

CASE REPORT

Joseph A. Prahlow,¹ M.D.

Homicidal Cerebral Artery Aneurysm Rupture*

ABSTRACT: When a normally natural mechanism of death is induced by physical injury or intense emotional stress, it is appropriate to rule the manner of death as something other than natural. When the case-specific circumstances are such that the death occurs as a result of the criminal activity of another person, it is acceptable to rule such deaths as homicides. Presented herein is a case of homicidal cerebral artery aneurysm rupture occurring in an intoxicated, 46-year-old man who was punched in the face by another individual. The details of the case are presented, followed by a discussion of the controversies that exist when dealing with such cases. Guidelines for investigating similar deaths are presented, with emphasis on the timing of the trauma in relation to onset of symptoms due to aneurysm rupture.

KEYWORDS: forensic science, cerebral aneurysm, berry aneurysm, homicide

Manner of death (MOD) determination is a well-known source of debate among forensic pathologists, particularly with regard to certain, specific case-types (1–4). Usually, MOD determination is based on logical, straight-forward reasoning. Occasionally, MOD determination is based on tradition or convention. In instances where debate exists about proper MOD determination, there are typically valid, cogent arguments brought forth by persons on each side of the debate. In many cases, such disagreement represents an academic exercise, fodder for entertaining debate among the forensic pathology and death investigator community. In other cases, however, MOD may have more far-reaching implications. This is particularly true when “homicide” is one of the options for MOD certification. The term “homicide” is considered a medical term and does not necessarily have any legal significance. Consequently, whether or not formal criminal charges are sought in a particular case should not depend entirely on the MOD. However, because MOD rulings are frequently used by prosecutors as part of the reason criminal charges are filed, such determinations should not be made lightly by forensic pathologists.

In 1977, Davis published his classic work, “Can sudden cardiac death be murder?” (5). In his paper, Davis presents a logical, well-reasoned argument for certifying certain cardiac deaths as homicides, so long as a set of specific criteria are met (Table 1) (5). Using Davis’ criteria, a mugging victim who suddenly collapses and dies from underlying severe coronary artery disease can be appropriately classified as a homicide victim. While there may be some forensic pathologists who do not agree with such a ruling, the phrase “homicide by heart attack” remains well-known within the forensic community.

¹ Forensic pathologist, South Bend Medical Foundation and associate professor of Pathology, Indiana University School of Medicine—South Bend Center for Medical Education at the University of Notre Dame, South Bend, IN.

* This paper was presented at the 2004 Annual Meeting of the American Academy of Forensic Sciences in Dallas, TX.

Received 16 Mar. 2004; and in revised form 7 May 2004; accepted 7 May 2004; published 4 Aug. 2004.

Cardiac disease is not the only natural disease process that can be considered the underlying mechanism of death in homicide. Subarachnoid hemorrhage related to traumatic rupture of a cerebral artery berry aneurysm or an arteriovenous malformation has received some attention in the medical and forensic literature (6–10). In this paper, we present a case of “minor” head trauma causing the rupture of a cerebral artery aneurysm. The MOD was considered “homicide.” The ensuing discussion will address this controversial topic and present a proposed set of criteria useful in making such a determination.

Case Report

An intoxicated, 46-year-old man, his wife, and their female friend returned to the friend’s home late one night, after having been out for dinner and drinks. The man was reportedly loud and boisterous. Upon hearing the commotion, the friend’s adult daughter, who had been sleeping, got up, came out of the bedroom, and asked them to be quiet, because her live-in boyfriend needed to get up early for work. She then stated that she was going back to bed. The 46-year-old man then proposed to the daughter that he might join her in bed. The daughter’s live-in boyfriend then emerged from the bedroom and confronted the intoxicated man. A short verbal altercation ensued, followed by a single punch, thrown by the boyfriend, that landed on the other man’s face. The man immediately collapsed to the floor, totally unresponsive. Emergency resuscitation was initiated by the owner of the home. Emergency medical services responded to a 911 call and transported the victim to the hospital, where work-up and imaging studies revealed diffuse basilar subarachnoid hemorrhage and a ruptured berry aneurysm in the basilar artery. He died approximately 36 h after the initial collapse.

Autopsy confirmed the presence of diffuse, basilar subarachnoid hemorrhage, as well as a ruptured, 9 mm basilar artery aneurysm. Brain examination was consistent with global ischemia. The remainder of the autopsy was significant for cardiomegaly (500 g), with concentric left ventricular hypertrophy, as well as mild to moderate atherosclerotic cardiovascular disease involving the aorta and coronary arteries. There were no facial injuries identified at

TABLE 1—Davis' criteria for homicide by heart attack (5).

1. The criminal act should be of such severity and have sufficient elements of intent to kill or maim, either in fact or statute, so as to lead logically to a charge of homicide in the event that physical injury had ensued.
2. The victim should have realized that the threat to personal safety was implicit. A logical corollary would be a feared threatening act against a loved one or friend.
3. The circumstances should be of such a nature as to be commonly accepted as highly emotional.
4. The collapse and death must occur during the emotional response period, even if the criminal act had already ceased.
5. The demonstration of an organic cardiac disease process of a type commonly associated with a predisposition to lethal cardiac arrhythmia is desirable.

autopsy. A blood ethanol level from the time of hospital admission was 153 mg/dL.

Police investigation confirmed the story as presented above. The victim's wife, the home-owner, her daughter, and the boyfriend all related similar scenarios as they recalled the events. The cause of death was ruled "subarachnoid hemorrhage due to ruptured basilar artery aneurysm following blunt head trauma." The manner of death was ruled "homicide."

Discussion

In 1977, Davis published a classic forensic manuscript entitled, "Can sudden cardiac death be murder?" (5). The case presented herein may cause one to consider a similar question: can death from a ruptured cerebral artery aneurysm be murder? More specifically, can head trauma, such as that delivered by a punch from a fist, cause fatal subarachnoid hemorrhage via rupture of a pre-existing cerebral artery aneurysm? A related question is as follows: can the emotional stress of a situation result in fatal rupture of an aneurysm? Similar questions related to cerebral arteriovenous malformations may also be considered. In the ensuing discussion, these topics will be addressed, with presentation of a set of criteria for establishing a diagnosis of traumatically-induced (or emotionally-induced) rupture of a cerebral artery aneurysm (or arteriovenous malformation).

When reviewing the literature with regard to traumatic rupture of cerebral artery aneurysms, there are occasional references that deal specifically with the topic (7,9,10), or similar topics (AVM rupture) (12). Other reviews deal with the less specific traumatic basilar subarachnoid hemorrhage, whether or not it is related to aneurysm rupture (6,8,11,13,14). In addition, several of these reviews were published prior to 1970, and the concept of vertebral artery injury is not thoroughly addressed (6,8,13,14). Consequently, care must be taken when evaluating the literature with regard to traumatic basilar subarachnoid hemorrhage. During the remainder of this section, every attempt will be made to indicate when cited references are referring to the less-specific traumatic subarachnoid hemorrhage or the more-specific traumatic aneurysm (or AVM) rupture.

Whether or not trauma can cause the rupture of a cerebral artery aneurysm remains a matter of debate within the medical and forensic literature. Many authors acknowledge that traumatic berry aneurysm rupture is possible (6,8,10,11), while some remain skeptical that such an occurrence is possible (9). Most believe that such an occurrence is a relatively rare event. Many reviews of spontaneous ruptured cerebral artery aneurysms have addressed the issue of trauma-induced rupture, but the link between trauma and rupture has not been established with certainty (15–19). In fact, a vast majority of all ruptured cerebral artery aneurysms occur without associated trauma, fear, or intense emotional stress (15–19). De-

spite this fact, the potential exists for berry aneurysm rupture to occur in the setting of trauma, fear, or intense emotional stress; given the correct setting, such deaths can be considered homicides (19). Having acknowledged this, it is vitally important to remember that a vast majority of ruptured cerebral artery aneurysms can be considered "spontaneous," occurring as natural events and having nothing to do with trauma.

McCormick's work (9) is frequently referred to by those who agree that traumatic aneurysm rupture is not possible. In this review, McCormick acknowledges that traumatic aneurysm rupture is theoretically possible, but then argues that, "... critical review of the literature and extensive personal experience indicates no or at best a very weak relationship between blunt closed-head trauma and the rupture of a preexisting saccular intracranial aneurysm" (9). This conclusion is based on McCormick's review of 362 patients with ruptured or unruptured saccular intracranial aneurysms detected at autopsy (9). The patient population included many hospital (non-forensic) cases (percent not specified); in addition, the review does not specify whether or not the data includes *all* forensic case cerebral aneurysms during the study period, or only those cases referred to McCormick's neuropathology service (9). As such, this study might grossly underrepresent forensic case material. Whatever the case, out of McCormick's 362 cases, 18 had evidence of closed head injury, 12 recent and 6 remote (9). Twelve patients had "severe" head injuries, but only three of these experienced aneurysm rupture (9). In the remaining six cases with closed head injuries, the injuries were not considered severe, and aneurysm rupture did not occur at the time of trauma (9). From these data, McCormick concludes that, "trauma, even very severe, will not always (or even usually) cause a preexisting saccular aneurysm to rupture" (9). McCormick also states that, "it seems very unlikely that closed head trauma causes rupture of preexisting saccular aneurysm in the majority of patients where the two coexist" (9). While such statements may be fodder for those who claim that traumatic rupture is not possible, it is evident from McCormick's own words that he acknowledges that traumatic rupture is possible. In fact, elsewhere in the review, McCormick states that, when attempting to make an association between trauma and aneurysm rupture, "the time relationships between the trauma and the aneurysm rupture should be such that the likelihood of an association is really present" (9). Thus, McCormick's work (9), like other large reviews of ruptured cerebral aneurysms (15–19), confirms that a vast majority of cases have no association with trauma; however, the study does not rule out the possibility of an association in select cases.

In 1958, Newbarr and Courville investigated the possibility of traumatic rupture of intracranial aneurysms, with review of the international literature (7). The reader is referred to the Newbarr and Courville paper for an extensive review of the older medical literature regarding this topic. Much of the older literature deals with attempting to implicate remote trauma in the formation of and/or eventual rupture of cerebral aneurysms, sometimes occurring years after the trauma (7). Newbarr and Courville provide a useful set of postulates for cases of suspected traumatic berry aneurysm rupture (7). Many of these have been incorporated into the criteria provided at the end of the present manuscript.

Experts differ in their opinion regarding the severity of trauma necessary to provoke aneurysm rupture. Some claim that traumatic subarachnoid hemorrhage of any type requires "severe" or "significant" head injury (6,9,10,12), such that there are associated brain contusions or skull fractures. Others purport that "minor" to "moderate" head injury can induce subarachnoid hemorrhage (7,8,11,13,14). In such cases, evidence of facial or head trauma in the form of abrasions, lacerations, contusions, and subscalpular

hemorrhage are the typical findings; however, as the present case demonstrates, some cases with an unquestionable history of trauma (such as a punch to the face) do not necessarily have to have identifiable injuries at autopsy. The converse is also true. Just because there is evidence of minor facial/head trauma at autopsy does not mean that trauma preceded or caused the aneurysm rupture. Facial and head injuries certainly may have preceded the aneurysm rupture, but not caused the rupture; thorough investigative information is required to make such a determination. In addition, facial and head injuries can occur as a result of the person's collapse following a spontaneous aneurysm rupture (the so-called "signs of innocence") (19).

The anatomic location of the aneurysm is felt, by some, to play a role in whether or not the aneurysm is susceptible to traumatic rupture, with aneurysms in the anterior and middle cerebral artery distributions being relatively immune to trauma-induced stress forces (10). If such is the case, then the basilar artery location in the current case represents an additional risk factor for traumatic rupture.

As alluded to earlier, it is worth noting that the emotional and physiologic responses to the situation likely act as contributing factors in aneurysm rupture. Emotionally stressful situations are known to induce sudden death, with most deaths occurring in susceptible individuals with underlying cardiac disease (20–23). In one such study of 43 cases of medicolegal deaths deemed to be sudden deaths triggered by stressful events, most cases involved underlying cardiac disease, but two cases were attributed to subarachnoid hemorrhage; unfortunately, more specific details (whether or not cerebral aneurysms were detected) were not provided (23). In cases of cerebral artery aneurysm rupture where emotional stress is felt to be contributory, the stress of the situation probably results in a transient elevation of blood pressure (7,10,12), likely induced by catecholamine release (10). Such stressful situations may involve excitement, fear, pain, violent exertion, or other stresses (12). Some authors believe that, in "minor" head injury cases, the emotional component is largely responsible for the subarachnoid hemorrhage (6,7). For legal reasons, it might be beneficial to be able to differentiate the emotional/physiologic component from the physical/traumatic component of the situation; however, such differentiation is not easily accomplished. As such, it is advisable to acknowledge that the emotional/physiologic response that typically accompanies the trauma likely plays a contributory role in aneurysm rupture, via transient blood pressure elevation and perhaps other mechanisms. It is also conceivable that, in certain cases, fear or intense emotional stress alone (without any physical contact) may induce similar physiologic responses with subsequent berry aneurysm rupture. Given the correct situation, such cases may also be considered homicides.

Several authors contend that ethanol intoxication appears to play a contributory role in certain cases of ruptured berry aneurysm (11,13). They further contend that this association is particularly evident in those cases related to trauma (11,13). Ethanol intoxication reportedly causes considerable dilatation of the intracranial blood vessels, thus theoretically making them more vulnerable to trauma (8). As such, ethanol intoxication may have placed the victim in the presented case at risk for traumatic aneurysm rupture. Another consideration is that ethanol intoxication might contribute to confrontational situations, thus increasing one's chance of experiencing trauma.

In previous reports dealing with traumatic cerebral aneurysm (or AVM) rupture, various recommendations have been proposed to aid in making such a diagnosis (6–12). Despite various issues of debate, including how much force is necessary to cause aneurysm rupture and how much of a role the emotional component contributes to the

rupture, most authors agree that traumatic rupture of an aneurysm is, in fact, possible. The most frequently cited, important and useful recommendation relates to the timing of the trauma and the subsequent symptoms related to intracranial hemorrhage (7,9,10,12). Barnard and Hirsch state this importance as follows: "to conclude that trauma ruptured . . . an aneurysm, there should be a close temporal relationship between the injury and the onset of symptoms. The shorter the interval, the more likely the relationship" (12). The case presented herein probably represents the most ideal situation for making a definitive diagnosis of trauma-induced aneurysm rupture. In this case, there were multiple witnesses (including the perpetrator) who all agreed that the collapse immediately followed the punch to the face.

Another important concept relating to the timing of aneurysm (or AVM) rupture involves investigating the possibility that the hemorrhage occurred *prior to* the trauma. Historical information plays a key role in such investigations. Microscopic examination of the hemorrhagic region may show evidence of chronic damage/leakage or repair in cases where hemorrhage preceded trauma.

In order to rule a ruptured aneurysm (or AVM) death as a homicide, it is advisable to follow the same logic that Davis presents in his "homicide by heart attack" article (5). Given that the timing of the trauma (or emotionally stressful event) can be conclusively implicated in the aneurysm rupture, it is appropriate to rule such a death as a homicide if the trauma (or threatened trauma or emotionally stressful event) occurred during or as a result of an event that would normally be considered a criminal activity. When considering ruling such a death as a homicide, it is helpful to review one definition of homicide, as it appears in the National Association of Medical Examiners *Guide for Manner of Death Classification*: "Homicide occurs when death results from a volitional act committed by another person to cause fear, harm, or death. Intent to cause death is a common element but is not required for classification as homicide" (1). With this definition in mind, it is clearly appropriate to rule the case presented as a homicide.

The following is a list of important points and recommendations for death investigators and pathologists who are faced with a ruptured cerebral artery aneurysm case where trauma (or fear, etc . . .) has been implicated as a possible contributing factor. The list is a synopsis of recommendations made by many authors in previous publications (5,7,9,10,12).

1. Since cerebral artery berry aneurysms most commonly rupture spontaneously, great care must be taken when evaluating a case of ruptured aneurysm suspected of being related to trauma.
2. Care must be taken so that trauma caused by a fall following spontaneous aneurysm rupture is not mistaken for trauma that induced the aneurysm rupture.
3. Traumatic rupture of berry aneurysms is possible. Evidence of "severe" head injury (skull fractures and/or brain contusions) is not necessary in order to implicate trauma in aneurysm rupture.
4. The emotional stress associated with various traumatic (or threatened traumatic) events likely plays at least a contributory role in aneurysm rupture. In select cases, the emotional stress may be considered the only or the major cause for aneurysm rupture. In order to implicate the emotional stress of an event as a cause or contributing cause of the intracranial hemorrhage, the victim should have realized that the threat to personal safety was implicit, and the circumstances should be of such a nature as to be commonly accepted as highly emotional. Differentiating the extent of contribution of the physical force

(injury) from that related to emotional stress is usually not possible.

5. Autopsy findings should be consistent with the history as it relates to facial/head trauma. Occasionally, there is no external or internal evidence of injury at autopsy, even when physical contact has occurred. In such cases, investigative information must exist which confirms that trauma did, in fact, occur.
6. Ethanol intoxication may predispose berry aneurysms to rupture.
7. Aneurysms in the posterior/basilar/vertebral artery distribution are more susceptible to traumatic rupture than those in the anterior and middle cerebral artery distribution.
8. Reconstruction of the timing of the event is the key to making a determination of traumatic aneurysm rupture. The closer the onset of symptoms to the event, the more likely there is a cause and effect relationship between trauma (or fear) and rupture. Onset of symptoms during the traumatic event, or immediately following the traumatic event, provides good evidence that the trauma likely played a role in aneurysm rupture. Onset of symptoms during the highly emotional aftermath of a situation lends support to the emotional stress of the situation playing a causative role. Aneurysm rupture that occurs hours to days following a traumatic or emotional event may or may not be related to the traumatic/emotional event.
9. It is important to remember that each case must be evaluated on its own. Death scene investigation and witness statements are often of paramount importance in attempting to determine whether or not trauma or emotional stress played a role in aneurysm rupture.

The following are a set of proposed criteria for ruling a ruptured cerebral artery aneurysm case as a homicide:

1. The onset of symptoms related to aneurysm rupture should occur immediately following the trauma (or fear), or in the short time of emotional/physical distress which typically follows such an event.
2. The autopsy must reveal the presence of a ruptured cerebral artery aneurysm with no findings indicating that the rupture occurred prior to the trauma/emotional stress.
3. In cases where trauma is implicated, investigation must conclusively determine that trauma did, in fact, occur. Autopsy findings must corroborate the history, keeping in mind that some forms of minor facial/head injury may not be evident at autopsy.
4. In cases where emotional stress is implicated, investigation must conclusively determine that intense emotional stress did occur, and that it immediately preceded the onset of symptoms related to aneurysm rupture.
5. The manner of death appropriately can be ruled a homicide if the trauma (or intense emotion) occurred during or as a result of an event that would normally be considered a criminal activity, or as the result of a volitional act by another individual to cause fear, harm, or death.

In many cases, there is insufficient historical/investigative information to satisfy the above criteria. If suspicion exists that the case might represent a homicide, it is appropriate to rule the MOD as “undetermined,” with or without a statement suggesting that the case may represent a homicide. As a corollary, a modified form of the criteria may be utilized in an attempt to determine whether or not accidental trauma can be implicated in deaths related to intracranial hemorrhage. Such modification would require changing

the final criterion to read as follows: The manner of death appropriately can be ruled an accident if the trauma (or the intense emotion) occurred during or as the result of an activity normally considered accidental in nature. Analogous criteria may also be employed when dealing with other natural mechanisms of death that are induced by trauma or intense emotion.

References

1. Hanzlick R, Hunsaker JH, Davis GJ. A guide for manner of death classification. St. Louis, MO: National Association of Medical Examiners, 2001.
2. Goodin J, Hanzlick R. [Mind your manners, part II: general results from the National Association of Medical Examiners manner of death questionnaire, 1995](#). *Am J Forensic Med Pathol* 1997;18:224–7. [\[PubMed\]](#)
3. Hanzlick R, Goodin J. [Mind your manners, part III: individual scenario results and discussion of the National Association of Medical Examiners manner of death questionnaire, 1995](#). *Am J Forensic Med Pathol* 1997;18:228–45. [\[PubMed\]](#)
4. Hirsch CS, Flomenbaum M. Problem-solving in death certification. *ASCP Check Sample* 1995;FP95-1:1–31.
5. Davis JH. Can sudden cardiac death be murder? *J Forensic Sci* 1977;23:384–7.
6. Ford R. Basal subarachnoid hemorrhage and trauma. *J Forensic Sci* 1956;1:117–26.
7. Newbarr FD, Courville CB. Trauma as the possible significant factor in the rupture of congenital intracranial aneurysms. *J Forensic Sci* 1958;3:174–99.
8. Simonsen J. Fatal subarachnoid haemorrhage in relation to minor head injuries. *J Forensic Med* 1967;14:146–55. [\[PubMed\]](#)
9. McCormick WF. The relationship of closed-head trauma to rupture of saccular intracranial aneurysms. *Am J Forensic Med Pathol* 1980;1:223–6. [\[PubMed\]](#)
10. Cohle SD, Cunningham MR, Bauserman SC. Fate, fall, or fear: which was the culprit? *ASCP Check Sample* 1985;FP85-3:1–3.
11. Dowling G, Curry B. Traumatic basal subarachnoid hemorrhage—report of six cases and review of the literature. *Am J Forensic Med Pathol* 1988;9:23–31. [\[PubMed\]](#)
12. Barnard JJ, Hirsch CS. Which came first, the rupture or the impact? *ASCP Check Sample* 1989;FP89-5:1–4.
13. Simonsen J. Traumatic subarachnoid hemorrhage in alcohol intoxication. *J Forensic Sci* 1963;8:97–116. [\[PubMed\]](#)
14. Freytag E. Autopsy findings in head injuries from blunt forces. *Arch Pathol* 1963;75:74–85.
15. Helpert M, Rabson SB. Sudden and unexpected natural death: III. Spontaneous subarachnoid hemorrhage. *Am J Med Sci* 1950;220:262–71. [\[PubMed\]](#)
16. Dinning TAR, Falconer MA. [Sudden or unexpected natural death due to ruptured intracranial aneurysm: survey of 250 cases](#). *Lancet* 1953;2:799–801.
17. Freytag E. Fatal rupture of intracranial aneurysms: survey of 250 medicolegal cases. *Arch Pathol* 1966;81:418–24. [\[PubMed\]](#)
18. Bowen DA. [Ruptured berry aneurysms: a clinical, pathological and forensic review](#). *Forensic Sci Int* 1984;26:227–34. [\[PubMed\]](#)
19. Gonsoulin M, Barnard JJ, Prahlow JA. [Death resulting from ruptured cerebral artery aneurysm—219 cases](#). *Am J Forensic Med Pathol* 2002;23:5–14. [\[PubMed\]](#)
20. Malik MAO. Emotional stress as a precipitating factor in sudden deaths due to coronary insufficiency. *J Forensic Sci* 1973;18:47–52. [\[PubMed\]](#)
21. Eliot RS, Buell JC. Role of emotions and stress in the genesis of sudden death. *J Am Coll Cardiol* 1985;5:95B–98B. [\[PubMed\]](#)
22. Pasternac A, Talajic M. The effects of stress, emotion, and behavior on the heart. *Methods Achieve Exp Pathol* 1991;15:47–57.
23. LeComte D, Fomes P, Nicolas G. [Stressful events as a trigger of sudden death: a study of 43 medico-legal autopsy cases](#). *Forensic Sci Int* 1996;79:1–10. [\[PubMed\]](#)

Additional information and reprint requests:

Joseph A. Prahlow, M.D.
South Bend Medical Foundation
530 N. Lafayette Blvd
South Bend, IN 46601
E-mail: jprahlow@sbfmlab.org