

DECLARATION OF JOHN PLUNKETT, M.D.

I, John Plunkett, M.D., am competent to make this declaration and state and declare as follows:

1. I am a general and forensic pathologist. I attended the University of Minnesota Medical School and graduated in 1972. I had a one-year rotating (general) internship at St. Paul-Ramsey County Hospital after completing Medical School, followed by 5 years of residency in general and forensic pathology at Ramsey and the Hennepin County (Minneapolis) Medical Examiner's Office. I am certified by the American Board of Pathology in Anatomical Pathology, Clinical Pathology, and Forensic Pathology.
2. From 1978, when I completed my training, through December 2004 I served as the Regina Medical Center Laboratory/Pathology Director, Medical Education Director, and the Minnesota Regional Coroner's Office (MRCO) Coroner or Assistant Coroner. I retired as the Laboratory Director and Assistant Coroner on December 31, 2004, and as the Medical Education Director at the end of December 2005.
3. The MRCO is a division of the Regina Laboratory/Pathology Department, which provides contract services for seven Minnesota counties with a total population of approximately 750,000. In 2005, the year of my retirement, the office performed over 2,000 death investigations and 300 autopsies. I have personally performed over 3,000 autopsies, including over 200 on children under the age of two. I have performed three autopsies on infants or young children whose death were due exclusively to inflicted head trauma.
4. I have attached a copy of my CV, which details my academic and professional qualifications, experience, and publications. *See Attachment 1.*
5. I have a special interest in pediatric head injury. In 1999, I published an editorial piece examining some of the claims routinely made by prosecution witnesses at the time regarding fatal head trauma in children. I concluded that these claims were not scientifically supported. I published my first study related to fatal pediatric head trauma in 2001. *See Attachment 2.* At that time, my data was not accepted widely in the medical community, and was viewed as heretical by some. Since that time, medical knowledge has changed, albeit slowly and incompletely.
6. I have been asked by Robert Roberson's present counsel to describe, discuss, and reference evidence regarding pediatric head injury (especially injuries attributed to a "shaking" mechanism and what pediatricians often call "shaken baby syndrome" or "abusive head trauma") that was unavailable, not known, or disputed and controversial in 2002 at the time of Mr. Roberson's trial. There has been a fundamental change in medicine's understanding of pediatric head injury beginning in approximately 2000 and accelerating since 2005. This change involves at least twelve distinct areas:
 - i. The potential lethality of short-distance falls;
 - ii. The potential for a "lucid interval" prior to collapse or the onset of signs/symptoms;
 - iii. The specificity of retinal hemorrhage (RH) for inflicted injury or a specific injury mechanism;

- iv. The specificity of subdural hemorrhage (SDH) in an interhemispheric intracranial distribution for inflicted trauma;
- v. The role of traumatic axonal injury (TAI), often referred to as diffuse axonal injury (DAI) in pediatric brain damage;
- vi. The application of the principles of biomechanics to infant injury evaluation;
- vii. The natural disease “mimics” for inflicted trauma, and the concept of a “differential diagnosis”;
- viii. The improbability of “shaking” as a mechanism for *brain* injury;
- ix. The validity of “confessions” to support a medical diagnosis;
- x. The misinterpretation of injuries due to resuscitation or medical intervention as being due to inflicted trauma;
- xi. The application of principles of evidence-based medicine to evaluate the scientific literature regarding pediatric head injuries; and
- xii. The role that a child-abuse bias can play in distorting the medical inquiry at various stages from presentation of injury through autopsy.

7. It is important to note that although the literature and understanding has been consistently evolving, these views were in the minority in 2002, and experts willing to examine cases and testify in a manner consistent with this scientific knowledge were few and far between at that time.

8. I will discuss several of these areas in detail.

9. ***Short falls, lucid interval, SDH, and RH:*** I studied these areas in an article titled “Shaken baby syndrome and the death of Matthew Eappen - a forensic pathologist’s response”, published in the *American Journal of Forensic Medicine and Pathology* in 1999.⁽¹⁾ The assertions that I examined included the following:

- A low-velocity fall cannot cause serious injury or death;
- The location of a subdural hematoma (SDH) indicates whether an injury was accidental versus inflicted;
- A child with an ultimately fatal head injury does not have a period of time during which he/she appears to be “normal”, i.e., have a lucid interval; and
- Retinal hemorrhage (RH) with specific characteristics indicates an ultimate cause, i.e., accidental versus “shaken baby syndrome”.

Applying principles of evidence-based science, I disproved each of the assertions identified above in a study published in the *American Journal of Forensic Medicine and Pathology* in 2001.⁽²⁾ I analyzed the U.S. Consumer Products Safety Commission (CPSC) database for fatal head injuries associated with the use of playground equipment. I showed that a low-velocity impact *can* cause fatal head injury; that a lucid interval *can* occur in a fatal head injury; that the particular location of an SDH *does not* indicate an abusive cause; and that RH with particular characteristics *does not* indicate whether the injury was “accidental” or was “inflicted.”

10. The *American Journal of Forensic Medicine and Pathology* is a peer-reviewed journal and is the official journal of the American National Association of Medical Examiners. My article was a direct response to assertions made by physicians in a *Letter to the Editor* published

in *Pediatrics* in 1998.⁽³⁾ My critique did not attempt to prove or disprove the statements in the *Pediatrics* letter, but rather to examine their scientific foundations. I concluded that the assertions were scientifically unsound and not based on acceptable reasoning or methodology

11. **Biomechanics:** Professor Werner Goldsmith and I discussed the relevance of the principles of biomechanics to infant injury evaluation in an article published in the *American Journal of Forensic Medicine and Pathology* in 2004.⁽⁴⁾ Ommaya, Thibault, Uscinski, and Prange have also emphasized the contribution of mechanics to pediatric injury analysis.⁽⁵⁻⁸⁾ Biomechanics applies the principles of mechanics (Newton's analysis of motion) to living tissues. Engineers and other scientists have used biomechanics to design safer automobiles and roads; protective equipment for contact sports; devices for fracture repair; stents for blood vessel replacement; playground equipment; safer flooring surfaces; and industrial and household items, among others. Many if not most non-physicians assume that physicians, skilled in the art of medicine, must have particular knowledge of injury mechanisms. This assumption is wrong. Medical schools and post-graduate residency programs (except for orthopedic surgery, physical medicine/physiatry, and occasionally neurosurgery and vascular surgery) do not teach injury mechanics.

12. Physicians (except as above) need not know or apply injury mechanics if they are responsible for diagnosis and treatment. For example, for treatment purposes, it is not necessary to know if a fall from a ladder or a blow from a baseball bat caused a fracture. However, if a physician ventures from diagnosis and treatment to speculation of an ultimate mechanical cause for an injury, he/she *must* understand mechanics and the relevant literature in order to give a reliable expert opinion. Only a few practicing physicians understood these concepts in 2002.

13. Biomechanical analysis of infant head injury and related

14. **Traumatic Axonal Injury (TAI)/Diffuse Axonal Injury (DAI):** Neuroscientists and physiologists have analyzed the mechanical causes for brain nerve fiber damage using sophisticated experimental techniques. These studies have led to the concept of "diffuse axonal injury" (DAI). The theory states that stretching of the nerve fibers may cause either reversible or irreversible damage. These scientists have shown experimentally that impact to the scalp and skull and/or differential acceleration of the brain relative to the scalp and skull may stretch nerve fibers. The degree of stretching is direction-dependent and varies in different parts of the brain, even with apparently identical mechanical input. The scientists performing these experiments have noted that the damage has a typical sequence and distribution. The least severe damage, often referred to as "minimal DAI," involves the junction of the gray and white matter in the cerebral hemispheres. The next area affected is the connection between the right and left cerebral hemispheres, a tissue called the corpus callosum. The most severe damage, and the injury responsible for the most dangerous signs and symptoms, is in the brain stem. Experiments on primates, verified by medical imaging studies in humans, have indicated that the distribution of damage due to impulsive loading is centripetal, in that the damage first affects the gray-white matter interface, then the corpus callosum, and finally the brain stem. Impulsive loading does not cause primary traumatic brain stem damage unless there is also corpus callosum damage; corpus callosum damage does not occur unless there is gray-white matter interface damage.

15. The name “diffuse axonal injury” itself is a misnomer. Studies have shown that the damage is not diffuse, but rather occurs in discrete areas of the brain. Also, although neuroscientists initially used the concept of DAI to describe the effects of mechanical trauma, morphologically identical damage is found in a number of other conditions, including anoxia (or “respirator brain”, a condition that can occur when a person is kept alive on a ventilator for a period of time after collapse). It is therefore imperative to differentiate traumatic axonal injury (“TAI”) from anoxic axonal injury (“AAI”), but this is not always possible. In 2011, a study of 24 children under the age of three years found that experts could not always distinguish between natural and traumatic causes of death on the basis of β -APP immunostaining, which until then pathologists considered a reliable marker of traumatic axonal damage.⁽⁹⁾ The authors concluded, “Children who did not sustain head trauma but who survive resuscitation to linger on ventilator support may have extensive axonal staining that may be interpreted as false-positive evidence of traumatic injury”.⁽⁹⁾

16. Dr. Jennian Geddes, a neuropathologist, and other neuroscientists studied traumatic axonal injury (TAI) as a causative mechanism for infant brain damage in a series of articles published in 2001-2003.⁽¹⁰⁻¹¹⁾ She and her co-authors also examined hypoxia/anoxia as a potential cause for SDH and RH in subsequent studies.⁽¹²⁻¹³⁾ Dr. Geddes’ research raised the possibility that brain findings that previously had been attributed to traumatically torn axons from violent shaking might instead reflect hypoxia/ischemia from any condition that impedes blood flow to the brain. Many conditions may result in such a disruption, including, but not limited to, infection, metabolic abnormalities, and inherited disorders. Since the morphology and distribution of nerve fiber damage due to lack of oxygen may be identical to that of damage due to mechanical loading, it may be difficult, and many researchers say impossible, to differentiate these two potential causes based on the microscopic morphology and distribution alone. Further, physicians should interpret apparent TAI that does not follow a centripetal pattern and is only identifiable microscopically with great caution, especially when there are contemporaneous anoxic changes. Finally, apparent TAI discordant with the primary injury and potential loading conditions is most likely not TAI at all, but due to something else.

17. ***Mimics and the differential diagnosis for infant head injury:*** Dr. Patrick Barnes, a pediatric neuroradiologist at Stanford University Medical Center described the potential and limitations of pediatric neuroimaging in articles published in 2002 and 2007.⁽¹⁴⁻¹⁵⁾ His studies and others have shown that a number of different mechanical and physiological causes, including impact trauma, natural disease, hypoxia/anoxia, and inherited abnormalities of metabolism may lead to identical clinical signs and symptoms. These different causes are the “differential diagnosis.” It is rare in medicine or any biological system to have only a single cause for a clinical finding. It is necessary to have a differential diagnosis when evaluating anyone who presents to the physician’s office or hospital with apparent head trauma, or who dies and is autopsied. There are no “intent” receptors in the brain or eye. Neither structure knows whether a physiologic derangement was caused by an intentional impact, by an accidental impact, by anoxia (lack of oxygen), or by any of a variety of natural diseases. “Intent” does not determine the physiological response of the brain or the eye to an injury.

18. There are many medical conditions that cause or are associated with SDH, RH, and cerebral edema (brain swelling).⁽¹⁶⁻²³⁾ These include but are not limited to:

- a. A variety of infections caused by bacteria and viruses;
- b. Breakthrough bleeding associated with cortical venous thrombosis (CVT), sagittal sinus thrombosis (SST), or other large-sinus thrombosis;
- c. Inborn errors of metabolism such as glutaric aciduria;
- d. Inherited or acquired coagulation abnormalities, such as hypofibrinogenemia, Vitamin K deficiency, or thrombocytosis;
- e. Structural abnormalities such as an arachnoid cyst or increased extra-axial fluid;
- f. Vascular malformations such as, but not limited to, AV malformations; and
- g. Poorly understood inflammatory processes such as hemophagocytic lymphohistiocytosis.

19. Many physicians who completed training prior to 2002 are not aware that these conditions may imitate mechanical trauma unless they are familiar with current scientific literature.

20. **Shaking:** Guthkelch⁽²⁴⁾ and Caffey⁽²⁵⁾ independently proposed “shaking” as a cause for traumatic brain injury in publications in the United Kingdom and the United States more than thirty years ago. Their hypothesis was widely accepted despite existing experimental evidence that it was flawed. Simply stated, the levels of acceleration humanly achievable by an adult shaking an infant are lower by an order of magnitude than those known to cause brain injury. Additional research, published in 2005, established that shaking a toddler generates ten times *less* acceleration than shaking an infant,⁽²⁶⁾ making the already improbable shaking mechanism for infants even less likely in a case of a toddler.

21. Bandak, Prange, and others have evaluated the vulnerability of the infant neck to “shaking”⁽²⁷⁻²⁹⁾. These scientists conclude that a “shaking” capable of causing actual *brain* damage is likely to be associated with significant structural neck damage. There is a single case report in the peer-reviewed literature regarding “neck damage” associated with shaking.⁽³⁰⁾ However, what the authors described as “neck damage” is actually artifacts well recognized by forensic and general pathologists.⁽³¹⁾ The evidence suggests that “shaking” may cause brain stem and cervical spinal cord damage and even death, but there is no evidence that it causes SDH, RH, or brain swelling without causing significant and itself lethal neck damage.

22. The growing evidence of the lack of scientific foundation for “Shaken Baby Syndrome” has lead several government entities and courts to review and in some cases overturn convictions based on the flawed hypothesis. The Government of Ontario established The Inquiry into Pediatric Forensic Pathology on April 25, 2007, and authorized the Commission to conduct a systemic review of the practice of pediatric forensic pathology in Ontario. At these proceedings in 2007 and 2008, witnesses, including the Ontario Deputy Chief Coroner and the Ontario Chief Forensic Pathologist, testified that there is growing scientific doubt regarding the validity of a diagnosis of “shaken baby syndrome”. Dr. Albert Lauwers, the Deputy Chief Coroner, indicated that any “moral, ethical and just society” had a responsibility to review all convictions in which “shaking” was stated to be the mechanism of injury. The Commission adopted Dr. Lauwers’ recommendation.⁽³²⁾ Others, including the United Kingdom Court of Appeals and the Swedish Supreme Court have also questioned the specificity of various signs and symptoms for inflicted

trauma⁽³³⁻³⁴⁾. In the United States, government entities continue to question SBS. In 2014, a Federal judge called SBS “more an article of faith than a proposition of science.”⁽³⁵⁻³⁶⁾ Later that year, a New York court ruled, “the mainstream belief in 2001–2002, espoused by the Prosecution's expert witnesses at Trial, that children did not die from short falls, has been proven to be false.”⁽³⁷⁾

Confessions: Leestma and other experts have analyzed whether “confessions” are an acceptable tool for validating a medical hypothesis. They have concluded that confessions and statements of alleged perpetrators are inadequate to prove a causal mechanism⁽³⁸⁻³⁹⁾. Confessions are not scientific evidence when the researcher does not investigate the underlying events to which the confession refers. Scientific investigation and new knowledge about wrongful convictions have yielded reasons to be increasingly skeptical about the reliability of confessions⁽⁴⁰⁻⁴¹⁾. One review of child abuse literature spanning several decades found only 11 cases that involved both confessions to shaking and no signs of impact⁽³⁸⁾. Other studies have demonstrated that confessions did not reliably match the recorded medical findings, suggesting either that a confession may have understated the actions or that the shaking to which the accused confessed had nothing to do with the medical explanation for the collapse⁽⁴²⁻⁴³⁾.

23. **Evidence-based medicine:** Donohoe⁽⁴⁴⁾ used the principles of evidence-based medicine⁽⁴⁵⁻⁴⁷⁾ to evaluate the published literature regarding Shaken Baby Syndrome. Lantz⁽⁴⁸⁾ and Leestma⁽³⁸⁾ used the same method to evaluate retinal hemorrhage and “confessions associated with infant brain injury.” Both conclude that the published literature fails to meet minimally acceptable scientific evidentiary standards.

24. **Summary and conclusions:** In 2016, physicians, scientists, the public, and the Courts cannot support pre-2002 beliefs and testimony regarding infant head injury. In 2002, if an infant or young child suffered a head injury, physicians often believed “shaking” to be the cause. If there was evidence for an impact, such as a bruise in the scalp or a skull fracture, physicians taught other physicians that there must be a “shaking” component or that the impact must have been a “high-energy event”, equivalent to a motor vehicle accident or a fall from a 2-3 story window. Many non-physicians, such as biomechanical engineers, knew that these beliefs were false. However, biomechanical engineers seldom did a real time evaluation for a hospitalized infant, or assisted a pathologist if the baby died. Their role was to study infant injury mechanisms, and to reconstruct actual events with a goal toward improving protective devices for the motor vehicle industry and sports, among others. Simply stated, biomechanical engineers and clinical physicians rarely communicated. For the most part, they were ships passing in the night.

25. In 2002, many physicians taught other physicians that:

- a. A low-velocity fall cannot cause serious injury or death;
- b. The location of SDH indicates an ultimate cause of the injury (accidental versus inflicted);
- c. A child with an ultimately fatal head injury does not have a period of time during which he/she appears to be “normal”, i.e., have a lucid interval; and
- d. Retinal hemorrhage (RH) with specific characteristics indicates an ultimate cause, i.e., accidental versus “shaken baby syndrome.”

Scientific studies have now shown these assertions to be incorrect ^(2,4).

26. In 2002, it was a prevailing belief among physicians that diffuse axonal injury (“DAI”) was the mechanism of damage in infant head injury. This was wrong. ^(9-13, 49) Importantly, even strong proponents of the “shaking” etiology now acknowledge that the “the widespread cerebral and axonal damage in [abusive head trauma] cases is ischemic rather than directly traumatic.” ⁽⁵⁰⁾

27. In 2002, a practicing physician or pathologist rarely if ever requested a biomechanical analysis of a potential injury mechanism. In 2016, such an analysis is mandatory, because it is now known that the findings historically associated with shaking may be caused by household accidents ⁽⁴⁸⁾ and low-velocity falls. ⁽²⁾

28. In 2002, many physicians believed the only consideration in the differential diagnosis of infant head injury was “shaking” or “shaking with impact”, a “motor vehicle accident”, or a “fall from a multi-story building”. Physicians who testified at Mr. Roberson’s trial asserted that it was “impossible” for a short fall to cause the injuries at issue in this case. See 42RR at 18; 42RR at 84, 85; see also 42RR at 108 (claiming shaking had to be the mechanism of injury because “There had to have been something more than just impact.”)

By 2016, the clinical consensus, as reflected in many resources cited in the endnotes below, requires that the differential diagnosis include not only low-level falls, but also a variety of natural diseases and hypoxia/anoxia. Physicians in 2002 rarely thought of the various natural diseases, such as severe infections, that might mimic inflicted trauma.

29. In 2002, it was a prevailing belief among physicians that “shaking” caused SDH, RH, and brain swelling. In 2016, this belief is not supportable. Further, if it were possible for “shaking” to cause *brain* damage, in such instances it is likely that the infant will have significant *structural* neck damage. There has never been a reliable report of such damage.

30. In 2002, physicians used “confessions” to validate “shaking” as a brain injury mechanism divorced from a methodology that comports with the current clinical consensus. In 2016, a variety of sources, resources, and publications indicate that a “confession,” without other evidence, should not be used to confirm a theory of causation. ⁽³⁸⁻⁴⁰⁾

31. Very few publications prior to 2002 scientifically studied “shaking” as a mechanism of infant brain injury. Articles analyzing the legal evidentiary basis of this mechanism came even later, beginning in around 2003, ⁽⁵¹⁻⁵²⁾ as did calls to reexamine the mechanism as a medical diagnosis. ⁽⁵³⁾ Arguably the most comprehensive analysis of the evidentiary basis was not published until 2012. ⁽⁵⁴⁾

32. At least three studies ^(34, 38, and 42) and one Editorial ⁽⁴⁶⁾ published in the peer-reviewed literature since 2002 use the tools of evidence-based medicine to evaluate the shaken baby syndrome literature. These analyses conclude that the evidence to support “shaking” as an injury mechanism is weak or non-existent. Further, Lantz’ study indicates that a type of RH said to be a specific indicator of “shaking” is not in fact such an indicator. ⁽⁴²⁾ In 2002, there was very little questioning of “shaking” as an injury mechanism. Now such questioning is far more common.

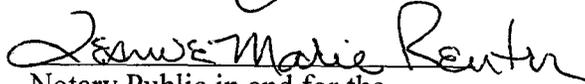
33. The annotations above are representative but not exhaustive. I have not annotated most of the pre-2000 literature. However, many of the annotations contain detailed references to the older studies. I have a complete bibliography of all of the references in the annotations, and they are available upon request.

I swear under penalty of perjury that the foregoing is true and correct.



John J. Plunkett, M.D.

Subscribed and sworn to before me this 7th day of June, 2016.



Notary Public in and for the
State of Minnesota

My commission expires: 1/31/18



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PERSONAL:

Date of Birth: April 15, 1947
Place of Birth: Saint Paul, Minnesota
Citizenship: United States of America
Family: *Spouse:* Donna McFarren Plunkett
Children: Matthew James (1971)
Benjamin John (1973)

EDUCATION:

BS, History and Chemistry (1972); University of Minnesota; Minneapolis, Minnesota; 1966 – 1969

MD (1972); University of Minnesota; Minneapolis Minnesota; 1969 – 1972

POSTGRADUATE TRAINING AND EXPERIENCE:

Rotating Internship; Saint Paul Ramsey Medical Center; Saint Paul, Minnesota; 1972 – 1973

Anatomic and Clinical Pathology Residency; Saint Paul Ramsey Medical Center; Saint Paul, Minnesota; 1973 – 1978

Forensic Pathology Fellowship; Hennepin County Medical Examiner's Office; Minneapolis, Minnesota; 1975 – 1976

BOARD CERTIFICATION:

Anatomic Pathology, Clinical Pathology and Forensic Pathology; American Board of Pathology; 1978

MEDICAL LICENSURE:

Minnesota and Wisconsin

EMPLOYMENT:

Hennepin County Deputy Medical Examiner; 1975 – 1984

Hennepin County Assistant Medical Examiner; 1984 – 1985

Laboratory and Medical Education Director, Regina Medical Center; Hastings, Minnesota; 1978 – 2004

Laboratory Director, Cannon Falls Community Hospital; Cannon Falls, Minnesota; 1981 – 2004

Coroner, Minnesota Regional Coroner's Office; Hastings, Minnesota; 1980 – 1998

Assistant Coroner, Minnesota Regional Coroner's Office; 1999 – 2004

PROFESSIONAL ORGANIZATIONS:

Twin Cities Medical Society

Minnesota Medical Association

American Medical Association

American Society of Clinical Pathologists (Fellow); 1976 – 2004

College of American Pathologists (Fellow; Emeritus: 2011 -)

Minnesota Society of Pathologists; 1978-2001

- Minnesota Medical Association Interspeciality Council Representative; 1991 – 1998
- Member of the MSP Executive Committee

Twin City Society of Pathologists; 1984 – 2001

Minnesota Coroners and Medical Examiners Association; 1974 – 2001

- President, 1981 and 1985)

National Association of Medical Examiners (Fellow)

American Academy of Forensic Sciences (Fellow)

SPECIAL APPOINTMENTS:

College of American Pathologists; Inspector, Laboratory Accreditation Program (1984 – 1994)

Minnesota Coroners and Medical Examiners Association; Executive Committee (1978 – 1998)

Regina Medical Center Operating Board (1991 – 1996)

Regina Medical Center Medical Staff Executive Committee (1985 – 1994)

- President of the Medical Staff (1987 – 1990)

Chairman, Regina Medical Center Infection Control Committee (1978 – 1990, 1993 – 1999)

Minnesota Department of Health, Epidemiology Section, Emerging Infectious Diseases Program (Appointed Member, Hospital-based Physician) (1988 – 1998)

Regina Medical Center Foundation Board Member (2011 – present)

Reviewer, *The Lancet*

Reviewer, *Forensic Science International*

Reviewer, *Acta Paediatrica*

Reviewer, *Journal of Forensic Sciences*

Reviewer, *British Medical Group*

Reviewer, *Journal of Forensic Biomechanics*

HOSPITAL STAFF APPOINTMENTS:

Regina Medical Center (Active, 1978 – 2004; Courtesy, 2005 – 2012; Honorary, 2012 - present)

Cannon Falls Community Hospital (Active, 1980 – 2004)

PUBLICATIONS AND NATIONAL PRESENTATIONS:

1. Tan RE, Noreen JP, Plunkett JJ. Chronic intussusception following intestinal bypass surgery for morbid obesity. *Abdominal Surgery* 1981;23:76-8.
2. Plunkett J. Sudden death and myocardial infarction in Minnesota. *NEJM* 1984;310:1187-9 (letter).
3. Plunkett JJ, Amatuzio JC. Clostridial sepsis and sudden death. Abstract presented at the AAFS National Meeting, February 1985.
4. Plunkett JJ, Amatuzio JC. Sudden infant death: I: Cost analysis of investigative procedures. Abstract presented at the ASCP Fall Meeting, 1985.
5. Plunkett JJ, Amatuzio JC. Sudden infant death: II: Ten years experience in three Minnesota counties. Abstract presented at the ASCP Fall Meeting, 1985.
6. Plunkett JJ, Amatuzio JC. Sudden infant death: III: Sudden non-SIDS natural deaths in infancy. Abstract presented at the AAFS Annual Meeting, February 1986.
7. Amatuzio JC, Plunkett JJ. Hemophilus influenzae sepsis in an asplenic adult. Abstract presented at the AAFS Annual Meeting, February 1985.
8. Plunkett J, Amatuzio JC. Electrical injury and death in three Minnesota counties. Abstract presented at the AAFS Annual Meeting, February 1986.
9. Plunkett J. Serum tests for diagnosis of iron deficiency. *AJCP* 1990;94:524-5 (letter).
10. Plunkett J. Minnesota infant death investigation guidelines. Minnesota Coroners and Medical Examiners Association, October 5, 1990.
11. Plunkett J, et al. Guidelines for Blood Component Transfusion. American Red Cross North Central Blood Services, ARC/NCBS (1997).

12. Plunkett J. Restricting the time of injury in fatal inflicted head trauma. *Child Abuse Negl* 1998;22:943-4 (letter).
13. Plunkett J, Thomas LC. Medical examiner and coroner systems. *JAMA* 1998;280:325 (letter).
14. Plunkett J. Shaken baby syndrome and the death of Matthew Eappen: a forensic pathologist's response. *Am J Forens Med Pathol* 1999;20:17-21.
15. Plunkett J. Sudden death in an infant caused by rupture of a basilar artery aneurysm. *Am J Forens Med Pathol* 1999;20:211-214.
16. Plunkett J. Recognizing abusive head trauma in children. *JAMA* 1999;282:1421-1422 (letter).
17. Plunkett J, Plunkett M. Physiologic periosteal changes in infancy. *Am J Forens Med Pathol* 2000;21:213-216.
18. Plunkett J. Clarity on the diagnosis line. *Ann Diagn Path* 2000;4:134 (letter).
19. Plunkett J. Fatal pediatric head injuries caused by short-distance falls. *Am J Forens Med Pathol* 2001;22:1-12.
20. Plunkett J. Author's response to Drs. Spivack and Levin. *Am J Forens Med Pathol* 2001;22:417-19 (letter).
21. Plunkett J. Author's response to fatal pediatric head injuries caused by short distance falls. *Am J Forens Med Pathol* 2002;23:103-04 (letter).
22. Geddes JF, Plunkett J. The evidence base for shaken baby syndrome. *Br Med J* 2004;328:719-20 (Editorial).
23. Goldsmith W, Plunkett J. A biomechanical analysis of the causes of traumatic brain injury in infants and children. *Am J Forens Med Pathol* 2004;25:89-100.
24. Miller M, Leestma J, Barnes P, Carlstrom T, Gardner H, Plunkett J, et al. A sojourn in the abyss: hypothesis, theory, and established truth in infant head injury. *Pediatr* 2004;114:326 (letter).
25. Plunkett J. Resuscitation injuries complicating the interpretation of premortem trauma and natural disease in children. *J Forens Sci* 2006;51:127-30.
26. Van Ee C, Moroski-Browne B, Raymond D, Thibault K, Hardy W, Plunkett J. Evaluation and refinement of the CRABI-6 anthropomorphic test device injury criteria for skull fracture. Proceedings of the ASME 2009 International Mechanical Engineering Congress & Exposition, IMECE 2009-12973.
27. Van Ee C, Raymond D, Thibault K, Hardy W, Plunkett J. Child ATD reconstruction of a fatal fall. Proceedings of the ASME 2009 International Mechanical Engineering Congress & Exposition, IMECE 2009-12994.

INVITED LECTURES, PRESENTATIONS, AND CONFERENCES (1989-1994):

1. "Fundamentals of Death Investigation"; Minnesota Bureau of Criminal Apprehension; Brainerd, Minnesota; March 7, 1990 (7 hours, POST approved).

2. "Preleukemia and Dysmyelopoietic Syndromes"; Regina Medical Center Medical and Professional Staff; March 28 and April 4, 1990 (1 hour, AAFP prescribed credit and AMA Category I credit approved).
3. "Arterial Blood Gas Analysis and Monitoring"; Northfield City Hospital Medical and Professional Staff; Northfield, Minnesota; April 18, 1990 (1 hour, AAFP prescribed credit approved).
4. "Cancer Genetics, Epidemiology and Primary Prevention"; American Cancer Society; October 3, 1990 (1.5 hours, Nursing CEU approved).
5. "Fundamentals of Death Investigation"; Minnesota Bureau of Criminal Apprehension; Saint Paul, Minnesota; October 11, 1990 (7 hours, POST approved).
6. "Plasma Cell Dyscrasias and Hypercalcemia of Malignancy"; Regina Medical Center Medical and Professional Staff; January 2, 1991 (1 hour, AAFP prescribed credit and AMA Category I credit approved).
7. "Plasma Cell Dyscrasias and Hypercalcemia of Malignancy"; Northfield City Hospital Medical and Professional Staff; January 8, 1991 (1 hour, AAFP prescribed credit and AMA Category I credit approved).
8. "A Critical Analysis of Recommendations for Hepatitis Immunization"; Regina Medical Center Medical and Professional Staff; January 10, 1991 (1 hour, AAFP prescribed credit and AMA Category I credit approved).
9. "A Rational Approach to Evaluation of an Anemic Patient"; Visiting Professor Series, University of Illinois, College of Medicine at Urbana-Champaign; February 21, 1991 (1 hour, AMA Category I credit approved).
10. "Evaluation of Thyroid Function"; Visiting Professor Series, University of Illinois, College of Medicine at Urbana-Champaign; February 21, 1991 (1 hour, AMA Category I credit approved).
11. "The Triumph of Hope over Science and Sanity: The Cholesterol Myth"; Visiting Professor Series, University Of Illinois, College of Medicine at Urbana-Champaign; February 22, 1991 (1 hour, AMA Category I credit approved).
12. "Infant Death Investigation"; Visiting Professor Series, University of Illinois, College of Medicine at Urbana-Champaign; February 22, 1991 (1 hour, AMA Category I, credit approved).
13. "Infant Death Investigation"; Regina Medical Center Medical and Professional Staff; February 27, 1991 (1 hour, AAFP prescribed credit and AMA Category I credit approved).
14. "Fundamentals of Death Investigation"; Minnesota Bureau of Criminal Apprehension; Worthington, Minnesota; March 20, 1991 (7 hours, POST approved).
15. "The Autopsy and the Role of a Pathologist in Wrongful Death Cases"; Minnesota Trial Lawyers Association; Minneapolis, Minnesota; May 2, 1991 (1 hour, CLE approved).
16. "Death Investigation"; Scott County Law Enforcement; Shakopee, Minnesota; June 6, 1991 (2 hours, POST approved).
17. "Physiologic Effects of Firearms"; Dakota County Law Enforcement; Rosemount, Minnesota;

June 12, 1991 (2 hours, POST approved).

18. "Fundamentals of Death Investigation"; Minnesota Bureau of Criminal Apprehension; Grand Rapids, Minnesota; August 14, 1991 (7 hours, POST approved).
19. "Selected Topics in Surgical Pathology"; Regina Medical Center Medical and Professional Staff; September 4, 1991 (1 hour, MFP prescribed credit and AMA Category I credit approved).
20. "How to Examine Medical Experts"; Minnesota State Bar Association; Minneapolis, Minnesota; October 1, 1991 (8 hours, CLE approved).
21. "Implication of Laboratory Test Results for Nursing Personnel"; South Suburban Medical Center Nursing Staff; Farmington, Minnesota; October 29, 1991 (1.5 hours, Nursing CEU approved).
22. "Peripheral Morphology, Bilirubin Determinations and Acute Leukemia"; Regina Medical Center Medical and Professional Staff; October 30, 1991 (1 hour, AAFP prescribed credit and AMA Category I credit approved).
23. "Selected Topics in Laboratory Medicine"; Northfield City Hospital Medical and Professional Staff; November 19, 1991 (1 hour, AAFP prescribed credit approved).
24. "Infant Death Investigation"; Minnesota Bureau of Criminal Apprehension, Advanced Child Abuse Investigations; Rochester, Minnesota; November 20, 1991 (2.5 hours, POST approved).
25. "Death by Natural Causes"; Minnesota Chiefs of Police Association; March 25, 1992 (1 hour, POST approved).
26. "Infant Death Investigation", Minnesota Chiefs of Police Association, March 25, 1992 (1 hour, POST approved).
27. "Infant Death Investigation"; Minnesota Bureau of Criminal Apprehension; Saint Paul, Minnesota; April 1, 1992 (2.5 hours, POST approved).
28. "Cervical Cytology and the Bethesda Classification System"; Regina Medical Center Medical and Professional Staff; May 27, 1992 (1 hour, AAFP prescribed credit and AMA Category I credit approved).
29. "Infant Death Investigation"; Minnesota Bureau of Criminal Apprehension; Alexandria, Minnesota; October 8, 1992 (2.5 hours, POST approved).
30. "Fundamentals of Death Investigation"; Minnesota Bureau of Criminal Apprehension; New Ulm, Minnesota; October 14, 1992 (2.5 hours, POST approved).
31. "The Laboratorian's Role in Forensic Medicine"; Divine Redeemer Memorial Hospital; South Saint Paul, Minnesota; October 2, 1992 (1 hour, AMA Category I credit approved).
32. "Decision Analysis in Laboratory Medicine"; Northfield City Hospital Medical and Professional Staff; December 15, 1992 (1 hour, AAFP prescribed credit approved).
33. "How to Examine Medical Experts"; Minnesota State Bar Association; Minneapolis, Minnesota; March 11, 1993 (8 hours, CLE approved).
34. "Medical Investigation of Motor Vehicle Fatalities"; Minnesota Chiefs of Police Association;

March 24, 1993 (2 hours, POST approved).

35. "Infant Death Investigation"; Minnesota Bureau of Criminal Apprehension; Fairmont, Minnesota; May 27, 1993 (2.5 hours, POST approved).
36. "How to Examine Medical Experts"; Minnesota State Bar Association; Minneapolis, Minnesota; October 14, 1993 (8 hours, CLE approved).
37. Invitational Working Conference, Vulnerable Adult Act Issues; State of Minnesota, Office of the Attorney General; Saint Paul, Minnesota; November 9, 1993.
38. "Time of Death Determinations"; Northfield City Hospital, EMT/Paramedics; March 14, 1994 (1 hour, EMT/Paramedic CEU).
39. "How to Examine Medical Experts"; Minnesota State Bar Association; Minneapolis, Minnesota; March 24, 1994 (8 hours, CLE approved).
40. "Selected Topics in Laboratory Medicine"; Regina Medical Center Medical and Professional Staff; March 30, 1994 (1 hour, AAFP prescribed credit and AMA Category I credit approved).
41. "Decision Analysis in Laboratory Medicine and Pathology"; Minnesota Society of Pathologists; Minneapolis, Minnesota; April 29, 1994 (1 hour, AMA Category I credit approved).

INVITED LECTURES, PRESENTATIONS, AND CONFERENCES (MAY 1994 THROUGH-2005):

I did not maintain a list of lectures, presentations, and conferences during this time. However, I made presentations to several state and local Public Defender organizations; to the Neurosciences Unit at the Radcliffe Infirmary (Oxford, England); and for the American Society of Neuroradiology; among others.

INVITED LECTURES, PRESENTATIONS, AND CONFERENCES (2006-PRESENT):

1. "Differential Diagnoses in Infant Brain Injury"; Eaton Foundation; Royal College of Medicine, London, United Kingdom; May 16, 2006.
2. "Mechanisms, Mimics, and Differential Diagnoses in Infant Brain Injury"; South Carolina Association of Criminal Defense Lawyers; Greenburg, South Carolina; July 14, 2006.
3. "Mechanisms, Mimics, and Differential Diagnoses in Infant Brain Injury"; Ohio Association of Criminal Defense Lawyers; Columbus, Ohio; October 6, 2006.
4. "Infant Injury Evaluation"; Oregon Criminal Defense Lawyers Association; Portland, Oregon; December 2, 2006.
5. "State v. Plunkett: When the State Loses, The Expert Gets Indicted"; Oregon Criminal Defense Lawyers Association; December 2, 2006.
6. "Mechanisms, Mimics, and Differential Diagnoses in Infant Brain Injury"; Los Angeles County Public Defenders Association; Los Angeles, California; September 15, 2007.

7. "Mechanisms, Mimics, and Differential Diagnoses in Infant Brain Injury"; Texas Criminal Defense Lawyers Association; Dallas, Texas; March 4, 2008.
8. "Mechanisms, Mimics, and Differential Diagnoses in Infant Brain Injury"; Wisconsin Criminal Defense Lawyers Association; Milwaukee, Wisconsin; March 14, 2008.
9. "The Differential Diagnosis for Subdural Hemorrhage in Children under the Age of Two"; Evidence-Based Medicine and Science Symposium; Denver, Colorado; February 21, 2009.
10. "Infant and Toddler Falls"; Hershey Medical Center Pediatric Abusive Head Trauma Conference; Jackson Hole, Wyoming; June 26, 2009.
11. "The Differential Diagnosis for Subdural Hemorrhage in Children under the Age of Two"; Iowa Public Defender Criminal Law Seminar; Tama, Iowa; June 18, 2010.
12. "The Differential Diagnoses for Apparent Head Trauma in Infants and Toddlers"; Illinois State Appellate Defender Seminar; Collinsville, Illinois; October 15, 2010.
13. "The Mechanics and Mimics for Apparent Head Trauma in Infants and Toddlers"; National Association of Criminal Defense Lawyers and the Innocence Network (supported by a grant awarded by the Bureau of Justice Assistance); Cincinnati, Ohio; April 7, 2011 (with Bridget McCormack).
14. "EBM, Mechanics and Mimics in the Evaluation of Apparent Head Trauma in Infants and Toddlers"; Evidence-based Medicine and Social Investigation; Surrey, BC; August 5, 2011.
15. "The Mechanics and Mimics for Apparent Head Trauma in Infants and Toddlers"; Georgia Innocence Project; Atlanta, Georgia; October 12, 2011.
16. "The Current State of Shaken Baby Syndrome. How Did We Get To This Place?"; The Capital Aggravator: Death of a Child under Age 10. A Program for the Defense; The Center for American and International Law; Plano, Texas; March 1, 2012 (with Sue Luttner).
17. "Mimics for Apparent Inflicted Head Trauma in Infants and Toddlers"; Evidence-based Medicine and Social Investigation; Surrey, BC; August 5, 2011.

SPECIAL INTERESTS:

Decision analysis in laboratory medicine and pathology

Continuing education for the medical and legal profession, law enforcement and the community

Head injury in children