writer, compensability would still exist.


149See n. 116.

It is questionable whether in head injuries finer distinctions beyond that of localization on and within the head itself are useful. In a Louisiana "fall" case the external wound was on the left side of the head, while the brain area involved was on the other side. The reviewing court made use of the discrepancy in order to deny compensation.

Who is to say with any degree of accuracy what a fall of about 12 feet may do to the head of the victim, even though the starting point of the fall-impact is in a certain location? The skull itself is somewhat elastic. There are fissures, filled in with cartilage, a substance still more springy than bone. The brain, by its membranes, is suspended and floating in its liquid. A coup may be followed by a counter coup. It is conceivable that the part of the brain closest the impact starting point of the violence escapes injury, while some other part of the cerebral substance, farther away and of less resistance, does not do so. The head is not a solid body so that the violence wave would travel only in one direction. The semi-elasticity and the semi-liquid consistency rather calls for other, corresponding physical laws.

Neither does the fact that the brain is seemingly so well protected against external injuries contradict such a conclusion. Outside and inside lesions need not tally exactly nor even comparatively. In the Louisiana case it would have been better to point out more distinctly that the "location criterion" has corroborative value only, because the "time element" negatived causal connection. As a criterion the location is usually only ancillary to the "time element". But, when seen from this point of view, location has some evidentiary importance.

Otherwise are conditions in "strain and lifting" cases, for instance. Definite, visible points of attack by the vulnerating force cannot be ascertained with any degree of accuracy, though they exist in fact. The same holds true of the vulner-

151Chilton v. Louisiana Cent. Lumber Co., 12 La. App. 299, 125 So. 457 (1929)—W. C. It must be remembered that by external injury is NOT MEANT the paralysis of the arm, or leg, or of the whole side of the body. These constitute only after: effects of the brain injury. Their value consists only in determining the approximate seat of lesion within the brain substance.

152"It appears from the medical testimony that a blow on the head may cause a fracture of the skull with no internal hemorrhage, or it may cause a hemorrhage without a fracture" etc., see n. 116.

ation track. Only the terminal point, the brain lesion itself, is fairly well determin-
able. The vulnerating forces act thru the rather ubiquitous circulatory system, with
its uncertainty and perplexing indefiniteness as to the route taken by the violence-
wave. External marks of violence are evidentiary facts of the post-apoplectic
period in most cases. Where, therefore, the vulnerating force attacks the system
of the body rather than a specific part thereof, the location criterion loses its
value. A man suffers an apoplexy by reason of mental excitement or strain
while in the act of falling. He bumps his head on the ground thereafter. The excite-
ment raises the blood pressure, and a hemorrhage occurs. Or, a man becomes
paralyzed because of the inhalation of some noxious gas. Thereafter he col-
lapses. His fall injuries, again, are only of secondary order in the evaluation of
causal connection.

Finally, there are cases in which there may be evidence of localized and
generalized attacks by the violence. Thus, the fall may not have induced suffic-
ient excitement to cause apoplexy. The impact of the head upon the ground pro-
duced the latter. Or, the fall may at first have created a mental excitement with a
small apoplexy, whereafter the fall impact aggravates the apoplectic condition.

The difference between these divers categories of calamities and their causal
connection with apoplexy rest upon the diversity of the devitalizing influence of the
external force, whether acting locally upon the head, or generally upon the system,
or upon both. Hence the following conclusions are to be drawn:

1. In order to evaluate the "location criterion" it is necessary to establish not only
   a starting point of the vulnerating wave, but also its track, and its terminal
   point;
2. The terminal rather than any other part is the connecting link between apo-
   plexy and primary injury;
3. The terminal point may be remote from the perceptible track of the vulnerat-
   ing force by reason of intervention of media of the body, solid, semi-liquid,
or liquid thru which the violence wave is transmitted;
4. The "location criterion" is of relative value where there is localized devitali-
   zation, but it becomes worthless, when the cerebral trouble is based upon
generalized devitalization, such as increase or decrease of blood pressure,
or, perhaps, disintegration of the blood quality;
5. The "location criterion" may permit recovery of damages, when based upon
   localized, or generalized, or localized generalized devitalization theories,
as the case may be;

\[154\] Townsend Grace Co., Inc., v. Ackerman, 158 Md. 34, 148 Atl. 122 (1930)—W. C.
\[156\] Where the "localized" devitalization is doubtful, it is better to form, if possible, a
theory of generalized devitalization, since in the latter the injury quantitatively is more extensive.
The "location criterion" is ancillary to the "time element";
The "location criterion" applies to origination, superinducement, and aggravation apoplexies alike.

F. THE "TYPE" ISSUE.

The "type" issue of apoplexy may possibly enter litigation. Must in a given set of facts a certain type of brain lesion follow, without exception and by necessity so as to make such a type of exclusive and absolute characteristic of an accidental vulnerating force of a given, specific kind, or may there be deviations? And, if so, how far may such a deviation extend into the field of proof and away from the type of the classical accident?

Certain decisions, either in the syllabus or in the text, speak of apoplexy, stroke, stroke of paralysis or of apoplexy, cerebral apoplexy, or other words and descriptions of a rather vague and neutral color. Other texts express the morbidity in terms of injury causing paralysis, brain lesion, lesions of the blood vessels of the brain, vascular-cortical lesions, and blood clots formed in the brain. It appears that, in many instances, the "type" issue has been immaterial for some reason or the other.

On the other hand, operative and autoptic findings lay down the hypothesis of apoplexy genesis as a matter of necessity. The plaintiff cannot aver and prove anything by speaking of a hemorrhage, where there is indisputably a stagnation, for instance. Also, cross-examination and hypothetical questions must not be framed along lines alien to the issue.

United States Casualty Co. v. Matthews, 35 Ga. App. 526, 133 S. E. 875 (1926)—W. C.
Fink v. Sheldon Axle & Spring Co., 270 Pa. 476, 113 Atl. 666 (1921)—W. C.
Roach v. Oswald Lever Co., 274 Pa. 139, 117 Atl. 785 (1922)—W. C.
Manning v. Pomerene, 101 Neb. 127, 162 N. W. 492 (1917)—W. C.
Nofsinger v. Paup, 96 Neb. 805, 148 N. W. 967 (1914)—W. C.
Furthermore, where the violence is so strong that medical expert testimony becomes superfluous,\textsuperscript{168} it does not matter what type of apoplexy occurred. Any fine distinctions would only confuse the mind of the trier of facts.

One may, however, infer a like occurrence from the previous mode of destruction of the cerebral area. Thus, a previously torn vessel will in all probability lead to another tear. The scar tissue after the first tear is only substitute structure of inferior elasticity and of minor resistance giving quality.\textsuperscript{169}

Similarly, one may expect that one stagnation be followed by another one. The peculiarities of the blood vessel have probably not been eliminated after the first brain lesion happened.

Even a blockade apoplexy at a later time may be supposed to be a continuance of the prior one. The morbid conditions presumably have remained in existence during all that time.\textsuperscript{170}

Yet, in all cases a certain amount of speculation and conjecture is unavoidable. With deviations from the character of the vulnerating force deviations from the type of apoplexy may occur.\textsuperscript{171}

In certain injuries, however, the type is apparently fixed to a certain degree. For instance, only the blockade theory may satisfy the genesis of apoplexy and the hypothesis of proximate causation. There is no high blood pressure. There are no brittle arteries. No preexisting infirmities can be found which by reason of the original injury could have brought about an apoplexy. There is only some external wound, on some part of the body other than the head. It is indeed difficult to see how a vessel tear, for example, could have occurred. But, if one is to assume a blockade by reason of some particle of flesh having entered the circulation and having caused a cork-like closing up of the cerebral vessel, the logic is forcefully convincing.\textsuperscript{172} However, even here one meets with limitations.\textsuperscript{173}

There remains a discussion of some other cases. The plaintiff argues that the medical experts for the defense indulge in subtle analysis in order to arrive at theoretically possible, but scientifically more or less strained, interpretations of the results of violence inflicted. Their purpose is to becloud the clear issue of the chain of events as much as the obscure genesis of apoplexy will permit. There is

\begin{itemize}
  \item \textsuperscript{168}McCoy v. Spriggs, 102 Pa. Superior Ct. 500, 157 Atl. 523 (1931)—W. C., syll. 4.
  \item \textsuperscript{169}See n. 116.
  \item \textsuperscript{170}State v. District Court of Ramsey County, 147 Minn. 10, 179 N. W. 217 (1920)—W. C.
  \item \textsuperscript{171}A blockade may at first occur in the lungs whereafter some particle of the lung blood clot enters the brain circulation.
  \item \textsuperscript{172}See n. 170.
\end{itemize}
an injury from which a paralysis may reasonably follow. The "time element," the continuity of the events, leads to the conclusion of proximate causation.

But the defense disavows such intentions. Take the case of hardening of the arteries. How, it is asked, could overexertion, mental excitement, or some other vulnerating force calculated to raise the already dangerously high blood pressure, cause a stagnation apoplexy or a blockade? Is it not reasonable to assume that there was a sudden increase in blood pressure which would propel the column of blood faster thru the diseased blood vessel than it would have moved without such a violence? A considerable tear with a quickly appearing apoplexy rather than stagnation would be the result. If there was stagnation, the alleged vulnerating force must have been absent. There was no accident.

In medicine there are, in fact, symptoms which have been believed to be specific in regard to the different types. Thus, it is quite true that in stagnation the apoplexy is generally slow in appearance, while in a vessel tear there is suddenness of manifestation of morbidity. But, a blockade apoplexy may also occur suddenly. Where a tear is small, symptoms may appear just as slowly as in stagnation. Starr points out that as to the type one may arrive at a positive and accurate diagnosis in only about one-half of the cases. Struempell says that a diagnosis can scarcely ever be made with absolute certainty, since tearing, blockade, and stagnation may exhibit almost identical phenomena. Some speculation is, therefore, inherent in this problem and at any rate in the living victim.

As to the explanation of the defense in regard to tear or stagnation some words need be said. The mechanism of stagnation is a weakening of the heart action, a morbid stasis of the blood in the vessel, predisposed thereto. A general state of weakness, a syncope, the reaction which follows mental excitement, emotional shock, a fright, or a condition of malnutrition or starvation, as after effects of the original injury, may thus lead to stagnation.

In hemorrhage, on the other hand, there is indeed a strong heart action. But a strong heart, too, may for an instant become weak by reason of some physical or psychic influence. What it amounts to, then is that the violence may have two accident vulnerable peaks, due to the strong oscillations of the blood current tension. There is a positive "vessel-wall-tearing-high tension" peak and a negative "low-pressure-coagulation" summit.

In the first instance the tension is at once increased beyond the breaking point; in the other case the blood pressure goes down so far that coagulation occurs. Who can tell with any definiteness what actually happened within the circulatory tree of the body at the very moment of the accident and when the apoplexy comes into existence? One cannot deny that a violence may be accompanied by shock, mental or physical, sufficient to bring about stagnation rather than tear apoplexy. This is corroborated by the medical fact that the same kind of violence in many instances may induce either type of paralysis.
Facing such uncertainties one must return to purely legal standards. "The mysteries of death are inscrutable and baffle complete scientific solution by mortal men." . . . "The best that can be done is to establish a chain of causation—to show a cause (an injury) from which an effect (death) might reasonably follow." . . . "We know, without doctors' advice, that men have been killed by falls. We know, too, that death may follow weeks of pain and suffering from inward hurts, having their origin in such a fall." 174

It is, therefore, not surprising that in two decisions Fowler v. Risedorph Bottling Co., 175 a lifting, strain case, and in La Veck v. Parke, Davis and Company, 176 an overexertion artificial heat decision, judgment went for the plaintiff.

The reviewing court may have considered the "type" issue as too speculative and too conjectural to deserve any serious attention. It may also have adopted the commission's view that a tear was somewhat more likely than mere stagnation. The final conclusion may also have been that generally either stagnation or vessel tear could be accidental, and that, even assuming there was a stagnation, the plaintiff was entitled to compensation.

(To be continued)

175 App. Div. 224, 161 N. Y. S. 535 (1916)—W. C.
176 190 Mich. 604, 157 N. W. 72 (1916)—W. C.