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Pediatric Forensic Pathology: Limits and Controversies

Commissioned by

The
Inquiry into Pediatric Forensic Pathology

Ontario, Canada

(As amended, 15 February 2008)



This laboratory is registered under the registration scheme of
the National Association of Testing Authorities, Australia and
The Royal College of Pathologists of Australasia.



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November 2007

Title Page

This paper was written by Stephen Cordner (1), Jonathon Ehsani (2), Lyndal Bugeja (3), and Joseph Ibrahim (4) from the Victorian Institute of Forensic Medicine in Australia for the Inquiry into Pediatric Forensic Pathology in Ontario, Canada. The authors thank Dr Jeffrey Hubbard, Albany, New York for his comments on this paper. Any faults are ours, however. Editorial decisions and the opinions expressed are the responsibility of the first author and do not represent those of the Victorian Institute of Forensic Medicine or Monash University or of the Inquiry into Pediatric Forensic Pathology or the Commissioner.

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The authors acknowledge with thanks the assistance of Ms Kerry Johannes, Library Manager at the Victorian Institute of Forensic Medicine.

Soothsayer: *In nature's infinite book of secrecy*

A little I can read.

—*Antony and Cleopatra*, Act 1, Scene ii

Abbreviations

BAPP	Beta-amyloid precursor protein
CPR	Cardio-Pulmonary Resuscitation
CSF	Cerebro-spinal fluid
DAI	Diffuse Axonal Injury
FII	Fabricated or Induced Illness (see also MSbP)
HDN	Hemorrhagic disease of the newborn
IHT	Inflicted Head Trauma
ILO	International Labour Organisation
LQTS	Long QT Syndrome
MSbP	Munchausen Syndrome by Proxy (see also FII)
NAI	Non-accidental injury
NAHI	Non-accidental head injury
NCIS	National Coroners Information System
NT	Northern Territory
QAP	Quality Assurance Program
SA	South Australia
SDH	Subdural hemorrhage
SBS	Shaken Baby Syndrome
SIDS	Sudden Infant Death Syndrome
SUDI	Sudden Unexplained Death in Infancy
UK	United Kingdom
USA	United States of America
VIFM	Victorian Institute of Forensic Medicine
WA	Western Australia
WHO	World Health Organization

Glossary and Definitions

Acute	Of recent origin.
Anoxia	Absence of oxygen in the blood or tissues.
Ante-partum	Before the beginning of labour.
Anterior*	Anatomical term: to the front (of).
Anthropophagy	The generally post-mortem phenomenon of animals, sea creatures or insects consuming human tissue.
Artefact (artifact)	Artificial product. In relation to autopsy, it is a sign or finding imitating pathology, disease, or injury occurring in life.
Asphyxia	A complex and confusing term used in varying ways by different authors. It is discussed in detail in this paper. The common notion of asphyxia is a mechanical interference of some sort with breathing.
Atrial septal defect	“A hole in the heart”; usually small and of little significance.
Autopsy	The post-mortem examination of a body involving its external and internal examination and incorporating the results of special tests. In a full autopsy, the internal examination involves, but is not limited to, examining the contents of the cranium, chest, and abdomen. Further dissection can and should occur in particular circumstances.
Axon	A nerve fibre.
Bilateral	Both sides (of the body).
Biochemistry	The chemistry of biology, the application of the tools and concepts of chemistry to living systems [1].
Born	Complete expulsion of the baby from the mother.
Born alive	Complete expulsion from the mother and an independent existence from the mother (established breathing and circulation).
Brainstem	The connection between the brain and the spinal cord; an enlarged extension of the spinal cord attaching to the brain within the skull.
Bruise, bruising	Bleeding into tissues from damaged blood vessels, usually as a result of external injury. Most commonly understood as bruise in or under the skin, but can occur in any tissue, e.g., muscle, heart, liver, etc.
Calvarium	The vault of the skull.
Caput succedaneum	Diffuse swelling of the scalp in a newborn caused by pressure from the uterus or vaginal wall during a head-first (vertex) delivery.
Cardiac	To do with the heart.

Case series	A study reporting observations on a series of individuals, usually all receiving the same intervention, with no control group [2].
Cardiopulmonary resuscitation	Compression of the chest and artificial respiration in attempts to restart the beating of the heart and spontaneous breathing.
Cerebellar tonsillar herniation	Protrusion, and consequently compression of, the cerebellum into the foramen magnum as a result of raised pressure inside the skull. This is a very serious and often fatal development when associated also with compression of the brain stem (coning).
Cerebral edema	Swelling of the brain due to an extracellular accumulation of watery fluid.
Cerebral parenchymal injury	Injury to the substance of the cerebral hemispheres (the largest part) of the brain
Cerebro-spinal fluid (CSF)	The clear fluid circulating around and within the spaces—ventricular system—of the brain and spinal cord.
Choking	Internal obstruction of the larynx, laryngo-pharynx (i.e., the back of the throat), such as by a large piece of food. Sometimes colloquially used to equate with manual strangulation.
Clinical	Relating to patients.
Computerized tomography (CT) scanner; computerized axial tomography (CAT)	CT scanning computes multiple X-ray images to generate cross-sectional and other views of the body's anatomy. It can identify normal and abnormal structures and be used to guide procedures [1].
Congenital	Born with.
Coning	Compression of the brain stem in the foramen magnum because of raised pressure in the skull (or technically, raised intra-cranial pressure).
Conjunctiva, -ae, -al	The thin membrane lining of the inner aspect of the eyelids and covering the eyes.
Contusion	Bruise, bruising.
Coronary atherosclerosis	Hardening of the arteries of the heart, heart disease, heart attack, myocardial infarction.
Cutaneous	Relating to the skin.
Cyanosis	A bluish colour of the skin and the mucous membranes reflecting the absolute amount of insufficiently oxygenated blood. For example, the lips may show cyanosis. Cyanosis can be evident at birth, as in a "blue baby" who has a heart malformation that permits blood that is not fully oxygenated to enter the arterial circulation. Its significance as an observation post mortem (as opposed to a clinical observation in life) is minimal [1].
Dermis	The leathery part of the skin, covered by the scaly epidermis.
Duodenum	The first part of the small intestine encountered by food as it leaves the stomach. It

	connects with the jejunum.
Encephalopathy	See hypoxic ischemic-encephalopathy
Embolus, embolism	See Thrombus
Epicardium, epicardial	The outer aspect of the heart.
Epidermis	The scaly outer layer of the skin overlying the leathery dermis.
Extradural	Outside the dura, the membrane lining the inner aspect of the skull, and between it and the skull. Usually used in reference to extradural hemorrhage accompanying head trauma in some circumstances.
Extravasation	The presence of blood cells in the tissues outside blood vessels; bleeding; hemorrhage.
Filicide	The killing of a child by a parent.
Fontanel	The fontanel is a "soft spot" of the skull between the skull bones that has not yet hardened into bone. There are normally two fontanels, both in the midline of the skull: the anterior and posterior fontanel. The posterior fontanel closes by the age of about 8 weeks in a full-term baby. And the anterior fontanel closes at 18 months of age on the average but it can close as early as nine months. If they are sunken or tense, that might indicate a problem [1].
Foramen magnum	The hole at the bottom of the skull through which the spinal cord joins the brain stem and, thus, the brain.
Forensic	Relating to the courts, or more generally, the law.
Forensic Medicine	The application of the principles and practices of medicine to the needs of the law. The term (and not the practice) encompasses forensic pathology, clinical forensic medicine, forensic psychiatry and, in more recent times, the area of medical law and ethics.
Forensic Pathology	Part of pathology and is particularly concerned with the investigation of sudden and unexpected deaths from all causes. The discipline of pathology concerned with the investigation of deaths where there are medico-legal implications.
Forensic Science	The application of science to the needs of the law. Operationally, in the developed world, there are few forensic scientists. Rather, there are forensic biologists, forensic chemists, botanists, toxicologists, entomologists, etc.
Frenulum, -a	A thin small isthmus of tissue, usually referring to the inside of the mouth where the upper and lower lips meet the gums at the midline.
Frontal (region of head, skull)	Forehead region.
Galea, sub-galeal	The tough fibrous tissue immediately applied to the outer part of the skull. Sub-galeal refers to the potential space between the galea and the outer skull. Bleeding into this potential space is associated with fractures, but can occur in the absence of fractures.

Hemorrhage	Bleeding.
Histochemical	Chemical alterations in tissue which can be demonstrated and viewed under the microscope.
Histology (histological)	Microscopic study of the structure of tissues; a small piece of tissue, perhaps the size of a coin, is removed at the autopsy, fixed in formalin, and then prepared in such a way that a very thin slice (called a histological section) can be examined under the microscope to make findings not visible to the naked eye.
Humerus; -al	The long bone of the upper arm; relating to.
Hyperaemia	Congestion with blood.
Hypoxia	Sometimes referred to as hypoxaemia or suboxia. Low levels of oxygen in the blood.
Hypoxic-ischaemic encephalopathy	A disorder of the brain due to reduction of the flow of blood to the brain, and/or a reduction of the oxygen in the blood flowing to the brain, causing brain damage. It is regarded as one of the features of Shaken Baby Syndrome. It occurs in other circumstances and conditions as well.
Infant, infancy	In this paper, a baby up to the age of 12 months. Sometimes refers to the period following the neonatal (qv) period and up to 12 months of age.
Infanticide	<p>A woman is guilty of the crime of infanticide who causes the death of her child in circumstances that would constitute murder and, at that time, the balance of her mind was disturbed because of:</p> <ul style="list-style-type: none"> - her not having fully recovered from the effect of giving birth to that child within the preceding 12 months; or - a disorder consequent on her giving birth to that child within the preceding 12 months. <p>(This definition, which is essentially the legal definition of a crime, may vary between jurisdictions.)</p> <p>The word appears to have been used historically in forensic pathology to indicate all forms of homicide of babies around the time of birth.</p>
Inflicted Head Trauma	As opposed to accidental head trauma. Incorporates blunt trauma, shaking, and a combination of the two.
Inferior*	Anatomical term: below.
Intra-cranial	Within the cranium; in this paper, often in relation to hemorrhage, in which case it includes extra-dural, sub-dural, sub-arachnoid and intra-cerebral hemorrhage.
Intra-oral	Inside the mouth.
Intra-partum	During labour; during the process of birth.
Manner of death	The way, or circumstances, in which the death occurred; broadly, whether accidental, suicidal, homicidal, or natural.
Mechanical asphyxia	The common understanding of the term asphyxia. Mechanical interference with breathing including smothering, choking, throttling (manual strangulation), ligature

	strangulation, hanging, and severe sustained compression of the chest (and abdomen) termed traumatic asphyxia.
Medicine	The science and art concerned with the cure, alleviation, and prevention of disease, and with the restoration and preservation of health. Also, that branch of medicine that is the province of the physician (as opposed, for example, to the surgeon).
Metaphysis; -eal	A term used describing an aspect of the developing skeleton. Long bones grow outwards from a primary ossification centre in the middle of their shaft (or diaphysis) towards each end of the bone, by converting cartilage to bone. Secondary ossification centres, or epiphyses, develop at each end of the bone. The part of the diaphysis immediately adjacent to the epiphyseal cartilage is the site of advancing ossification, is known as the metaphysis, and is the part most vulnerable to damage when subject to trauma; relating to the metaphysis.
Micron	A unit of length: one millionth of a metre; one thousandth of a millimetre.
Neonate, neonatal	The period from birth to 28 days post-partum; sometimes the period from birth to seven days postpartum.
Occipital (region of the skull)	The region of the back of the head.
Edema	Abnormal collection of watery fluid within tissue; it can be extracellular and/or intracellular.
Pediatrics	That branch of medicine involving the diagnosis and treatment of illness in children. It has long been recognized as a separate medical speciality because of the different medical issues that children and adults face.
Pediatric Forensic Pathology	Pediatric forensic pathology is a subspecialty of forensic pathology dealing with cases involving children. They define it as the study of diseases and injuries of children with subsequent medico-legal interpretation of findings for police and the courts [3].
Parietal (region of the skull)	The region of the top of the head and toward the back of the head.
Pathogenesis	The path by which a particular pathology is arrived at.
Pathology	The study of disease, and the ways in which disease processes affect our bodies. Recognizing the pattern that disease takes allows an understanding of the root of a problem, enabling accurate diagnosis, treatment, and prevention.
Perinatal	Around the time of birth; that is, shortly before, during, or shortly after birth.
Peritoneal cavity	The more technical term for the most part of the abdominal cavity lined by the peritoneum, a very thin layer of cells.
Petechiae; petechial hemorrhages	Hemorrhage, usually pin point in size but sometimes more coarse. Sometimes used interchangeably with Tardieu's Spots.
Pleura, pleural	The outer lining or aspect of the lungs (visceral pleura) or, immediately next to this, the inner lining of the chest wall (parietal pleura).
Population studies	An observational analytical study based on aggregated secondary data. Aggregate

data on risk factors and disease prevalence from different population groups is compared to identify associations. Because all data are aggregate at the group level, relationships at the individual level cannot be empirically determined but are rather inferred from the group level.[4].

Positional asphyxia	A form of mechanical asphyxia where the position of the deceased, often aggravated by some other factor affecting the consciousness of the individual or his/her ability to move, results in obstruction to or other impairment of respiration.
Post-mortem	After death; short hand for post-mortem examination—See Autopsy.
Postpartum	The period after birth.
Posterior*	Anatomical term: behind; to the back of.
Primary studies	Studies based on original data, not reviews of other studies.
Prospective studies	Data collection and the events of interest occur after individuals are enrolled (e.g. clinical trials and cohort studies). This prospective collection enables the use of more solid, consistent criteria and avoids the potential biases of retrospective recall. Prospective studies are limited to those conditions that occur relatively frequently and to studies with relatively short follow-up periods so that sufficient numbers of eligible individuals can be enrolled and followed within a reasonable period [4].
Proximal jejunum	The small bowel is divided into the duodenum (q.v.), the jejunum, which is the first half of the remaining small bowel, terminating in the ileum. Proximal refers to the part closer to the stomach than the more distal part closer to the ileum.
Purulent peritonitis	Purulent means pus-generating; peritonitis is inflammation of the peritoneum, the lining of the abdomen, and purulent peritonitis is indicative of serious abdominal disease or injury.
Pulmonary	To do with the lungs.
Radionuclide bone scan	A form of radiological investigation of bone
Retinal hemorrhages	Hemorrhages seen in the retina during life, or by pathological examination after death. The specificity and sensitivity of the association of this finding with Shaken Baby Syndrome is one of the controversies in pediatric forensic pathology.
Retrospective studies	All events of interest have already occurred and data are generated from historical records (secondary data) and from recall (which may result in the presence of significant recall bias). Retrospective data is relatively inexpensive compared to prospective studies because of the use of available information and is typically used in case-control studies. Retrospective studies of rare conditions are much more efficient than prospective studies because individuals experiencing the rare outcome can be found in patient records rather than following a large number of individuals to find a few cases [4].
Sampling bias	A systematic difference in characteristics between those who are selected for study and those who are not [2].
Section (histological)	A very thin slice of tissue, around seven microns thick, which is placed on a small glass slide, colourized with chemicals, and viewed under the microscope.

Shaken Baby Syndrome	Sometimes called Shaken Infant Syndrome or, more generally, included with Inflicted Head Trauma (IHT) A serious illness characterized by subdural hemorrhage, petechial and other hemorrhages in the retina, hypoxic/ischaemic encephalopathy, usually in circumstances where there is no evidence of blunt impact to the head. Injuries to the neck such as hemorrhage around cervical spine nerve roots may also be present.
Shaken Impact Syndrome	A combination of the signs of Shaken Baby Syndrome together with evidence of blunt impact trauma to the head.
Siderophages	Particular cells, macrophages, which have a scavenging function, in this case containing iron that can be seen under the microscope. Iron, being a constituent of haemoglobin, which is carried by red blood cells, can be marker of previous hemorrhage at that site. Such hemorrhage or bleeding may have been due to injury.
Simulation studies	An approach to a research question using models constructed to re-create the parameters involved as faithfully as possible. Particularly important in head injury research because of the difficulty of translating the results of animal studies to humans.
Smothering	External obstruction of the mouth and nose, accidentally or deliberately imposed as in an assault or homicide.
Spiral fracture	Usually of a long bone, a fracture which spirals around and through the shaft regarded as indicating a twisting mechanism to its causation. Whether the spiral nature of a fracture can be confidently asserted from radiological appearance alone is controversial.
Status thymo-lymphaticus	Also known as Status Lymphaticus, Status thymico-lymphaticus, or lymphatism. It was regarded as a disease resulting in sudden unexpected death, mostly in children or young people, and often following a trivial stress. It was seldom diagnosed while patients were alive, with sudden otherwise unexplained death being its main feature. It was first described in 1889 and had disappeared as a recognized condition by about 1960 [5].
Subarachnoid	Beneath the arachnoid mater, the membrane loosely adherent to the brain, and between it and the underlying brain. Subarachnoid hemorrhage is bleeding into this area.
Subcutaneous	Beneath the cutis, which is the epidermis and dermis together. The layer beneath the leathery dermis, which is often fat, hence, subcutaneous fat.
Subgaleal hematoma (cephalhematoma)	Hemorrhage between the actual bone of the skull and the immediately externally attached layer of fibrous tissue. When seen in a new born, referred to as a cephalhematoma and indicates the existence of a circulation, at least during delivery.
Suboxia	Low levels of oxygen in the blood (sometimes used in place of hypoxia).
Sudden Infant Death Syndrome	The sudden unexpected death of an infant under 12 months of age, with onset of the fatal episode apparently occurring during sleep, that remains unexplained after a thorough investigation, including performance of a complete autopsy and review of the circumstances of the death and clinical history [6].
Sudden Unexpected	A broader categorization of deaths in infancy that includes other unexplained

Death in Infancy	deaths than SIDS.
Subdural hemorrhage	Hemorrhage or bleeding into the space or potential space between the brain and the dura mater (which is found adherent to the inner aspect of the skull).
Suffocation	Deprivation of air; smothering; interruption of respiration; stifling; choking; throttling (manual strangulation).
Superior*	Anatomical term: above.
Systematic review	A review of a clearly formulated question that uses systematic and explicit methods to identify, select, and critically appraise relevant research, and to collect and analyse data from the studies that are included in the review. Statistical methods (meta-analysis) may or may not be used to analyse and summarise the results of the included studies [2].
Throttling	Manual strangulation; compression of the neck by hand/s.
Tardieu's spots	Petechial hemorrhages found on the outer surface of the lungs and epicardium and regarded by Tardieu as diagnostic of mechanical asphyxia, by which he meant mechanical means of interference with respiration.
Tentorial tearing	Tearing of the fibrous sheaths dividing the cranial cavity into compartments occupied by different parts of the brain.
Thrombus	A solid formation from the constituents of blood in the circulation of a living person. If it dislodges from the point of its formation and lodges down stream it is referred to as a thrombo-embolus or, simply, an embolus or embolism.
Thymus	An organ of the immune system, situated in the front of the upper chest extending into the base of the neck, which involutes or shrinks during childhood. Its apparent large size during infancy and early childhood, which is normal, was misinterpreted historically as being pathological.
Transverse fracture	A fracture in the horizontal plane of a bone considered in the anatomical position.
Traumatic asphyxia	A form of mechanical asphyxia involving compression of the chest and usually the abdomen such that it is physically not possible for the victim to move the chest and diaphragm to breathe. It can occur being trapped under a car for example, or crushed in a crowd.
Uncal herniation	Herniation , or protrusion, of the uncus, a part of the brain at its base and close to the top of the brain stem, through the tentorium cerebelli as a consequence of increased pressure in the cranium (raised intra-cranial pressure)
Venous outflow	Referring to the flow of blood in veins out of a part heading back to the lungs and heart.
Vital	Occurs in life.
Vitreous humour	Often simply referred to as vitreous, more technically, the vitreous body. The viscous, clear substance, or gel, forming the bulk of the contents of the globe or eye ball.

*Anatomical terms are used in relation to the body in the anatomical position. This is the body in a standing position, facing to the front, looking straight ahead with the palms turned to the front.

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Preface

We were asked to prepare an account of some of the major controversies in pediatric forensic pathology, an account that enabled these to be seen against the broader background of medicine and forensic pathology. What was sought was a paper of a technical kind, but accessible to a lay readership.

“Pediatric Forensic Pathology: Limits and Controversies” could well be the title of a textbook. For a start, many of the limits are those of forensic pathology itself. This is one reason to conceive the practice of pediatric forensic pathology as a dynamic collaboration between forensic pathology and pediatric pathology. Each subdiscipline of anatomical pathology will usually have significant contributions to make to every case.

Writing this paper in a way that is more accessible than a purely technical paper to a lay readership has been a particularly fascinating challenge. Such a task should be second nature for the discipline generally, as its conclusions must be well understood in court to be properly effective. One practical step has been to include a glossary. In trying to improve the accessibility of the material, we have almost certainly disappointed both lay readers and medical colleagues at different points along the way. We apologize in advance for the deficiencies.

This paper is not based on a detailed evaluation of actual cases that have been brought to the attention of the Inquiry. However, the authors are aware of some of the issues highlighted by these cases, and these have influenced some of what we have chosen to write about. However, there are limits we have not identified nor discussed. We have had to be selective.

There are four messages we hope you will keep in mind as you read this paper.

- The first, already mentioned, is that many of the limits of pediatric forensic pathology are limits of forensic pathology more generally.
- Second, the limits and controversies would have been described differently twenty years ago when the knowledge base of pediatric forensic pathology and the emphasis on

evidence-based conclusions was different. Today, with less credence attached to professional standing, there is tougher testing of evidence.

- Third, there is a group of pediatric forensic pathology entities that generate the most controversy and debate, some of which we have tried to describe. These include Shaken Baby Syndrome (SBS), whether fatal head injuries can be sustained from short falls, “asphyxia,” and determining whether a particular baby found dead around the time of birth was “born alive.”
- Fourth, the entire field is dependent on the work and application of knowledge by people operating in an emotionally charged area. Thus the findings and conclusions may be prone to biases that are well recognized whenever humans analyze and draw conclusions.

The forensic pathologist’s expectation is, or should be, that his or her opinions and conclusions would find support within a respected body of peers and may well be tested in a legal framework. S/he could also reasonably expect that his/her work environment is a collegial one that supports the maintenance of current knowledge and skills. Ultimately, however, forensic pathologists expect to be held individually responsible for the opinions they provide.

A discussion of the issues in a scientific or medical discipline is really inseparable from a discussion of its practice. The professional and organizational (i.e., system) issues related to the practice of pediatric forensic pathology are included in a companion paper: “A Model Forensic Pathology Service.” A consideration of both, together with other papers commissioned by the Inquiry, should help clarify what our society faces when investigating, managing, and ultimately preventing harm to children.

The authors would like to acknowledge the contribution of Professor Kent Roach whose questions and suggestions have improved this paper.

We hope the paper will provide some useful background for those interested in the Inquiry.

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Chapter 1—Pediatric Forensic Pathology: Some Background

Chapter Overview

Clinical medicine serves patients; forensic pathology serves the state to find out why its citizens die. Being involved in investigating possible crime is very different from treating patients. Medicine has developed quite a strong evidence base to its practice, and this has not been mirrored to the same extent in forensic pathology. The massive expansion in the size of the knowledge base of medicine has had implications for forensic pathology. Forensic pathology is a very small operational medical specialty; pediatric forensic pathology is a subset of cases within forensic pathology, and is not an operational specialty. Knowledge in forensic pathology evolves, not always in a uniform forward progression.

Introduction

Medicine exists to serve patients. Starting with doctors' training as medical students, everything revolves around the patient. Doctor's obligations to patients are central. This culture, imbued during medical training, survives intact through to the practice of virtually every branch of medicine, including all the disciplines within pathology, with the exception of forensic pathology. In forensic pathology there is no patient.

Inclined as doctors are to look for a patient, forensic pathologists sometimes see one in the family of the deceased. Certainly many forensic pathologists feel instinctively that they should provide, by one means or another, information to the family about what they have found. This might be to allay the family's usually unfounded feelings of guilt about the death, or to provide understanding generally about the death, which is important in itself and can affect the course of grieving. Increasingly forensic pathologists are discovering information during the course of the autopsy of a genetic kind that may be of direct medical significance to surviving members of the family.

However, important as these vestiges of medical obligation are, forensic pathologists have become involved at the behest of the state primarily to help look after the state's interest in understanding why its citizens have died. This assistance is provided through the medium of the law, a medium in which

doctors have little or no formal education and whose method of operation is quite different, even strange, to many of them.

The practice of medicine is geared toward diagnosis as a step along the way to treatment and cure or control. This is an entirely different paradigm to diagnosis and conclusions made as part of investigating possible crime, charging the accused, and having a trial. But, it has to be said, there has been relatively little done formally to build solid bridges between the two paradigms.

It is insufficiently appreciated that despite being the public face of pathology, forensic pathology is a very, very small discipline. Of the 2,500 fellows of the Royal College of Pathologists of Australasia, approximately 35 are full-time forensic pathologists, the smallest recognizable grouping within the College. None are full-time pediatric forensic pathologists; Australia, with a population of 20 million people, has no full-time pediatric forensic pathologist. The number of such full-time salaried positions around the world must be perishingly small. In the U.S.A., there are seven pathologists with American Board of Pathology certification in both forensic pathology and pediatric pathology [7]. We are not aware if any of those seven occupies a full-time salaried position in pediatric forensic pathology. We suspect not. This simply serves to introduce the thought that there is no separate, operational sub-subdiscipline of pediatric forensic pathology. There is a subset of forensic pathology cases that is properly regarded as pediatric forensic pathology, and that generates some academic literature of its own. This organizational aspect is of practical importance as one thinks of ways to support the medico-legal investigation of child deaths.

The Evolution of Medicine

The practice of medicine has evolved from a cottage industry into a massive, partly corporate, enterprise over the last century [8]. This occurred because of the social transformation of medicine and the development of new technology [9]. Table 1 summarizes the major factors.

Table 1—Major factors influencing the evolution of medicine

Major Factors	Notes
Changing nature of medical practice	<ul style="list-style-type: none">- No longer a cottage industry- Has acquired a complex organizational structure with political, professional, technical, administrative, and financial components
Cost of health care	<ul style="list-style-type: none">- Now a significant proportion of Gross Domestic Product- This results in increased accountability- Malpractice and adverse events, in addition to their individual consequences, contribute to cost, attracting scrutiny
Research and new technology	<ul style="list-style-type: none">- Development of information technology- Variation in medical practice between places and over time
Success of quality and safety improvement programs	<ul style="list-style-type: none">- Recognition of patient safety and quality of care as a major issue with new initiatives to address improvements

Starr [5] (1982) describes the transformation of American medicine, for example, occurring in two main phases (Table 2). These phases were the rise of professional sovereignty followed by the transformation of medicine into an industry with the growing role of corporations and the state.

Table 2—The transformation of medicine: A timeline based on Starr (1982)

Phase	Timeline	Period	Major Events
One	1850–1950	Consolidation of Professional Authority	<ul style="list-style-type: none">- Development of pathology and understanding of disease- Discovery of bacteria and viruses- Development of antiseptics, anaesthesia, surgery, and antibiotics
Two	1960	Health Reform	<ul style="list-style-type: none">- The reconstitution of the hospital- Antibiotic development accelerated- Vaccines developed- Prolongation of life span
	1970	“Health Crises” End of a Mandate	<ul style="list-style-type: none">- Increased access to medical care- Universal health insurance- The generalization of rights- The generalization of doubt
	1980	Coming of the Corporation	<ul style="list-style-type: none">- Competition between health providers- Growth of corporatized medicine

Until the 1970s the community had allowed health-care professionals and, in particular, doctors virtually complete autonomy and authority regarding the organization, structure, and provision of medicine. This faith was based on the advances in medical science during the first half of the 20th century.

By the 1970s, health-care provision was becoming increasingly complicated and the general community and the state were beginning to exert their influence. Further changes were also in train, including a general loss of confidence in society’s institutions [8]. The increasing cost of medicine focused attention on the institutions of health care. At the same time, the general community came to see health care as a right, not a privilege [8], with questions also being raised about the benefits of medicine and science. These attitudinal shifts were reflected by increased recourse to the law courts. These and other factors led to much more questioning of medicine, not least by doctors themselves, which in turn led to the search for answers through research.

[New Technology and Basing Medicine on Research Evidence](#)

The need to understand the health-care system led to the early adoption of new information technology systems to strengthen research into health service delivery [9, 10]. The use of computers in the 1960s allowed the large volume of information collected concerning health services to be centralized and analyzed [11]. The activity of individual health-care institutions [12] and whole geographic areas was thus able to be characterized and quantified [13].

Profound changes in medical science occurred in the later part of the 20th century, including the development of clinical trials and epidemiology, and an explosion in medical literature and new analytical techniques. The volume, range, and scope of research expanded as did research methods. Examples include the Cochrane Collaboration and meta-analyses.

Established in 1992, the International Cochrane Collaboration developed an organization to prepare, maintain, and disseminate systematic reviews of health care based on randomized controlled trials [14]. The vision of the Cochrane Collaboration is that

[h]ealthcare decision-making around the world will be informed by high-quality, timely research evidence ... and (the Collaboration) will play a pivotal role in the production and dissemination of this evidence across all areas of health care.

The major product of the Collaboration is the Cochrane Database of Systematic Reviews, which is published quarterly and serves as a guide to quality-of-care. Editorial teams oversee the preparation and maintenance of the reviews, as well as application of the rigorous quality standards for which Cochrane Reviews have become known [15]. The Cochrane Library is a collection of databases that contain high-quality, independent evidence to inform health-care decision making. Cochrane Reviews represent the highest level of evidence on which to base clinical treatment decisions.

Several years are required to complete a systematic review and continual updates are required. A group from the United Kingdom, with some funding from the Home Office for the purpose, is forming a

forensic medicine interest group for Cochrane, with clinical forensic medicine and forensic pathology sub-groups [16]. This funding presumably represents governmental understanding that it has an interest in strengthening the evidence basis of forensic medicine, the practice of which has caused public concern in that jurisdiction.

Forensic pathology has lagged behind in its research and the development necessary to build the evidence base for its practice because:

- it is a small discipline dominated by operational public service obligations, generally located outside the better resourced health-care sector;
- it is inadequately represented, if at all, in medical schools and there are few if any forensic medicine institutes with mandated research responsibilities. The strongest research-based branches of medicine are those represented in medical schools, or increasingly, in special medical research institutes. In Australia there is one academic department of forensic medicine; in Canada there is none.
- generally speaking, forensic pathology is not a priority for health-care research funding bodies;
- research skills are not a major part of the training for forensic pathologists, compounding the above;
- the nature of forensic pathology research requires study designs that are different to the typical randomized clinical trials;
- the small number of cases available for study limits the ability to address complex research questions that are important for applied practice. Ideally multi-centre collaboration would be required, otherwise much of the research becomes descriptive case series with expert commentary.

This means that forensic pathology (including pediatric forensic pathology) research is different from clinical medical research.

Impact of Medicine's Evolution on the Limits of Pediatric Forensic Pathology

Changes in medicine have had major implications for pathology. These include the rapid rise of specialties and subspecialties in medicine, increasing its fragmentation into ever smaller craft groups or disciplines.

Historically, a medical doctor was considered, almost, medically omniscient and surgically omnipotent; both a physician and a surgeon. This has all but disappeared in developed countries, and medicine (in Australia) now has 18 distinct, recognized professional craft groups, specialties, or disciplines. Within each discipline many subdisciplines may be recognized, and within each subdiscipline, sub-subdisciplines are being developed.

Appendix 1 highlights these narrowing subdivisions, a measure of the increasing trend to specialization in medicine. The implication for forensic pathology is that the field of knowledge, with which a forensic pathologist might be expected to be familiar, is expanding rapidly. It is clearly impossible for a forensic pathologist to command more than a tiny portion of it. This has major implications for practice, and is at the core of our view that there needs to be some dynamism at the interface between forensic pathology and the range of other disciplines and subdisciplines upon which it most frequently interacts and upon which it relies.

In the Australian context, forensic pathology has recently been identified as a separate subdiscipline within the Royal College of Pathologists of Australasia. It has its own Advisory Committee reporting to the Council of the College, and has its own group of examiners responsible for the examination of candidates undertaking the final College examinations in forensic pathology. Previously, forensic pathology was regarded as a subset of anatomical pathology, itself a subdiscipline within pathology. Pediatric pathology remains a subdiscipline of anatomical pathology. It has no formal craft group representation within the College structures. As mentioned previously, in the U.S.A., there are seven pathologists with American Board of Pathology certification in both forensic pathology and pediatric pathology [7].

It is interesting to note that clinical forensic medicine (also known as the work of police surgeons) is not mentioned in the list of specialist practices and colleges in Appendix 1. Many of the more systematic issues discussed in this paper about forensic pathology are also true for clinical forensic medicine, but compounded by the fact that it does not have a craft group or discipline home in the Australian, Canadian, or American context. In the U.K., The Royal College of Physicians has recently developed a Faculty of Forensic and Legal Medicine that is providing such a home, not only for clinical forensic medicine, but also others who provide opinions for various branches of the law.

[Steps in the Evolution of Pediatric Forensic Pathology](#)

The identification of a subset of forensic pathology cases as pediatric forensic pathology is much more recent than might be expected. An understanding of medical progress described above only partially explains this. A more thorough understanding of our social progress better explains why the field is so new.

It was just over 200 years ago that children were working under intolerable conditions in coal mines in developed countries. Even today many countries still depend on child labour in their economies. The International Labour Organisation report in 1998 states that

[c]hild labour is simply the single most important source of child exploitation and child abuse in the world today [17].

Children in some parts of the world are press ganged into military service. It was only in 1989 that the United Nations Convention on the Rights of the Child stated that the child has the right to a safe environment [18].

Many of the types and causes of death in children that are commonly recognized today, including some that are relevant to intentional harm, have only been characterized and accepted in the past 30 to 40

years. For example, the entity of child abuse itself is regarded as being first described in modern times by Caffey in 1946¹ [19].

Sudden Infant Death Syndrome, although identified in an ad hoc way previously, was formally described and defined in 1969. It is still evolving as an entity, and generates a substantial literature of its own. Articles are now appearing discussing its genetics [20]. The entity of Munchausen Syndrome by Proxy has recently entered a phase of re-evaluation.² This follows the turmoil surrounding the eminent physician Sir Roy Meadow who first described it in the late 1970s and continued over ensuing years to characterize it. The identification of reflex anal dilatation as a sign of child sexual abuse, and the over reliance upon the sign by clinicians in Cleveland, England, led to a Public Inquiry in 1987/88. Issues around Shaken Baby Syndrome, whether short falls can cause fatal head injury, and many others are not settled and it will be many years before there is a completely uniform approach to them.

Apparent lack of priority and urgency in areas of medicine is something that surprises some members of the public when particular issues begin to affect them. To understand this requires an appreciation of a further aspect of medical knowledge. Shaken Baby Syndrome, for example, was first recognized in 1974; yet the first Australian summit on the issue was in 2001. While the entity was discussed and debated at meetings of pathologists over the years, the organization of a summit represented a priority hitherto missing. This is an example of knowledge discovered from research and its relatively informal introduction into medical practice generally. In clinical medicine it is generally considered that it takes at least 10 or more years for original research come into routine practice [21].

Table 3 sets out some events in the evolution of pediatric forensic pathology.

¹ The entity was first described in forensic literature by Professor Auguste Ambroise Tardieu in 1860. He wrote of child abuse as follows: "... a horrendous problem that would unsettle the soul of a moral philosopher." He described "visible lesions to the brain, especially in very young infants submitted to such abuse. I have discovered effusions of blood on the surface of the brain (obviously subdural haemorrhage) manifestly the result of blows to the head...."

² We do not doubt the existence of the entity. We can appreciate the difficulty of establishing the diagnosis in particular cases to the degree required for criminal and family court purposes.

Table 3—Timeline: 1800s to 2007

Year	Entity or Observation
1800s	Burial clubs—parents insuring against the death of their newborn children, providing incentive for the murder of such children, meant that discussion of “infanticide” was a dominant theme in the forensic literature of these times [22].
1800–1900	“25% of all deaths recorded in the nineteenth century (in the UK) were of infants less than one year, and this is probably an underestimate [23].”
1850–70	Modern approach to autopsy: Carl Rokitansky (1804–78) in Vienna. Matthias Schleiden (1804–81) and Theodore Schwann (1810–82) first articulate the theory of the cell as the building block of all living organisms.
1856	Friedrich Ludwig Meissner in Germany reports probably the first case of long QT syndrome (LQTS). He describes a deaf girl who collapses and dies while being publicly admonished at school. When the parents are informed, they indicate that two brothers of the girls have already died suddenly after a violent fright or rage [24].
1858	Tardieu describes visceral petechial hemorrhages, especially of the lungs and heart as indicating mechanical asphyxia.
1860	Tardieu the first to write about fatal child abuse.
1870–1900	Rudolf Virchow (1821–1902) first to state that all cells derive from previously existing cells and that therefore pathological reactions are the reactions of cells.
1873	1392 foundlings left at the New York Foundling Asylum; 122 infants found dead in New York alley ways, rivers, and elsewhere; 30 organizations in the City of New York concerned with children in need of help [25].
1880	Commonest cause of death at the Birmingham Coroners Court (U.K.) was “ex visitatione divina”—visitation by God.
1890–1940	Status thymo-lymphaticus in vogue as a cause of death. At one stage, operations to remove the (actually normal) thymus were undertaken.
1902	“Let us now consider the question of who should make the medico-legal post mortem. Should it be any medical practitioner ... or should it be a person of recognized experience and capacity to perform the duty irrespectively of other considerations. It would be ludicrous if it were not such a serious matter to reflect that in this advanced age and in an enlightened and humane country the law still permits any medical practitioner to be summoned to make a post mortem examination, without any regard to his knowledge, his previous experience or his capacity to fill the duty thus imposed on him. [26]” It is still the case in some parts of Australia in 2007 that non-pathologists (e.g. general practitioners) undertake some post-mortem examinations.
1910–1940	Sir Bernard Spilsbury (U.K.) regarded as almost infallible. Commits suicide in 1947.
1922	Infanticide Act (U.K.) introducing infanticide as a defence to murder for mothers of newborn infants. Amended in 1929 to extend the period to one year following the birth.
1946	Caffey describes classical child abuse—multiple injuries of different age occurring over a period of time with delay in seeking medical attention and histories that did not match the injuries [27].
1950	General signs of “asphyxia” recognized as being signs of death from very many causes.
1956	Barratt introduced the term “cot death” (crib death) to describe unexpected infant deaths without obvious explanation [28].
1958	Lawful chastisement used (unsuccessfully) as a defence to homicide in Victoria, Australia.
1969	Sudden Infant Death Syndrome defined.
1974	Shaken Baby Syndrome described [29].
1977	Munchausen Syndrome by Proxy/Meadow’s Syndrome described (also called: Fabricated or

Year	Entity or Observation
	Induced Illness).
1988	Over reliance on reflex anal dilatation as a marker of child sexual abuse leads to a Public Inquiry into Cleveland, England [30].
1990s	Rapid developments in molecular biology and genomics.
1990s	Reduction in the incidence of SIDS begins in the developed world related in part to research evidence that babies lying on their front while sleeping were particularly prone to SIDS. This led to campaigns to get parents to lay their babies on their back when putting them to sleep.
1995–1996	The identification of three LQTS genes takes place within nine months, between March 1995 and January 1996 [24].
1997	Haemosiderin laden pulmonary macrophages as a possible indicator of a cause of death other than SIDS [31].
1998–2001	The Bristol Royal Infirmary Inquiry conducted into the management of the care of children receiving complex cardiac surgical services at the Bristol Royal Infirmary between 1984 and 1995 [32]. Final report revealed the retention of organs at autopsy without parental knowledge or consent. Pediatric pathology as a subdiscipline seriously affected in the U.K. and becomes very unpopular.
2004	Lord Goldsmith announces a review of 258 cot-death cases in U.K. where parents have been convicted of killing children under the age of two.
2007	Goudge Inquiry

[The Prominence of Child Abuse/Non Accidental Injury](#)

Reductions in other infant deaths enabled greater attention to be paid to those remaining. In the 1900s the majority of childhood deaths were due to natural causes, such as poor nutrition and infections. Now, in developed countries, while Sudden Infant Death Syndrome (SIDS) is still the major single cause of death after the neonatal period in the first year of life, its incidence has reduced quite dramatically since around 1990. Many of the remaining infant deaths are due to non-intentional trauma; child neglect and intentional harm are likewise proportionately more prominent. As well, there is a heightened social awareness of, or sensitivity to, intentional harm to children that was missing, broadly speaking, in the developed world until the second half of the 20th century.

Child Mortality and Pediatric Forensic Pathology Case Loads in Victoria

A better understanding of the pediatric forensic pathology caseload begins with an understanding of the major causes of neonatal,³ post-neonatal,⁴ infant,⁵ and child⁶ deaths [33]. The collection and analysis of this body of knowledge is a relatively recent phenomenon and is due to the development of injury epidemiology and prevention rather than forensic pathology. Death from injury is increasingly relevant as a cause of mortality with the improved survival rates from birth and reduction of early childhood diseases (e.g., infections).

To illustrate the issue, consider the situation in 2005 Victoria, Australia, with a population of 5,022,346 people of whom 32,606 died. Of these:

- 247 neonates died in the first month of their life;
- 82 post-neonatal/infants died between one and twelve months of their life;
- 43 children died aged one to four years and 34 children died aged five to nine years;
- 31 children died aged ten to fourteen years; and
- 57 children died fifteen to seventeen years.

Table 4 shows that in Victoria in 2005, of the 247 neonatal deaths, 3 (2.7%) resulted from causes other than natural. The natural cause category included causes such as congenital abnormality, extreme prematurity, and infection.

³ Liveborn infant who dies within 28 days of birth [of at least 20 weeks gestation or if gestation is unknown weighing at least 400g].

⁴ Death between 29 and 364 days.

⁵ Death occurring within one year of birth.

⁶ Death occurring after and including the first birthday and up to, but not including, the 18th birthday.

Table 4—Neonatal deaths in Victoria Australia in 2005 by cause and gestational age

Cause of Death	Gestational Age at Birth				Total n (%)
	20–27 Weeks n (%)	28–31 Weeks n (%)	32–36 Weeks n (%)	37+ Weeks n (%)	
Natural Cause	170 (100.0)	12 (100.0)	27 (96.4)	34 (91.9)	243 (98.3)
Other Cause	-	-	1 (3.6)	3 (8.1)	4 (1.6)
- <i>SIDS</i>	-	-	-	1 (2.7)	1 (0.4)
- <i>Trauma</i>	-	-	-	1 (2.7)	1 (0.4)
- <i>Other</i>	-	-	1 (3.6)	1 (2.7)	1 (0.4)
- <i>Unknown</i>	-	-	-	-	-
Total	170 (100.0)	12 (100.0)	28 (100.0)	37 (100.0)	247 (100.0)

Source: The Consultative Council on Obstetric and Pediatric Mortality and Morbidity, Annual Report 2005.

a – Congenital abnormality includes terminations ≥ 20 weeks (45 neonatal deaths)

b – Extreme prematurity includes terminations ≥ 20 weeks for psychosocial indications (2 neonatal deaths)

Table 5 shows that of the 247⁷ deaths of children aged 29 days to 18 years in Victoria in 2005, 47 (19%) died from unintentional injury and 7 (2.8%) from intentional trauma.

Table 5—Causes of postneonatal infant and child deaths by age group, Victoria, Australia, 2005

Category	Age Group				Total
	29–364 Days	1–4 Years	5–9 Years	10–17 Years	
Determined at birth	49	14	6	21	90
Sudden Infant Death Syndrome^a	15	-	-	-	15
Acquired disease	12	15	17	22	66
Unintentional injuries	4	11	8	24	47
<i>Motor vehicle</i>	-	6	4	16	26
<i>Drowning</i>	2	1	2	2	7
<i>Asphyxiation</i>	2	-	-	-	2
<i>Other</i>	-	4	2	6	12
Intentional injury	2	1	1	19	23
<i>Intentional trauma</i>	2	1	1	3	7
<i>Suicide</i>	-	-	-	16	16
Undetermined^b	-	2	2	2	6
Total	82	43	34	88	247

Source: The Consultative Council on Obstetric and Pediatric Mortality and Morbidity, Annual Report 2005.

a – There was an infant whose death was coded as “unclassified sudden infant death” because no autopsy was performed. There were two neonatal infants (<29 days of age) who were classified as SIDS Category II and are not included in this table.

⁷ Note that it is a coincidence that the number of neonatal deaths (n = 247) is the same as the infant and child deaths.

b – There were two child deaths whose cause of death was undetermined after autopsy, and four child deaths whose cause of death was coded undetermined due to incomplete information. These four deaths will be assigned codes when more information is available.

Clearly the number of pediatric cases that a forensic pathologist examines where the cause of death is due to intentional injury is quite small. To illustrate this issue, consider the situation at the Victorian Institute of Forensic Medicine, which serves the population of Victoria. Of the 32,606 people who died in 2005:

- approximately 5,000 were reported to the Victorian State Coroner's Office, 3,465 of whom were brought to the Victorian Institute of Forensic Medicine for post-mortem examination; (see Table 6);
- 105 were under the age of 18 years, 75 of whom were under the age of 5 years;
- 3 under the age of 5 years, and a further 4 between the age of 5 and 18 years were determined to be a result of interpersonal violence; and
- 14 of the 55 deaths under the age of 1 year were either unclassified or regarded as of unknown in terms of manner of death.

If the workload is shared evenly across the forensic pathologists, each pathologist would perform relatively few autopsies on a child and would be involved in many fewer cases of intentional injury.

While these numbers will vary from jurisdiction to jurisdiction, we suspect the underlying message is fairly consistent: few jurisdictions in the developed world would have caseloads that could sustain a pediatric forensic pathology workload as a distinct operation, even if it was desirable to do so.

Table 6—Number of medico-legal death investigations conducted at the Victorian Institute of Forensic Medicine 2005–06 (July 1–June 30)

Case Type	Age Group					Total
	< 1	1–4 Years	5–9 Years	10–17 Years	18+	
Natural	35	6	3	3	1890	1937
Intentional self-harm	0	0	0	2	430	432
Unintentional	4	5	8	8	469	494
Interpersonal violence	2	1	2	1	65	71
Unknown	7	6	2	2	457	474
Unclassified	7	0	0	1	47	55
Total	55	18	15	17	3360	3465

Chapter 2–Building the Evidence Base for Pediatric Forensic Pathology: A Review of Short Distance Falls

Introduction

It is an uncomfortable reality that in many instances child abuse may not be distinguished, on scientific grounds alone, from one or more accidents. Some assert that this literature is robust and thriving [34], others lament the poor quality of the science and the fact the major controversies are unresolved [35-37]. The advent of evidence-based medicine means new or established treatments are required to undergo systematic evaluation and scrutiny. When the same standards are applied to the knowledge base of child abuse the results are poor. Studies differ on the most basic definitions, and sample sizes are small and highly selective, introducing the potential for skewed results or results that are difficult to compare. In addition, the predominant study design in child abuse and pediatric forensic pathology research is the “retrospective record review” and “case series” which, at best, provide a weak link for establishing cause and effect [37].

The limitation of the science acquires a particular significance in the legal system. Families wrongly accused lose (at least) their child, while a child returned in error to an abusing family may lose its life [38]. It is imperative then that both courts and clinicians appreciate that the science behind the diagnosis of child abuse is imperfect [37], and that uncertainties should be shared with the court. The need is clear for a defined evidence base to answer the controversies in diagnosing child abuse.

Until recently, the extensive literature on child abuse and pediatric forensic pathology lacked rigorous review papers summarizing the findings of all studies on a subject to date. The work by the Welsh Child Protection Systematic Review Group (2005) [39] represents a leap forward in efforts to systematically review the literature on the subjects of bruising, fractures, injuries to the mouth, burns, and bites.

The authors here present their review of short distance falls as an example of a systematic approach to summarizing and interpreting the existing knowledge on a given subject. We also explore some of the methodological and practical issues with undertaking a systematic review.

Short Distance Falls

Background

The question is: in infants and children who have died of head injury, is a claim that the injury was sustained in a short distance fall a credible claim? The height from which children could fall to sustain a fatal or serious head injury is a question that lacks a single, easy answer [40], despite Helfer (1977) having raised it as long ago as 1977 [41]. Discussion of and research into the issue has gathered some pace since about 1990. Today it remains a matter of controversy.

The factors that may influence the severity of the head injury following a fall include [42]:

- the distance fallen;
- the nature of the surface onto which the child falls;
- forward or sideways protective reflexes;
- whether the fall is in some way “broken”;
- whether the child has been propelled or ejected;
- the mass of the body and the head;
- the proportion of total energy absorbed in deforming the skull, the brain, and the rest of the body;
- whether or not energy is dissipated in causing fractures; and
- whether the contact with the ground is concentrated to one point or onto a flat surface.

Review papers summarizing what is known on these subjects generate conflicting conclusions [40, 43, 44]. Experiments on monkeys and in some cases on deceased infants suggest that a short distance fall is capable of causing serious harm [45-48]. Simulation testing with life-size dummies and computer modelling also assert that serious injury is possible [49-53], although both these study types come with the caveat of not being able to replicate real-life circumstances [43]. Studies of hospital, community, and

household-based falls in infants have also failed to provide a conclusive answer. Large population studies of childhood injuries on the whole indicate the likelihood of severe head injury from falls is rare. This is counterpointed by anecdotal case reports that suggest it can happen. The difficulty for police, pathologists, lawyers, the courts, and carers lies in the fact that while the statistical evidence suggests that the potential for fatalities from falls are rare, they do occur. If the phenomenon occurs rarely, it can happen again, whatever the statistical weight against its frequency [54].

In an attempt to provide a comprehensive picture of the debate, the authors conducted a systematic review of the literature. This process aims to evaluate and interpret all available research evidence relevant to a particular question [55], in this case: In infants and children who have died of head injury, is a claim that the injury was sustained in a short fall a credible claim? Or perhaps more succinctly, “can short distance falls cause death in infants and children?”

Method

Relevant literature was identified using the computerized database MEDLINE. The criteria for the inclusion of studies were as follows:

1. They were written in English and published between 1977 and 2007 (the last search was carried out in September 2007);
2. The target population included infants and children <18 years old and results were reported for this group;
3. “Accidental falls” as a Medical Subject Heading (MeSH) term or “fall” or “falls” or “falling” as a text word combined with; and
4. “Craniocerebral trauma” as a MeSH term or “craniocerebral trauma” or “head injury” or “head injuries” as text words.

The search strategy was supplemented with a cited reference search of the study on falls in infants by Helfer (1977) [41], which prompted the forensic pathology community to begin examining this issue with greater scrutiny. Using two reviewers, we screened study abstracts for inclusion in our review. Research

articles that addressed the question of whether short distance falls could cause serious injury or death were then independently scored by the reviewers using a five-point scale that uses criteria for establishing cause and effect [56]:

- a. Study design;
- b. A clearly identified comparison group(s);
- c. One or more non-caretaker witnesses observed the fall;
- d. Height of the falls was clearly stated; and
- e. Means of ascertainment of injury clearly stated.

Where the two reviewers disagreed on scoring, they reached consensus by revisiting the study and scoring in pairs.

Results

A total of 1,055 references were reviewed for inclusion. Following review, a total of 30 studies were found that addressed the research question. These included 3 review articles (Table 7), 4 biomechanical simulations of trauma to the infant skull not included in the review articles (Table 8), and 20 studies based on community or hospital data (Table 9). The search strategy was deliberately broad and aimed to capture the sequelae of serious head trauma including death. Including “death” as a MeSH term or “death” or “cause of death” as text words limited the search to 241 references.

Review Studies

Three review studies spanned almost 15 years and provide a summary of the extensive literature on falls in infants. Their findings (presented in Table 7) reflect the nature of the ongoing debate. The two recent papers by Oehmichen and colleagues (2005) [44] and Goldsmith and Plunkett (2004) [43] combine the medical literature with findings from biomechanical modelling and experimentation. The earlier review by Reiber (1993) [40] places greater emphasis on studies from hospital, community, and household settings, although he also draws from the biomechanical literature to support his conclusions. These reviews demonstrate the inseparable relationship between the measurement of the forces from the impact of a fall

(biomechanics) and the observed injuries that result in infants and children (as contained in the medical literature), in seeking to answer the question of whether a short distance fall can cause serious injury or death. This interrelationship brings with it practical consequences in forensic pathology: relatively few forensic pathologists, or pediatricians, could independently evaluate the biomechanics literature.

- Findings

Oehmichen et al.'s review, published in 2005, is the most recent and aims to shed light on discriminating between the injury patterns caused by falls and those caused by abuse, shaking in particular. Drawing from 56 primary studies dating back to 1972, Oehmichen et al. (2005) present epidemiological, biomechanical, morphological findings and clinical data to build a case that falls are distinguishable from abuse. They assert that "as a basic principle, simple injuries are caused by simple mechanisms like falls, whereas life threatening injuries should be attributed to abuse until proven otherwise." In the same paragraph they concede that based on the literature falls from less than 1.5 metres can lead to severe brain injury, though no definition of severe is provided. In the final section Oehmichen et al. (2005) present their own checklist for distinguishing abuse from accident:

1. Lack of congruency between type, localization, and age or date of the lesion and reports of the caregiver;
2. Lesions of different ages and at different parts of the body in suspected assaults;
3. Age less than 12 months; and
4. General physical as well as mental signs or symptoms of an abuse syndrome.

Goldsmith and Plunkett's (2004) review on the biomechanical causes of traumatic brain injury in infants and children presents data from 106 citations and provides an in-depth discussion of the use and limitations of simulation modelling and experimental studies involving dummies. Starting with Newton's second law of motion, Goldsmith and Plunkett (2004) negotiate this complex field by presenting concise summaries of the literature on measuring impact due to falls, scaling and loading, deformation and failure characteristics of the human body, and the differences between an infant and adult skull. The final section

presents supporting evidence from the medical and forensic pathology literature for Goldsmith and Plunkett's (2004) argument that "it is not possible to differentiate a deliberate impact from an accidental fall under the same mechanical circumstances by biomechanical analysis since the mechanism and injury patterns will be identical."

Goldsmith and Plunkett (2004) assert that it is not usually possible based on pathology to determine if a given head injury is due to an accident or abuse and advise against the scaling of empirical data from biomechanical studies using adults for application to children and infants.

Rieber's (1993) paper begins with a group of three children under five years of age who suffered a fall of 10 feet or greater. His review cites 20 original studies and discusses 10 in detail, including their strengths and limitations. Based on his analysis, the literature can be classified into three groups, those which provide evidence that

1. Minor injuries result from minor falls;
2. Major injuries result from major falls; and
3. Major injuries result from minor falls.

While he states the literature is clearly conflicting he does highlight the areas of consensus where there is no contradiction in the primary studies, namely that the presence of a skull fracture alone is not evidence of abuse and that there is a lack of major non-head trauma in short distance falls.

Based on his case series and the literature, he states that short falls can result in fatality in the absence of skull fracture or significant head trauma, but adds that such events are an extreme rarity. Cases with extensive skull fracture and brain contusion or laceration, or major head injury combined with major body injury, render a short fall history questionable. In considering the cause of the injury, one has to take into account the mechanics of the fall, the age and condition of the child and the shape and consistency of the contact surface. Finally the fall mechanics and the injury pattern should be correlated.

- Limitations of Review Studies

Current practice in preparing a systematic review involves scoring the quality of the evidence presented in each paper using the basic science behind the study. None of the three review papers provided a systematic rating protocol of the literature from which they drew their conclusions. This lack is particularly disappointing from the more recent papers by Oehmichen et al. (2005) and Goldsmith and Plunkett (2004), although it should be noted that these reviews drew heavily from the biomechanical and simulation modelling literature for which the authors could not identify an accepted scoring system. Nevertheless both of these studies draw extensively from the medical literature and do not discuss the limitations of the primary studies upon which they base their conclusions. Reiber's (1993) review presents an attempt at rating the quality of the primary studies, which, considering it was published in 1993, was ahead of its time. The review studies have also failed to detail the search terms used for sourcing the literature, the number of reviewers reading the literature, and if reviewers were working independently. In addition, Oehmichen et al.'s (2005) approach appears to be somewhat circular. In trying to resolve a controversy, the checklist proposed for distinguishing abuse from accident contains elements of controversy. For example, reliance upon the ability to age injuries; reliance upon interpretation of the version of events given by the carer; an arbitrary cut-off age of 12 months as the age to which the checklist applies; and reliance upon general physical and mental signs or symptoms of an abuse syndrome. In addition, the last mentioned also falls into the difficulty of assuming that, even if it can be established that a child has been abused in the past, how does one assess the relevance of that—as tempting as it might be to assume so—to the circumstances of the fatal injury?

Table 7—Review studies of falls in infants

Study	Year	Country	Number of Citations	Conclusions
Oehmichen et al.	2005	Germany	56	<ul style="list-style-type: none"> - Simple injuries are caused by simple mechanisms; extreme violence is necessary to cause life-threatening injury. - If a parent or caregiver attributes a severe traumatic injury in a child to a household fall, the claim should be regarded as false, unless proven otherwise. - If a child is injured by a fall, the parent or caregiver will immediately seek medical care and express extreme anxiety regarding the fate of the child. - If the child is injured by abuse the perpetrator almost always waits to see if the child recovers spontaneously.
Goldsmith et al.	2004	U.S.A.	106	<ul style="list-style-type: none"> - It is not possible, based on medical signs and symptoms, to determine if a given head injury is due to accident or abuse. - Tolerance thresholds for infants, in particular those aged three months or less, should not be scaled from adult acceleration or deformation data. - The known mechanical properties of the infant skull permit construction of biofidelic dummies that can be subject to experimentation. It is not currently possible to construct a biofidelic infant neck because the mechanical properties of the infant neck are not known.
Reiber	1993	U.S.A.	20	<ul style="list-style-type: none"> - In assessing the cause of a head injury attributed to a fall, one has to consider the possible mechanics of the fall, the age and condition of the child, and the shape and consistency of the contact surface. Correlation of fall mechanics and injury pattern should also be considered. - Cases with extensive skull fracture and brain contusion or laceration, or with major head and body injury, render a short fall history highly questionable. Findings of retinal hemorrhage and diffuse axonal injury indicate accelerative injury. - While children on occasion suffer fatal injury from short falls, such events are an extreme rarity.

Simulation Studies

Simulation and biomechanical studies not included in the review papers by Oehmichen et al. (2005) or Goldsmith and Plunkett (2004) were included in this review.

- Findings

Using a child test dummy, Bertocci et al. (2003, 2004) [49, 50] demonstrated that surface type played a critical role in head acceleration (and possible subsequent injury). Head acceleration (or the change in velocity) was significantly higher in falls onto wood, linoleum, or padded carpet when compared with padded playground foam.

In Weber's (1984, 1985) series of experiments, he dropped deceased infants from "table height" or 82 centimetres onto a variety of surfaces and found that fractures were common [47, 48]. He concluded that falls from that height impacting the head directly will probably result in skull fracture in infants. In 1984 he dropped 15 deceased infants onto three different surfaces—stone, carpet, and foam-backed linoleum. In each case skull fractures were seen. In three cases the fractures crossed the sutures. In 1985 he dropped 35 deceased infants from the same height onto soft cushioning and found that fractures were less frequent but still occurred in one case on thick foam and in four out of twenty five when dropped onto folded blankets [48].

- Limitations

The use of dummies and cadavers in assessing real-life injuries is controversial. Measurement of the forces applied to the skull do not permit calculation of forces imposed on the brain, or how much force and for how long is required to produce various types and severity of injury [42].

Bertocci et al. (2003, 2004) concede that because biomechanical response data on children are scarce, child test dummies are less likely to behave like humans than their adult counterparts. Inaccurate scaling techniques from adult to child, based on geometry and mass, are often used in the development of smaller dummies, rendering them potentially less able to represent an actual child. Moreover, test dummies were originally designed for high-energy impact events, such as motor vehicle crashes, which

render their transferability to low energy settings, such as a short distance fall, questionable. The additional gauges placed in the dummy by Bertocci et al. (2003, 2004) to measure forces on the legs and pelvis had also not been previously validated for biofidelity [49]. Active muscle responses and protective reflexes that may be present in infants and children during a fall are also not accounted for in dummies or cadavers [43].

Table 8—Simulation studies of falls in infants

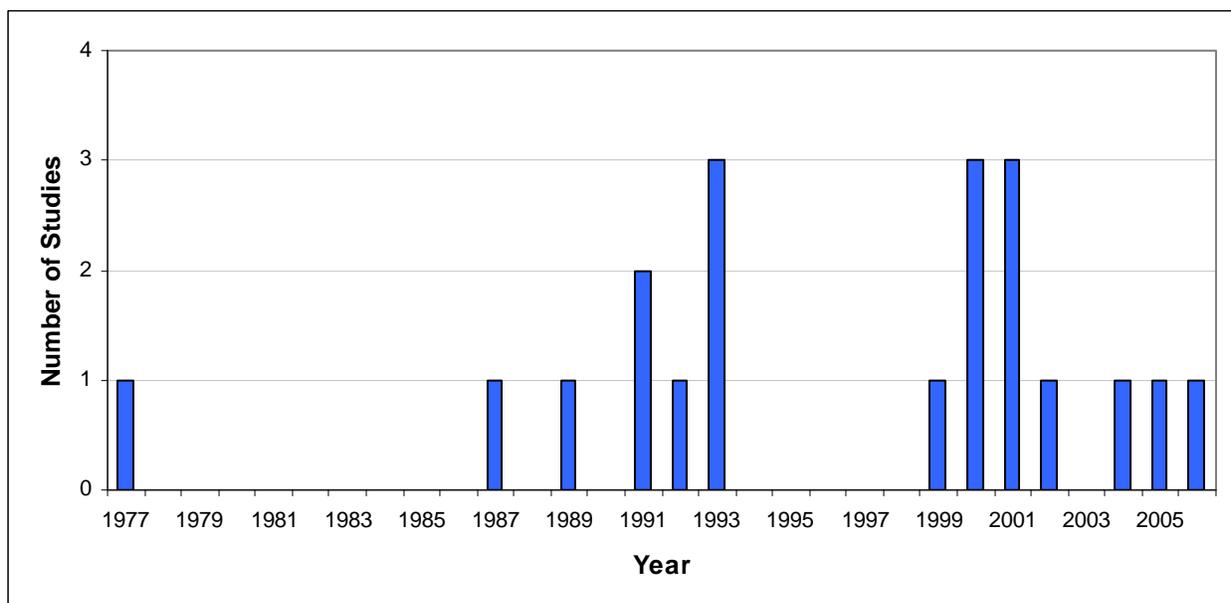
Study	Year	Country	Findings
Bertocci et al.	2004	U.S.A.	<ul style="list-style-type: none"> - Impact surface type and fall height were found to influence biomechanics associated with injury risk in feet-first free falls as assessed through experimental mock-ups using an instrumented child test dummy. - When comparing different types of impact surfaces in a 27 in. (0.69 m) fall, head acceleration associated with falls onto playground foam was significantly less than that associated with falls onto wood, linoleum, or padded carpet. - Feet-first falls from short distances (27 in.; 0.69 m) were associated with a low risk of contact-type head injury as assessed using HIC, irrespective of impact surface type.
Bertocci et al.	2003	U.S.A.	<ul style="list-style-type: none"> - Rolling falls from surfaces such as a bed or sofa from a side-lying posture onto different surfaces have a low risk of direct contact head injury in young children. - Impact surface type significantly affects head injury risk and lower extremity injury in side-lying posture rolling falls from 0.68 m (27 in.). - Detailed clinical histories are required when attempting to differentiate between abusive and non-abusive injuries.
Weber	1985	Germany	<ul style="list-style-type: none"> - Falls onto hard surface (stone, carpet, and foam-backed linoleum) resulted in parietal skull fractures in every case. - In falls onto a 2cm thick foam rubber mat, one fracture occurred. - In falls onto the double folded camel hair blanket, four fractures occurred. - Measurements along the fracture fissures showed bone thickness of 0.1–0.4 mm. The fracture injuries originated in paper-thin single-layer bone areas without diploe, which can also be considered the preferred regions for skull fractures of older infants following falls from low heights. These results indicate that it is not possible to assume that the skull of infants is not damaged after falls from table height.
Weber	1984	Germany	<ul style="list-style-type: none"> - Experimental falls from 82 cm onto three different surfaces—stone, carpet, and foam-backed linoleum—were carried out. In each case skull fractures were seen. In three cases the fractures crossed the sutures. - Each fall of an infant from the height of a table may cause a skull fracture, which may lead to death. - When abuse is suspected, all circumstances must be taken into consideration.

Primary Studies

- Findings

Primary studies are those that collect original data from subjects [57]. Of the 20 studies identified that fit the criteria for inclusion, 4 had the strongest study design of prospectively recruiting and reviewing infants and children [58-61]. Twelve studies reviewed medical and coronial records to identify participants who matched their inclusion criteria, of which 2 drew additional data from questionnaires [41, 62-72]. Two studies presented a series of cases where the event surrounding the injury was described in addition to the nature and extent of the injuries themselves [40, 73]. These were two population studies and they used different methods. The first was based on data from questionnaires completed by parents, asking them to tally the number of injuries their children sustained over a discrete period of time [74]. A second analyzed the injury characteristics of a population of 9,036 children and found that 2,658 had suffered falls [75]. Twelve studies found a predominance of male infants and children suffering falls and subsequent injuries [58-60, 62, 63, 65, 67-69, 72, 73, 75]. Research in this area is concentrated to the early nineties and the beginning of this century (Figure 1).

Figure 1—Published studies on falls in infants and children



Prospective Studies (n=4)

Prospective studies are recognized as the more robust study design because they enable data collection using consistent criteria and reduce potential biases compared to retrospective studies. These had the strongest study design. Johnson et al.'s (2005) prospective study of 72 children under five years of age with head injury found that in the vast majority of domestic accidents children do not suffer significant harm. Skull fractures are rare and probably occur in less than 5% of cases. To cause a skull fracture the fall needs to be from over 1 metre or, if from a lesser height, then a small-area impact point should be considered an integral component of the injury [60]. Four infants in the study sustained skull fractures, three after falling heights of 1 metre or less and one falling 3 metres. None of these children were readmitted to hospital and no child developed neurological symptoms following the fall.

Feldman's et al.'s (2001) study examined the cause of infant and toddler subdural hemorrhage (SDH) from intentional and non-intentional causes using a prospective design. Using a schema for determining the level of certainty in diagnosing abuse, they recruited 66 children, the injuries in 39 of whom were classified as abuse, in 12 were of undetermined cause, and in 3, the infants were witnessed as falling greater than 10 feet and sustaining subdural hemorrhages as a result. The three falls cases were clinically distinct from those who had sustained subdural hemorrhages from abuse. Children who had fallen only had acute subdural hemorrhages, while those that had been intentionally injured were more likely to have chronic or both acute and chronic subdural hemorrhage [59].

Duhaime and Christian's (1999) study of 100 hospitalized infants under two years of age included 73 who had sustained injuries from falls. For instances where the falls were not witnessed, the authors devised a classification for determining the likelihood of abuse. It was found that linear skull fractures were equally likely from falls of less than and greater than 4 feet. However, complex skull fractures were associated with the greater mechanical impact forces generated from higher falls. Of the four fatalities in the sample, one was classified as accidental and three were inflicted. The injury history of the accidental

death was not provided. They concluded that retinal hemorrhages were seen in serious accidental head injury but were more common in abuse [58].

Williams' (1991) study of injuries in infants and children less than three years of age from witnessed free falls included a sample of 106 individuals who had fallen between 3 and 70 feet. No life-threatening injuries occurred in the three patients who fell less than 10 feet. These three had small, depressed skull fractures without loss of consciousness from falling against an edged surface. Only one death occurred in this series, resulting from a fall of 70 feet. Based on the findings, he argued that infants and small children are relatively resistant to injuries from free falls, and falls of less than 10 feet are unlikely to produce serious or life-threatening injury [61].

Prospective studies corroborate each other on the point that fatal head injuries from short distance falls in infants are rare and, in general, infants and small children do not sustain life-threatening injuries from short distance falls.

[Retrospective Studies \(n=12\)](#)

The retrospective reviews provide conflicting viewpoints. Of the 12 studies reviewed using this design, 6 provide evidence to support the fact that short distance falls can be serious and in some cases fatal while the remaining 6 suggest short falls are not capable of inflicting lethal injury.

The study by Park et al. (2004) [68] of preschool children in Korea found that although significant intracranial injuries were more common in falls above 1 metre, significant intracranial injuries were also sustained in falls of less than 1 metre. Calvarial fractures were the most frequent type of head injury and were more common in the shorter falls. Powell et al.'s (2002) analysis of the National Electronic Injury Surveillance System data in the United States on injuries related to high chairs found that serious head injury from falls is extremely rare but can occur [70]. Kim et al.'s (2000) review of 729 pediatric head injuries found 22 patients who suffered intracranial hemorrhages following falls of less than 3 feet. There were four fatalities within the group, all showing abnormal findings on computerized tomography

scanning. He found that intracranial injury was the major source of fall-related death in children and was sustained with equal frequency in low and high-level falls [64].

Reece and Sege (2000) retrospectively reviewed the medical records of 287 children with head injuries aged between one week and 6 1/2 years, 62 of which were attributed to accidental falls of less than 4 feet [71]. Of these children 38 had simple linear skull fractures, 5 had complex skull fractures, 2 had brain contusions, 5 had subdural hemorrhages, and none had retinal hemorrhages. There was a single accidental fall fatality in the group, caused by injuries sustained in a fall from two stories onto a hard surface. Tarantino et al.'s (1999) study of infants aged 10 months or less who had suffered a short distance fall [72] found that the majority of patients (85%) had minor or no injury. However, significant injuries were sustained by 25 cases including 16 with a closed head injury (12 with skull fractures). The characteristic found to be independently associated with significant injury was being dropped by the caretaker. Hall et al.'s (1989) review of medical examiner's files identified 18 children who fell less than 3 feet and died from head injuries without any associated injury [63]. Two of these occurred while the children were in hospital care, leading Hall (1989) to conclude that short falls can be lethal.

The remaining six retrospective studies provide data to suggest fatal head injury from short distance falls does not occur. Pitone and Attia (2006) reviewed the records of 787 patients, which included 326 children aged less than four years [69]. Falls from chairs and beds were a common cause of injury with infants aged two years or less predominantly sustaining head injuries. None of these infants had intracranial hemorrhages or required neurosurgical intervention. They concluded that routine household falls generate little or insignificant injury. Falls from stairs and furniture are relatively low risk.

Mayr et al. (2000) described the pattern of injury in 281 children who had sustained injuries from falls off bunk beds [66]. They found that although 91 (41.7%) had sustained major injuries, including three polytrauma, seven skull fractures, and 44 cerebral concussions, follow-up examination did not reveal any behavioural changes, neurological deficits, or growing skull fractures. Lyons and Oates' (1993) review of bed falls [65], studying 207 children aged five years and younger who had fallen from bed during a hospital stay, found 31 cases of injury; 29 cases resulted in contusions and small lacerations, and

2 resulted in fractures (clavicle and skull). However, loss of consciousness was not reported in any cases. They concluded that falls from short heights do not typically produce clinically significant injuries.

Chadwick et al.'s (1991) medical record review of children whose mechanism of injury was recorded as a "fall" [62] found that seven deaths occurred in 100 children who fell 4 feet or less and one death occurred in 117 children who fell 10 feet to 45 feet. The 7 children who died in short falls all had other factors in their cases, which suggested false histories. Based on this finding, they argue that when children incur fatal injuries in falls of less than 4 feet, the history is incorrect. Nimityongskul and Anderson's (1987) [67] study of corroborated bed falls during hospital stays found that only one child was reported to have a skull fracture after falling between 0.3–0.9 metres. The authors concluded that severe head, neck, and extremity injuries are extremely rare when children fall from hospital beds, and child abuse should be suspected when a child is seen with severe injury from a reported "fall at home."

Helfer et al. (1977) [41] studied hospital incident reports over a six-year period where a fall was report in children aged five years and younger. They found a total of 85 children who had fallen approximately 0.9 metres. In 57 incidents there was no apparent injury, 17 had small "cuts", 20 had a bump or bruise, and 1 child sustained a skull fracture with no serious or apparent sequelae. Their study found a low incidence of fracture and no serious head injuries. The authors conclude that physicians should be suspicious of child abuse if they examine a child with a serious head injury when the cause is reported to be a fall from a bed or sofa.

[Case Series \(n=2\)](#)

Plunkett's (2001) case series [73] of the United States Consumer Product Safety Commission database for head injury associated with the use of playground equipment during January 1988 to June 1999 is considered among the more controversial studies in the field. He identified 18 fall-related head injury fatalities of which the youngest was 12 months and the oldest 13 years. The falls were from 0.6 to 3 metres (2–10 feet). A non-caretaker witnessed 12 of the 18, and 12 had a lucid interval. Four of the six children in whom fundoscopic examination was documented in the medical record had bilateral retinal hemorrhage. Plunkett (2001) concluded that an infant or child may suffer a fatal head injury from a fall of less than 3 metres (10 feet) and that the injury may be associated with a lucid interval and bilateral retinal hemorrhage [73]. Reiber (1993) [40] found falls from the top bunk and from a rocking chair resulted in death and SDH in two children. Notwithstanding, he argues that the overwhelming balance of evidence is that fatalities and serious injuries from short distance falls are rare in young children.

[Population Study \(n=2\)](#)

Warrington et al. (2001) sent postal questionnaires to parents of infants at six months of age and asked them to describe any accident since birth [74]. They found falls in young infants were common, but injuries were infrequent, predominantly trivial, and almost entirely confined to the head. Falls from beds and settees did not result in skull fractures. Rivara et al.'s (1993) study used hospital discharge data from Washington State for traumatic injury from 1989–90 [75]. From the 9,036 cases identified in patients below 19 years, 2,658 were due to falls and 680 occurred in children aged 4 years or younger. One child below the age of 10 died, but the authors did not specify the age or the cause of the injuries sustained.

Table 9—Primary studies of falls in infants, 1977–2006

Study	Year	Country	Sample Size	Age	Study Design	Comparative Group	Witnessed by Non Caretaker	Exact Height of Fall	Ascertainment of Injury Clearly Described
Pitone et al.	2006	U.S.A.	787	≤ 13 years	Retrospective record review	✓	×	Not exact	✓
Johnson et al.	2005	United Kingdom	72	< 5 years	Prospective	✓	✓	✓	✓
Park et al.	2004	Korea	68	≤ 6 years	Retrospective record review	×	×	✓	✓
Powell	2002	U.S.A.	40 650	≤ 3 years	Retrospective record review	×	×	×	✓
Plunkett	2001	U.S.A.	18	1–13 years	Case series	×	Not all	✓	✓
Mayr et al.	2000	Austria	1. 218 2. 991	< 12 years	1. Retrospective record review 2. Retrospective questionnaire	✓	×	×	✓
Feldman et al.	2001	U.S.A.	3 falls of 66 with SDH	≤ 3 years	Prospective	✓	✓	×	✓
Warrington	2001	U.S.A.	11 466	6 months	Retrospective questionnaire	✓	×	Not exact	×
Kim	2000	U.S.A.	729	< 15 years	Retrospective record review	×	×	Not exact	✓
Reece	2000	U.S.A.	287	< 6.5 years	Retrospective record review	✓	×	Not exact	✓
Tarantino et al.	1999	U.S.A.	167	≤ 10 months	Retrospective record review	×	×	✓	✓
Rivara et al.	1993	U.S.A.	2658	≤ 19 years	Population study	×	×	×	✓
Lyons et al.	1993	Australia	207	≤ 6 years	Retrospective record review	✓	✓	✓	✓
Reiber	1993	U.S.A.	3	≤ 5 years	Case Series	×	Not all	✓	✓
Duhaime et al.	1992	U.S.A.	73	≤ 24 months	Prospective	✓	Not all	Not exact	✓
Chadwick et al	1991	U.S.A.	317	0–13+	Retrospective record review	✓	×	✓	✓
Williams	1991	U.S.A.	106	< 3 years	Prospective	×	✓	✓	✓
Hall et al.	1989	U.S.A.	44	≤ 15 years	Retrospective record review	×	×	✓	✓
Nimityongskul et al.	1987	U.S.A.	76	≤ 16 years	Retrospective record review	×	×	✓	✓
Helfer et al.	1977	U.S.A.	1. 81 2. 161	≤ 5 years	1. Retrospective record review 2. Retrospective questionnaire	×	Falls occur in hospital	✓	✓

[Limitations](#)

The primary aim of each study varies from descriptive population studies designed to determine the frequency and nature of falls in children to the specific question of do short distance falls cause death. The study design is predominantly retrospective, using hospital or injury databases to access information. Case selection varies and primary inclusion criteria is “any trauma,” “falls only,” “head injury,” “head injury from falls,” “death,” “death from head injury,” and “death from head injury due to fall.” This creates a sampling bias and also limits the nature and type of information available about the circumstances of the fall, nature of injury, and ascertainment of the mechanism of injury (i.e., intentional or not).

The population studied is highly variable according to age group varying from six months of age through to including all children under the age of 18 years. The exact height of the fall is variable between studies and different cut-off points are used for short distance falls; ascertaining the height of the fall and the nature of the surface contact is not taken into consideration. The retrospective studies are further limited by the absence of witnesses to the falls in the majority of cases. The potential for a percentage of these injuries to be intentionally inflicted, hence skewing the findings, is real.

[Conclusion to Review](#)

This review required detailed analysis of the study design to consider whether the aim was specific to the question, whether data collection was prospective, and what if any comparison group was included. It was not logistically feasible to complete a systematic review according to Cochrane in the available time because of the availability of studies, resources, and access to primary data.

The review studies provided limited discussion of the weaknesses of the primary studies upon which they based their conclusions and failed to detail the search terms used for sourcing the literature and the number of reviewers.

The simulation studies used a test dummy that has not been validated for low energy settings. The experimental studies using dummies or cadavers did not take into account the active muscle response and protective reflexes that may be present in infants and children during a fall. Finally, measurement of the forces applied to the skull do not permit calculation of forces imposed on the brain, and do not provide an answer to how much force, for how long, is required to produce various types and severity of injury.

The remaining studies are limited by their retrospective design; their inability to ascertain or exclude intentional harm; their lack of specificity about the nature, height, and surface impact of the fall; and their failure to adjust or take into consideration other factors considered relevant to causing significant injury.

Therefore it is difficult if not impossible to address the question: “Do short distance falls cause fatal head injury in infants and children?”

Ideally a systematic review or meta-analysis would allow access to the primary data source to allow consistency in the include cases particularly around

- Age;
- Exact height of fall;
- Witnessed fall by a non-caretaker;
- Exclusion of intentional injury; and
- Impact surface.

Overall the issue of whether short distance falls cause significant head injury leading to death remains contentious. Large population studies of childhood injuries on the whole indicate the likelihood of severe head injury is rare. This is contrasted by the anecdotal individual case reports that suggest it does occur.

Reflection on our Experience

As described in Chapter 1, our experience confirms the need to build the evidence base for forensic pathology practice. In our review of the short falls literature, we did not identify a single systematic review on what is a highly contentious topic.

Our review of falls generated enormously diverse studies, the vast majority of which came from disciplines other than forensic pathology. The questions that are important for pediatric forensic pathology do not lend themselves to the typical randomized clinical trials.

For example, the evidence requires the consideration and integration of research that draws on diverse data sources ranging from routinely collected injury data sets, studies on cadavers, and biomechanical modelling.

Our review of falls was only possible because of a diverse range of skills available at our institute and among the project team. This included expertise in forensic pathology, public health and epidemiology, criminology, clinical practice, and systems-based approaches. We were not able to include an expert in biomechanics.

Notwithstanding this range of skills, conducting the review was challenging due to the time and resource limitations necessarily imposed on the research team for the purpose of preparing this document. Ideally researchers in this field would possess the majority of these skills, and other skills as well. Multi-centre collaborations and training may be required to assemble the necessary skills and resources.

The team took a more inclusive approach to the studies to illustrate the nature of a systematic review. If more rigorous inclusion criteria had been applied, very few of the studies would have remained.

The existing medical science approach to systematic reviews is designed to serve clinical practice, not forensic pathology in a courtroom setting. (This is but another example of the paradigm shift between clinical medicine and forensic pathology.) The question that is addressed

in a systematic review is critical. For example, a systematic review focusing on prevention of harm asks “Are short falls a significant cause of harm that merits prevention?” The answer is: short falls rarely cause harm and (probably) intervention strategies have not been implemented. A systematic review focusing on the medico-legal aspects is asking “Are short falls a cause of significant head injury and death?” Although the question appears only subtly different, the approach and conclusions have substantially different application in a court of law.

Our experience confirms our view that more work is needed to build the theoretical and applied evidence base for pediatric forensic pathology. What was particularly striking was Oechiden’s (2005) statement “that life threatening injuries should be attributed to abuse unless proven otherwise” compared with Goldsmith and Plunkett’s (2004) statement at “that it was not possible to differentiate an accidental fall from a deliberate impact”. Lawyers may find the first statement problematic because it suggests a mandatory presumption in the absence of other evidence that is at odds with the presumption of innocence - described as the golden thread through Anglo criminal law.

This highlights the different approaches taken when intentional harm is considered the issue in a clinical context as opposed to the courtroom setting. In a clinical context, a child who is still alive and suffered an injury the primary concern is to protect the child. Therefore, the most prudent approach is to assume the harm is intentional and act in a manner that ensures the safety and life of the child. The consequences of presuming that harm is not intentional is to place the child’s life at risk. Consider now a criminal court setting where the child is dead, the primary concern here is to ensure justice for the accused. The greatest potential for harm is a miscarriage of justice and therefore the most prudent approach is a presumption of innocence. This is diametrically opposed to the situation in the clinical setting. That is, the assumption in court is that harm is not intentional unless proven otherwise. The forensic pathologist is in the unusual situation of having to move their thinking between these two different settings.

Chapter 3—Controversies in Pediatric Forensic Pathology

Overview

Many of the controversies in pediatric forensic pathology are issues in forensic pathology more broadly.⁸ There are too many controversies to be set out here. Some we have not canvassed are set out in Appendix 5. Some we have set out here have been influenced by our reading of some of the cases in front of the Commission. For example, asphyxia was given as the cause of death in a number of cases.

The Problem of Artefact

Most people are not aware that many changes to the body before or after death can be misinterpreted as injuries or disease occurring before death. One of the important responsibilities for the forensic pathologist involved in investigating a death is to distinguish changes produced by the original injury or disease from those introduced by treatment, resuscitation, or post-mortem phenomena [76]. Collectively these changes are referred to as artefacts (or artifacts). Artefacts are artificial products [77]. In the context of an autopsy they are signs or findings that imitate pathology occurring in life. Artefacts can be due to interference with the body before (e.g., therapeutic measures), during (e.g., resuscitation attempts), or after (e.g., handling of the body) death. Artefacts can also be due to natural processes occurring after death (e.g. post-mortem lividity).

Familiarity with artefacts is part of the essential experience of a forensic pathologist. There is relatively little written on them in the major texts and too little has been done to explore them in research detail. The table below sets out some of the ways artefacts can arise and the traps awaiting the unwary, and on occasions, even the wary.

⁸ Byard and Krous (2004) have recently produced a short paper with almost the same title as this chapter.

Table 10—Artefacts: Their source and potential for confusion

Source of Artefact	Potential for Confusion	Comments
A. Interference with Body before Death		
i. Therapeutic interventions, e.g., surgery.	Injuries, e.g., scalp bruising following neurosurgery, mimicking injury prior to surgery.	Some interventions can obscure existing injuries—excision, ablation, or other interference with true injuries. This can be critically important in subdural hemorrhage, for example.
B. Interference with Body during the Process of Dying		
i. Resuscitation and Cardio-Pulmonary Resuscitation (CPR) (so-called heart massage by compressing the chest, perhaps in combination with mouth-to-mouth resuscitation). NB—Inexpert attempts at resuscitation are an important variation upon this theme.	- Rib or sternal fractures - Visceral trauma: lungs, heart, liver, pancreas, spleen - Retinal hemorrhages - Conjunctival petechiae - Possible injuries around the nose and on the inner aspect of the mouth	Distinguishing between resuscitation, sometimes inexpertly applied, related injury, and trauma prior to resuscitation may be a critical issue in some cases.
ii. Intubation—insertion of a tube into the air passage or trachea to assist breathing.	- Injuries around mouth, nose, inner aspect of mouth - Injuries to the neck, externally and internally	
iii. Intravenous access—insertion of a needle into a vein to allow the direct administration of fluids and drugs into the circulation.	- Intravenous access - Associated trauma; bruising	

Source of Artefact	Potential for Confusion	Comments
C. Post-Mortem Injury (i.e., injury after death). NB: Assailant may inflict injuries after death.		
i. Inadvertently during handling of the deceased, e.g., by ambulance, mortuary, or cemetery staff.	- Injury, e.g., fractures, lacerations, abrasions; ruptured biceps or other muscles	
ii. Embalming procedures	- Injuries - Chemical effects rendering toxicological analysis futile in some circumstances	
iii. Anthropophagy		Some can obscure existing ante-mortem injuries.
a) Terrestrial: Predation by animals, insects	- Injuries can mimic abrasions	
b) Airborne: Predation by airborne insects	- Small excoriations can mimic abrasions	
c) Aquatic: Predation by fish, sea lice, etc.	- Injuries	

Source of Artefact	Potential for Confusion	Comments
D. Effects of fire. NB: Some can obscure existing ante-mortem injuries.		
i. Direct effect of heat	- Injuries, e.g., extra-dural hemorrhage (heat hematoma); fractures; lacerations	
ii. Other extraneous effects: e.g., falling masonry during a fire	- Injuries	

Source of Artefact	Potential for Confusion	Comments
E. Post mortem changes, i.e., naturally occurring changes associated with the passage of time after death. NB: Some can obscure existing ante-mortem injuries.		
i. Hypostasis (post-mortem lividity) and pressure effects preventing its formation causing relative whitening or pallor	<ul style="list-style-type: none"> - Bruising - General signs of “mechanical asphyxia” in deceased whose head and neck are dependent (i.e., lower than the rest of the body), e.g., congestion, petechiae, conjunctival hemorrhage - Intestinal ischaemia/infarction - Apparent post-mortem bleeding from divided vessels in dependent positions - Pallor associated with the absence of lividity because of pressure in the post-mortem period interpreted as ante-mortem pressure (e.g., over the mouth and nose) (see for example Photo 15.34 in Dolinak, Matshes & Lew, 2005[78]) 	
ii. Effects of gastric fluid	<ul style="list-style-type: none"> - Tanning of the facial skin if in contact with gastric fluid for some time mimicking thermal or chemical injury - Gastric perforation 	
iii. Autolysis/Putrefaction (e.g., purple/black discolouration; purging of blood-stained fluid from the orifices; bloating; extrusion of the tongue; bulging eyes, etc.)	<ul style="list-style-type: none"> - Natural disease— e.g., pancreatitis - Altered appearance interfering with identification, e.g., mistaken corpulence - Skin slippage mistaken for thermal injury - Injuries—bruising, internal hemorrhage, “mechanical asphyxia” - Distension of anus, rectum, vagina, mouth associated with assault 	
iv. Post-mortem clot	<ul style="list-style-type: none"> - Ante-mortem thrombus 	

Source of Artefact	Potential for Confusion	Comments
F. Changes associated with the conduct of the autopsy.		
Some can obscure existing ante-mortem injuries.		
i. Reflecting scalp	- Apparent petechiae on galea and under surface of scalp	Regarded as simply a consequence of the scalp reflection.
ii. Associated with dissection	- Injury, e.g., fractures to larynx; bruising mimicking injury in the neck during dissection - Skull fracture associated with calvarium removal - Small subdural smears of blood - Pneumothorax - Air embolism	
iii. Scalpel marks in bone of skull	- Injury (in particular circumstances)	

Aspects of injuries, including their ageing

This is a particular example of a general forensic pathology issue, not specific to pediatric forensic pathology, but essential to it.

Does the shape and size of a bruise at autopsy represent the shape and size of the offending object?

A bruise is the result of injury causing damage to blood vessels resulting in bleeding into the adjacent tissues. Everybody is familiar with a bruise in the skin. A bruise in the skin is usually bleeding into the subcutaneous fat (the fat beneath the skin) and possibly also into the dermis (the leathery part of the skin). What is visible to the naked eye externally is generally less than what is in fact present because of the blanketing effect of the overlying skin. For example, the scalp (which is particularly thick compared to most skin) may completely block the appearance of widespread deep scalp or subgaleal bruising. This can obviously be of major forensic significance in clinical situations where conclusions that there is no scalp bruising might be incorrectly made. This simple fact may even contribute to significant differences in the approach of clinicians and pathologists to issues around Shaken Baby Syndrome because clinicians may “experience” less evidence of scalp injury in association with subdural hemorrhage than pathologists.

Since bruises represent bleeding from damaged vessels extending into adjacent tissues, the bruises will increase in size for as long as the bleeding continues. It is common experience to see a bruise apparently enlarge over the first 24 or so hours of its life. This does not necessarily mean the bruise is in fact enlarging. It may represent movement of the blood under the influence of gravity for example. If the surrounding tissues are more lax rather than compact, such as in the tissues around the eye (feel your own eyelids), the bruising will spread further than in, for example, the tough thick skin and tissues of the palm. Furthermore, relatively minor additional trauma can cause damage to the early healing of the damaged blood vessels causing additional bleeding, again resulting in enlargement. (Problems with clotting or other bleeding disorders can prolong this additional bleeding or render the individual susceptible to bruising from more trivial injuries than would cause bruising ordinarily.) The bleeding into the adjacent tissues is subject, over time, to the laws of gravity, so that the bruise can change position and shape. This means that the superficial site at which a bruise is seen may not even represent the site of the injury. The colour of the skin will affect the visibility of bruises in and beneath the skin. The coexistence of natural chronic illness, or the ingestion of therapeutic drugs such as steroids, may alter the time course and size of a bruise. A bruise may appear very quickly following the infliction of an injury, or it may take hours or even more than a day for deeper bruising to become visible from the surface. Deeper bruising can occur without more superficial bruising necessarily being caused. Sometimes, small superficial bruising may never be visible from the exterior (especially, for example, in the scalp), and larger bruises in deeper planes may never become visible from the exterior.⁹

These phenomena mean that making precise conclusions about the size and shape of offending instruments based on the size and shape of bruise may be fallacious, especially if there is no abrasion or laceration to precisely demarcate points of contact of the instrument with the

⁹ The assertions in this paragraph are not referenced, have not been subject to serious research, but are

skin. What will tend to be more persuasive will be specific peculiarities of shape that may be evident in the bruise (for example, the “handprint” on the buttocks in one of the case studies—see Chapter 5, Case Study 3), or specific representations of shape and size as demonstrated by abrasion and/or laceration or, on occasions, the size and shape of a burn. Impact in bone with fracturing that reflects the size and shape of the offending instrument, especially in more or less flat bone such as the skull, may result in closer correlation between the injury and the instrument.

Can a forensic pathologist tell with accuracy how much time has passed between the infliction of an injury and when the injury was examined clinically, or at autopsy? In other words, can injuries be aged?

Like time of death, the aging of injuries is one of those things the public think doctors generally, and forensic pathologists in particular, can do.

In relation to bruises, the more recent authors in forensic medicine agree on the caution that should be exercised in aging them. For example: *“In the present author’s experience it is impossible to comment on the age of a bruise less than 24 hours since infliction, except to say that it is ‘fresh’ as no visible colour changes occur in that time. It is not practicable to construct an accurate calendar of these colour changes as was done in older text books as there are too many variables for this to be reliable [79].”* Langlois and Gresham (1991), in an important paper entitled “The Ageing of Bruises: A Review and Study of the Colour Changes over Time,” have summarized different authors’ opinions on the time sequence of colour changes in bruises. The summary was presented by Langlois and Gresham (1991) to show the level of disagreement amongst the authors and to contrast with the final and only conclusion of their research: a bruise with identifiable yellowing is more than 18 hours old [80].

Table 11—Opinions on the time sequence of colour changes in bruises (Langlois and Gresham, 1991)

Source	Colour(s) in Bruise	Time
Camps and Cameron (1971) [81]	Red	Immediate

accepted experience-based comments about bruising.

	Dusky purple	Soon after
	Green	Days 4–5
	Yellow	Days 7–10
	Resolution	Days 14–15
Glaister (1973) [82]	Violet	Immediate
	Blue	Day 3
	Green	Days 5–7
	Yellow	Days 8–10
	Resolution	Days 13–18
Polson and Gee (1973) [83]	Red/dark red/black	< 24 hours
	Greenish tinge	Around day 7
	Yellowing	Around day 14
	Resolution	Up to 30 days
Smith and Fiddes (1955) [84]	Red	Immediate
	Purple/black	Soon after
	Green	Days 4–5
	Yellow	Days 7–10, but if small and superficial, day 3
	Resolution	Days 14–15

While there is undoubtedly an ability to establish that some injuries were sustained at different times (for example, a clearly fresh fractured rib with hemorrhage and no signs of new bone formation and a healing fracture with no hemorrhage and new bone formation; a fresh bleeding and bruised laceration, and a healing laceration with early scarring), there are a number of issues in the scientific aging of injuries that mean that the ability to conclude fine distinctions of aging is limited.

- Technical limitations: sampling: a histological section is about seven microns thick, so one histological section represents a tiny fraction of the whole injury, which possibly misrepresents the totality of the injury. Can the pathologist be sure that s/he has the oldest part of the injury represented in the section(s) taken?
- Pathological limitations: Super-added infection complicates assessment by delaying healing; other conditions will delay healing; even the seriousness of the injury itself may delay normal healing.

- Are the rates of healing the same between infants/children and all adults; much of the research involving humans, of which there is relatively little, seems to have involved adults.
- Different tissues heal at different rates and even with different processes (e.g., skin, nervous tissue, bone).
- Individual variation in biological responses.
- Lack of reliable studies and databases.
- As the injury ages, the age ranges become wider and more blurred (even at the early stages, the limitations are severe in terms of the discriminations sought).
- Histochemical investigations of injuries have not become routine.
- In a particular case, the importance of the age of a particular injury may not become apparent for several weeks or months following the autopsy, as it may be only then that the facts allow for a clear question of aging of one or more injuries to emerge.

It is for these, and perhaps other, reasons that the forensic pathologists' conclusions about the time that has elapsed between occurrence of an injury and death will normally be concluded in generalities. Most often the discrimination will be less than desired to answer the questions related to alibis and the like in cases of non-accidental injury.

Sudden Infant Death Syndrome

- What is SIDS?
- Is SIDS regarded as a natural cause of death?
- Is SIDS a real cause of death?
- Does a conclusion that an infant died from SIDS mean that the infant was not smothered?

SIDS is an enigma. SIDS is a particular form of Sudden Unexplained Death in Infancy (SUDI). SIDS as a diagnosis can be translated as: no cause of death discovered in this baby, aged

less than one year who died during a period of sleep, despite a full autopsy with many special tests, and there is nothing in the medical history, or the surrounding circumstances of the death, including evaluation of the scene, which really gives us any clue as to why the death occurred [6]. This means that, strictly, SIDS is NOT a diagnosis of natural causes, although it is likely that in the preponderance of cases, death was due to natural causes. Because various forms of non-natural death may leave no signs, accidental or homicidal smothering, for example, these remain possibilities in some cases of SIDS, although there is nothing in the particular case upon which to base such a conclusion. For some pathologists, SIDS has probably acquired the status of a natural cause death.

As much as anything, the entity of SIDS demonstrates that we still have a way to go to understand why many babies die.

Table 12—San Diego definition of SIDS: Krous HF et al. Sudden Infant Death Syndrome and Unclassified Sudden Infant Deaths: A Definitional and Diagnostic Approach. Pediatrics; Jul 2004; 114,1:234–238.

	Clinical History (CH)	Circumstances of Death	Autopsy
SIDS general definition	Sudden and unexpected death Under 1 year of age Lethal episode associated with sleep Death unexplained by CH	Unexplained after review of the circumstances	Unexplained after complete autopsy
Stratified definition			
- Category IA SIDS	(1) Older than 21 days, under 9 months (2) Normal CH (3) Full term pregnancy (≥ 37 weeks) (4) Normal growth and development (5) No similar deaths in siblings/relatives	Scene investigator performed and gave no explanation No evidence for an accident	(1) No lethal pathological findings (2) No unexplained trauma, abuse, neglect or unintentional injury (3) No substantial thymic stress (4) Toxicology, microbiology, radiography, vitreous chemistry and metabolic; screening
- Category IB SIDS	(1–5) Criteria for category IA SIDS	Scene investigation was not undertaken	(1-4); (5) One or more of the following analyses were not performed: toxicology, microbiology, radiography, vitreous chemistry and metabolic; screening
- Category II SIDS	Differences to category I criteria: (6) age range (0–21 days, 270–365 days) (7) Neonatal/perinatal conditions that have resolved by the time of death (8) Similar deaths in siblings, near relatives	Mechanical asphyxia or suffocation by overlaying not determined with certainty	(1-5); (6) Abnormal growth and development not thought to have contributed to death (7) More marked inflammatory changes or abnormalities not sufficient to cause of death
USID (Unclassified sudden infant death)	Criteria for category I or II SIDS are not fulfilled	Alternative diagnoses of natural or unnatural death are equivocal	Autopsy has not been performed

Data was sought from the Australian National Coroners Information System (NCIS) on deceased children up to the age of one year (0 to 12 months inclusive) and who died from Sudden Infant Death Syndrome (SIDS) or unexplained causes.

Table 13—SIDS cause of death terminology by Australian state/territory (where deceased is coded as 12 months old or less)

Cause of Death	ACT n (%)	NSW N (%)	NT n (%)	QLD n (%)	SA n (%)	TAS n (%)	VIC n (%)	WA n (%)	Total n (%)
SIDS	6 (55)	200 (73)	13 (38)	109 (69)	2 (4)	24 (80)	109 (80)	22 (25)	485 (62)
Undetermined	3 (27)	27 (9.9)	19 (55)	43 (27)	25 (54)	1 (3)	2 (1)	7 (8)	127 (16)
Unascertained	2 (18)	17 (6)	0 (0.0)	1 (1)	0 (0.0)	3 (10)	26 (19)	54 (61)	103 (13)
Undetermined (SIDS)	0 (0.0)	14 (5)	2 (6)	2 (1)	18 (39)	0 (0.0)	0 (0.0)	0 (0.0)	36 (5)
Other	0 (0.0)	15 (5)	0 (0.0)	4 (3)	1 (2)	1 (3)	0 (0.0)	4 (5)	25 (3)
Unascertained (SIDS)	0 (0.0)	2 (1)	0 (0.0)	0 (0.0)	0 (0.0)	1 (3)	0 (0.0)	1 (1)	4 (1)
Total	11 (100)	275 (100)	34 (100)	159 (100)	46 (100)	30 (100)	137 (100)	88 (100)	780 (100)

Source: National Coroners Information System, 2007.

Major findings include:

- 780 infants died from unexplained causes (including SIDS) nationwide over a period of six and a half years.
- Different jurisdictions use different terminologies to describe the causes of these deaths. Forensic pathologists in the Northern Territory (NT) and South Australia (SA) tend to use the term “unexplained” more frequently, while those in Western Australia (WA) commonly refer to the cause as “unascertained.” These are probably synonyms. All other jurisdictions tend to refer to the cause of death being “SIDS” most frequently.
- The definition of SIDS involving an infant under one year of age seems to be reflected in practice, with only one case after 2004 being found to refer to the cause of death being “SIDS” in an infant over 12 months of age.

The data shows variation in use of diagnostic terminology between jurisdictions. This could be explained by different experience of infant deaths between the states (for example, complexities in indigenous deaths complicating cases in WA, SA, and NT, resulting in greater usage of undetermined or unascertained. These complexities include the increased incidence of infective pathology but of insufficient degree to be regarded as the primary cause of death. It could also be that in some states and territories there are more comprehensive investigations of the scene, the circumstances, and the medical history increasing the likelihood of the discovery of factors complicating the assessment of the death.¹⁰) But it is also possible that pathologists are dealing with similar cases differently. The extent to which this is occurring needs to be investigated to reduce variation in similar cases to a minimum. As things stand, a researcher would have to revisit the specific cases to ensure that comparisons were valid if s/he wished to compare true SIDS and related death rates between states in Australia. Furthermore, it is possible

¹⁰ Conversely, there may be no scene examination in some jurisdictions, precluding the conclusion of SIDS (Category 1).

that families in similar circumstances across the country are being dealt with differently as a result.

CASE SUMMARY

A 10 week-old-baby boy, who was born two weeks prematurely, was found cold and not breathing between the parents in the marital bed when the parents awoke in the morning. An ambulance was summoned while resuscitation was commenced, but the infant was dead on the arrival of the ambulance. The child's paternal grandfather had three siblings die in infancy. An autopsy was conducted on the third day following death. The infant was normally developed for age. There were no injuries at all seen. A nappy rash was present. There were no epicardial petechiae but there were pleural petechiae over both lungs. Histological examination was unremarkable. There was pulmonary congestion and edema with one focus of intra-alveolar hemorrhage. Toxicology was negative for common drugs and poisons. Four plain radiographs covering the whole body showed no fractures. There were no conjunctival petechial hemorrhages; no intra-oral bruising; the frenula were not injured. The thymus was pale pink and showed no petechial hemorrhages. The middle ears were clear, and the CSF was also clear. Samples (left lung, right lung, liver, CSF) sent for microbiological culture showed no signs of pathogens. Samples from blood and spleen grew no organisms. Amino acid screen was negative showing a typical post mortem pattern. Post-mortem vitreous biochemistry was within normal limits and toxicological analysis was negative.

In Victoria, Australia, the cause of death was given as 1(a): Sudden Infant Death Syndrome (Category 2) because at autopsy there were no findings seen that could account for death, the overall appearances were those of Sudden Infant Death Syndrome (SIDS). The circumstances of having died co-sleeping with the parents rule out Category 1.

Consider now, if the position of the infant when discovered dead was with the mother's leg over the baby. There was no suggestion of parental alcohol and drug intoxication. The additional

autopsy findings now include fixed lividity over the front of the body with blanching over the mouth, nose and right side of the chin on external examination and microscopic examination of the lungs revealed moderate pulmonary edema and multi-focal intra-alveolar hemorrhage, with peribronchial and interstitial aggregates of chronic inflammatory cells.

This increases the possible options that pathologists might use for the cause of death, specifically:

- Category 2 SIDS;
- SIDS with overlaying;
- Overlaying;
- Smothering; and
- Undetermined.

This scenario was put to pathologists in a Quality Assurance Program (QAP) conducted by the College of American Pathologists [85]. The assessment of the pathologists is presented in Table 8, demonstrating the variation in their opinions.

It is interesting that 81.6% of the respondents classified the death as overlaying and an accidental death. The significance of the leg of one of the parents being over the infant depends to some extent on the details of this event. The choice of the cause of death will remain controversial since SIDS, (with subsequent positioning of the mothers leg over the infant) and smothering cannot be definitively ruled out; but neither can overlaying be definitely ruled in.

In the Australian context, the lividity and the leg being over the deceased might mean that fewer pathologists than otherwise would conclude this as SIDS Category 2 and would conclude 'Unascertained'. If we consider following the current international guidelines (see above), we would have expected that "overlaying" would feature somewhat less frequently than assessed by the pathologists in the QAP.

Table 14—The results of the CAP Quality Assurance Programme relative to the infant found dead in the parental bed with mother’s leg over the infant.

1. The immediate cause of death is	Number	Percentage
Overlaying	62	81.6
Pulmonary edema	5	6.6
SIDS	7	9.2
Viral pneumonia	2	2.6
2. The manner of death is		
Accident	61	81.3
Homicide	0	0
Natural	2	2.7
Undetermined	12	16.0

Key points:

1. Pathologists responding to this QAP vary in their conclusion as to the cause of death when provided with the same information. This variation requires explanation, and suggests improvements are required to produce more consistent outcomes;
2. Pathologists probably vary in their conclusions as to the cause of death on the same facts between countries. Agreed approaches will be needed to improve international consistency. The definitional work has been done to achieve this in relation to SIDS diagnosis, but considerable work is still required to develop consistency in the application of the definitions to particular situations.
3. Subtleties in interpretation of the cause and manner of death may not be visible to non-pathologists.

Cerebral Edema

Is cerebral edema an easy diagnosis in pediatric pathology? What are the causes and significance of cerebral edema?

Cerebral edema is one way in which the brain might increase in volume or swell. Brain volume depends upon:

- i) cerebral hydration—the water content of the brain.

- ii) intracranial blood volume—this can increase due to arterial hypertension; increased cerebral blood flow secondary to elevated cerebral perfusion pressure; decreased cerebrovascular resistance due to hypercapnia or hypoxia; obstruction to venous outflow.
- iii) Cerebrospinal fluid pressure, e.g., acute obstructive hydrocephalus.

So, if a pathologist believes that there may be brain swelling, s/he needs to ask: to what is this brain swelling due? Is it due to cerebral edema, or could it be due to congestion (another way of saying increased intracranial blood volume)? What inferences can properly follow if one or other of these conclusions, or a combination of both, is made?

“Brain mass and size are of little use in neuropathological evaluation of cerebral swelling due to their wide variability in children [86] [23].” The volume of the brain within its own cavity is what is important. So the “pouting of the brain tissue through the cervical incision is much more important than the mass of the brain ... the immature brain is thought to respond to severe mechanical load, infection or intoxication differently than the adult brain. Because the cranial vault of the infant is more pliable and elastic, the manifestation of increased intracranial pressure in the newborn differs from that in adults. In infants, the fontanelles and the open sutures permit distension or expansion of the cranium, thus weakening the force of downward pressure on the cerebellum and brain stem, reducing the likelihood of coning; secondary midbrain hemorrhages are therefore unknown in the newborn. Moreover, cerebellar herniation and acute tonsillar necrosis are hardly ever seen during the first year of life” [87]. The macroscopic manifestations are flattening of the gyri and narrowing of the intracerebral spaces, the ventricular system.

The commonest form of cerebral edema is vasogenic edema (or transfer of water from inside blood vessels to outside blood vessels and in brain tissue itself), which increases the extracellular fluid in the brain. This is another way of describing the breakdown in what is described as the blood brain barrier. This occurs as a response to trauma, tumours, infections, infarctions, hemorrhage, and toxins. It is thus a relatively non-specific response to a range of insults. As it

may take some time to occur (and how long this might be is probably quite variable depending on the severity of the insult and the duration of its application), reliance might be placed upon it in some circumstances to conclude that whatever caused a person's death took sufficient time to do so to allow cerebral edema to develop. In these circumstances, it would be vital to demonstrate that significant edema did indeed exist (and that what was observed was not simply brain swelling from congestion, possible associated simply with the fact of dying). Even if cerebral edema is demonstrated, heavy reliance upon circumstantial conclusions based upon its existence is not something often seen in the ordinary practice of forensic pathology.

It is tempting for pathologists to think that signs of cerebral swelling equate with edema. In many cases, this will be so. But when the observation is a critical one (such that the pathologist wishes to rely upon it in some way in the evaluation of the death), then it will be important to confirm that it is indeed edema. Histological assessment will be important in this regard. Such assessment may require neuropathological support for many forensic pathologists.

Asphyxia

What does the pathologist mean when s/he uses the term "asphyxia"?

It is difficult to discuss "asphyxia" coherently because it encompasses a number of concepts over time in forensic pathology, and it is used differently by current authors.

The confusion starts at the beginning, with its etymology and definition. Etymologically, the word has Greek roots: the prefix "a," meaning without; and the stem of the word "sphyx," meaning pulse. One definition of "asphyxia" is given as follows [77]:

1. stoppage of the pulse;
2. the condition of suspended animation produced by a deficiency of oxygen in the blood; suffocation.

Asphyxiate and asphyxiation are correspondingly defined.

Suffocation is defined in the same dictionary, by reference to the verb, as

1. deprivation of air;
2. smothering;
3. killing by stopping the supply of air through the lungs;
4. the interruption of respiration in a person;
5. stifling, choking;
6. throttling (the windpipe);
7. stifling (the breath).

It is important to appreciate some other words that overlap with asphyxia. These include hypoxia, suboxia, and anoxia. These simply mean a low level of oxygen in the blood or person (hypoxia, suboxia), or an absence of oxygen in the blood or person (anoxia).

The dictionary definition of suffocation resembles what most people probably think when they hear the word “asphyxia”: mechanical forms of interference with respiration or breathing. Numbers 1, 3, and 4 above are not necessarily mechanical in nature and encapsulate a general mode of dying by respiratory failure. The dictionary definition also includes some mechanisms that bring to mind homicide (smothering, throttling—synonymous with manual strangulation). Smothering of course can be accidental, for example, when an infant sleeps with one or both parents who accidentally obstruct the infant’s mouth and nose in one of many ways possible in such circumstances. (This is not the same as saying that all babies found dead after sleeping with their parents have been accidentally smothered. As we hope the reader will understand, forensic pathology usually cannot say.) Fatal choking can be accidental, for example, choking on a food bolus. Deprivation of air or stifling the breath can be natural, for example, asthma by constriction of the airways and/or their occlusion by tenacious mucus. On the ordinary dictionary test, “asphyxia” of itself is a relatively non-specific term as regards a particular mechanism interfering with breathing and, with the exception of throttling, non-specific as to the manner of its cause (i.e., natural, accidental, or homicidal). Already one can sense that for the word to be useful in a technical sense, it has to be explained and specified.

The table below shows something of how the word has been used in the technical forensic pathology literature. A further source of confusion among lay readers/consumers of forensic pathology is that “asphyxia” has been used by pathologists to describe the deprivation of oxygen at the level of cells and tissues, not just at the level of air entry into the body. On this basis, “asphyxia” has been regarded as a synonym of hypoxia or anoxia—a lowered level, or absence, of oxygen in the blood. There are innumerable causes of this, most of which are natural consequences of disease states.

Table 15—“Asphyxia”: What does it mean in the forensic pathology literature?

Literature	Meaning
Black & Black, 1946 [88]	Asphyxia is a mode or mechanism of death and is the equivalent of hypoxia or anoxia.
Bowden, 1949 [89]	Asphyxia is the same as hypoxia or anoxia and, from a forensic point of view, the interest is in the various forms of asphyxia “drowning, hanging, strangling, suffocation, and also asphyxia due to various poisonous gases, e.g. CO.*”
Gonzales, Vance, Helpem & Umberger, 1954 [90]	Asphyxia occurs when oxygen transfer from air into the blood in the lungs is interfered with. This includes mechanical forms of asphyxia, but also includes many natural conditions. Asphyxia also refers to anoxia.
Smith & Fiddes, 1955 [91]	Asphyxia is the same as anoxia or hypoxia. One should not attribute asphyxia to a violent cause unless the evidence of that cause is present.
Bowden, 1965 [92]	Asphyxia is the same as hypoxia or anoxia, and in forensic medicine we are mainly concerned with mechanical interference to the entry of air into the lungs.
Simpson, 1979 [93]	Asphyxia is the same as mechanical asphyxia.
Gordon & Shapiro, 1982 [94]	“The concept that asphyxia is a pathological entity which can be recognized by certain pathological changes, has led to considerable confusion in the literature on Forensic Medicine.” Asphyxia is not a distinct pathological entity. The word is used in a variety of ways. In forensic practice it is usually intended to convey mechanical interference with respiration.
Dolinak, Matshes & Lew, 2005 [78]	“Technically speaking, everyone dies of asphyxia. There comes a point, arising from either natural disease, injury, drug toxicity, or some combination thereof, at which blood flow to and from the brain, heart and other organs is insufficient, and terminal asphyxia is the end point of life. However, in the majority of these cases, the death is not attributed to asphyxia, but rather to the underlying condition leading to a cessation of respirations....”
Jaffe, 1999 [95]	“Asphyxia signifies a terminal state of oxygen lack and not the manner in which such a state was brought about.”

*CO is carbon monoxide

These definitional differences set the scene for the next complication in understanding “asphyxia” in forensic medicine: how the word should or should not be used when concluding the cause of death.

Can a pathologist properly use the term “asphyxia” as the cause of death?

Issues around the cause of death are set out separately in this paper. Suffice to say, for the purposes of this discussion, the internationally accepted standard format for setting out the cause of death comes from the World Health Organization (WHO) and shown below:

Table 16—WHO cause of death format

I	
Disease or condition directly leading to death.*	(a)
Antecedent causes (morbid conditions, if any, giving rise to the above cause, stating the underlying condition last).	(b)
II	
Other significant conditions (contributing to the death but not related to the disease or condition causing it).	(a)
	(b)

* This means the disease, injury, or complication that caused death NOT ONLY, for example, the mode of dying such as “heart failure, asthenia,” etc.

One needs to understand the form of the cause of death to appreciate what is required to complete the cause of death properly. The cause is divided into I and II. “I” is the direct cause of death or the disease or condition directly leading to death. “I” is subdivided, if necessary, into a and b (and theoretically c, d and e, etc., if necessary). “I(a)” is due to or a result of “I(b)” and so on. The last listed condition under “I” is the main or underlying cause of the patient’s death. “II” represents those other significant conditions contributing to the death but not directly related to the disease or condition causing it. In other words, conditions under “II” contribute directly (and by inference, independently) to death.

This is the internationally accepted form that tends to invite singular particular causes of death (which makes coding, classification, and statistics easier) and does not invite multiple interacting causes. There is an undefined distinction drawn between cause (Ia, b) and contribution

(IIa, b) that is essentially arbitrary, where the latter is some form of independent and lesser cause of death. (The inclusion of the word “significant” under “II” also adds further arbitrariness, emphasizing the role of opinion in concluding causes of death.)

Difficulties result when doctors simply put on the death certificate a mode of dying, rather than a cause. The common modes in times past were: heart failure, respiratory (lung) failure, or brain failure.¹¹ In old medical parlance, these modes of dying were referred to respectively as: syncope, asphyxia, and coma.¹² Definitions 1, 3, and 4 above represent the meaning of asphyxia when it is used in this way.

It can probably be appreciated that saying that someone has died of heart failure does not advance far one’s understanding of why that person has died. There are very many causes of, or diseases that might lead to, heart failure. (For example, was it coronary artery disease—or heart attack—in one or other of its manifestations; or was it due to aortic valve stenosis; aortic valve incompetence; myocarditis; cardiomyopathy; or constrictive pericarditis, to name some possibilities) In addition, each one of these conditions is directly the result of a further underlying condition. For example, aortic valve stenosis (narrowing of the aortic valve, meaning that the heart cannot easily pump blood around the body) might be due to congenital bicuspid valve (meaning that the patient was born with an abnormality which causes the later development of the narrowing). It is the underlying cause of the heart failure that is necessary to understand why the heart failure occurred and therefore why the person died. Clearly, before the advent of more modern understanding of pathology and medicine, the mode may have been all that was understood by those caring for the dying person, and therefore all that they could reasonably put

11 In contemporary practice, other modes include: acute or chronic renal failure; liver failure; multiple organ failure; coagulopathy; septic shock. These terms, not otherwise explained, are insufficient for the proper understanding of a death.

12 Syncope is a current technical medical term meaning a temporary loss of consciousness, or a faint, due to an often temporary disturbance of the heart’s function. Coma is a more permanent loss of consciousness involving, generally speaking, pathological processes in the brain. We have been discussing “asphyxia.”

on the certificate. Syncope and coma at least have the benefit of not being confused with anything more specific, unlike, as we have seen, with “asphyxia.”

When “asphyxia” appears alone on the death certificate or in the cause of death on an autopsy report, unless it is accompanied by a definition, the reader cannot know what is meant by it. Is it meant as a very general mode of death, perhaps equating with hypoxia or anoxia, which by itself is completely non-specific and meaningless as a cause of death?¹³ Or is it meant to equate with mechanical asphyxia? If so, there would then have to be reference to the specific form of mechanical asphyxia for the word to be in any way useful. The specific form having been specified, there is probably nothing to be gained by using the phrase “mechanical asphyxia.” The general signs of mechanical asphyxia, without signs to support conclusions of the specific form of mechanical asphyxia, can occur in many forms of death, and these general signs can also be mimicked by natural post-mortem changes.

Again, what have the learned authors had to say about this?

¹³ There would now be general agreement that even if clinically it was clear that the mode of dying was either heart, lung, or brain failure, the ability of a pathologist to distinguish between these modes is virtually nil, other than inferring it from the location of the primary pathology.

Table 17—Asphyxia as a proper term in the Cause of Death

Author	Authors' Interpretation
Black & Black, 1946 [88]	The general signs of asphyxia can occur in deaths from natural causes. Asphyxia is not really a cause of death unless the cause of the asphyxia is specified.
Rentoul & Smith, 1973 [96]	Asphyxia, along with coma and syncope, alone is not acceptable as a cause of death.
Gordon & Shapiro, 1982 [94]	The general signs of asphyxia, coma, or syncope cannot be distinguished post-mortem. Asphyxia, as a general phenomenon, is not a pathological entity or recognizable disease state. It therefore is not sensible to think that it could ever be a cause of death. Since the beginning of forensic pathology, there has been a tendency to over-interpret post-mortem findings, both findings of a general and a specific kind. "It is never justifiable to certify that a deceased person has died of asphyxia if this opinion is based only upon a finding of visceral congestion, petechial hemorrhages, cyanosis, cardiac dilatation and a condition of post mortem fluidity of the blood."
Joffe, 1999 [95]	"Asphyxia without qualification is not an acceptable diagnosis."

The answer to the question as posed is quite simple. Asphyxia, unqualified, is not meaningful if it purports to be the cause of death. If it is qualified, perhaps as "mechanical asphyxia," then it needs to be further specified as it is not possible to diagnose post-mortem a condition called simply and solely "mechanical asphyxia."

Does the concept of "mechanical asphyxia" play a useful role in forensic pathology?

"Mechanical asphyxia" is a convenient concept to collect together those entities where there is mechanical interference with the process of breathing. This is not to say that the deprivation of oxygen is the sole or even the main mechanism by which these entities cause death. Note that "traumatic asphyxia" and "positional asphyxia" are technical terms denoting, by popular forensic pathology usage, particular and specific types of "mechanical asphyxia."

Table 18 below sets out the different forms (or a classification) of mechanical asphyxia.

Table 18—A classification of mechanical forms of “asphyxia”

<p>1. External forms</p>	<p>1.1. Compression of the neck.</p> <p>1.2. Compression/obstruction of the mouth and nose.</p> <p>1.3. Compression of the chest (and abdomen).</p> <p>1.4. Combination of two or more of the above when infant gets into a dangerous position, e.g., between a mattress and a cot side; e.g., as with 1.2 and 1.3 above. Also occurs in adults, often in combination with alcohol intoxication.</p>	<p>1.1.1 Hanging (where the force on the ligature is the weight of the deceased’s own body, whether partially or wholly).</p> <p>1.1.2 Ligature strangulation (where the force on the ligature is applied by another person, or accidentally as in a curtain cord, or very rarely, suicidally).</p> <p>1.1.3 Manual strangulation (throttling).</p> <p>1.1.4 Law enforcement holds.</p> <p>1.1.5 Other (e.g., by arms, knees, feet).</p> <p>1.2.1 Smothering</p> <p>1.3.1 Traumatic asphyxia</p> <p>1.4.1 Positional Asphyxia</p>
<p>2. Internal</p>	<p>2.1. Obstruction of the larynx, laryngo-pharynx (back of the throat).</p> <p>2.2. Obstruction of the trachea (windpipe).</p> <p>2.3. Obstruction of small airways.</p> <p>2.4. Obstruction of alveoli.</p>	<p>2.1.1 Choking: e.g., by a food bolus (when it is called a “café coronary”), or material inserted into the mouth obstructing breathing.</p> <p>2.2.1 Compression of the windpipe (trachea) by tumour.</p> <p>2.3.1 Asthma: obstruction by constriction of the airways and/or mucus.</p> <p>2.4.1 Drowning: inhalation of fluid, usually water; blood from a tumour, tuberculosis, or facial fractures, for example. (Note that drowning is a more complicated entity than simply alveolar obstruction.)</p>

How do you diagnose the different forms of “mechanical asphyxia” at autopsy?

In relation to the various forms of mechanical asphyxia, some weight is attached in some circumstances to its general signs. These are:

1. Petechial hemorrhages: These may occur on the face, on the conjunctivae (the thin coverings of the eye and lining of the inner aspects of the eyelids); behind the ears; over the larynx at the back of the throat; on the inner aspect of the lips.
2. Facial congestion, especially if there is a demarcation above and below the level of compression (of the neck, for example).

Other signs have been referred to by numbers of authors, but suffer from confusion with a broader conception of asphyxia, which as we have seen is not sustainable. These other signs include: pleural and epicardial petechiae; cyanosis; fluidity of the blood post-mortem.

Sometimes these general signs are ascribed significance when seen in association with specific signs of, for example, compression of the neck by a ligature (e.g., the ligature mark itself encircling the neck, possible abrasions adjacent to the ligature mark suggesting attempts by the victim to loosen or remove it, possibly other signs of assault). The veins in the neck are compressed, preventing the flow of blood from the head back to the heart. Because, perhaps in the particular case, all the thicker walled arteries have not been compressed, blood flow continues into the head, and there is a buildup of blood dammed behind the constricting force around the neck. Added to developing hypoxia, terminal thinned walled capillaries and venules may rupture under this pressure, causing pin point hemorrhages in the laxer tissues of the face (e.g., around the eyes) and internally (e.g., around the larynx). For the same reason the head above the constricting force will be congested (that is, have a darkened colour compared to the rest of the body, and the darkening will not be confined to areas of lividity). Thus, what might be regarded as general and non-specific signs acquire some value in association with specific signs of, in this case, compression of the neck.

Particular care has to be taken to ensure that these general signs (petechiae, congestion) are not a post-mortem artefact associated with the head being in a dependent position post-mortem. If so, then the specific signs of the particular form of the mechanical asphyxia will probably need to be interpreted without the support of the general signs. Given the paucity of these in some cases, this may lead to serious investigative and prosecutorial problems. These may be aggravated occasionally by the presence of hemorrhages in the neck, possibly due either to lividity or to dissection artefact mimicking ante-mortem injury [94].

The current position is further buttressed by the overlap of many of the so-called general signs of asphyxia with, not only the processes of dying from almost any cause, but also post-mortem processes. These have been referred to in the section on artefact.

Have there been any more recent developments in relation to evaluating the possibility of smothering in infants?

A post-mortem diagnosis of homicidal smothering in an infant, based on autopsy findings alone, must be a rarity. Facial petechiae are rare (perhaps because significant respiratory effort is required to assist in their generation) and if broad, soft agents are used, usually no specific signs will be left. That is, there will be no facial or intra-oral bruises. An infant not being able to struggle effectively will not be able to resist, thereby reducing the necessary forces required for a fatal smothering compared to those required in a victim able to mount a defence. A 1997 paper entitled “Intra-Alveolar Pulmonary Siderophages in Sudden Infant Death: A Marker for Previous Imposed Suffocation” shows how important trying to find new pointers to these deaths has become [31].

Siderophages are cells containing iron, which is one of the breakdown products of blood or, more specifically, hemoglobin, the red pigmented oxygen-carrying substance in blood cells. Interest in the demonstration of siderophages in cases of sudden unexpected infant death started following the publication of the above paper. The authors concluded, amongst other things:

The identification of substantial numbers of intra-alveolar siderophages in the lungs of an infant dying suddenly establishes that there has been a major previous event causing intra-pulmonary hemorrhage. If further investigations do not reveal an accidental or natural cause, then we consider there are grounds for suspicion of previous episodes of imposed suffocation and of an unnatural cause of death. The finding is not specific for previous imposed suffocation and its absence does not exclude the possibility.

Thus the conclusion does not really substantiate the boldness of the paper's title. Subsequent studies have elucidated the situation somewhat [97].

Hanzlick and Delaney (2000) studied 59 infant deaths, prospectively submitting four sections of lung from each death for evaluation of haemosiderin. They scored the amount of haemosiderin as follows:

- No iron staining in the section = 0
- Occasional staining with iron with most fields negative = 1
- Focally abundant staining with most fields having no staining = 2
- Focally abundant staining with most fields showing positive staining = 3
- Prominent staining throughout the section = 4

The conclusion of the study was as follows:

The findings in this study suggest that the presence of hemosiderin in infant lungs, especially if focally abundant and present in many or most microscopic fields, should prompt special consideration that the cause of death may not be SIDS. The study also shows that an association of hemosiderin with higher than average pulmonary macrophage counts is a common finding.

A more recent paper is by Schluckebier et al. (2002) [98]. This was a retrospective study of infant deaths by two pathologists who scored the amount of hemosiderin as follows:

- < 5 siderophages per 20 high power fields = category 1
- 5–100 siderophages per 20 high power fields = category 2
- 100–500 siderophages per 20 high power fields = category 3A
- > 100 siderophages in a single lobe = category 3B
- > 500 siderophages per high power field = category 4

Forty-three cases were reviewed. The conclusion of the study was:

This study provides further evidence that unexplained pulmonary siderophages can be a marker for trauma or repeated hypoxia/asphyxia. Siderophages may also be increased for other reasons, but not to the same degree. Siderophages are not increased in SIDS or acute asphyxial deaths.... If siderophages are present in increased numbers without an obvious explanation, further investigation is warranted. [98]

The average (mean) score in the Hanzlick and Delaney (2000) study was six. Six cases had scores that were at least twice the mean. In five of these cases, death was caused by conditions other than SIDS, including one case in which asphyxia (sic) was given as the cause of death. (This was apparently a well-documented case of overlaying, meaning that as this may have occurred previously, this would be an explanation for previous pulmonary hemorrhage and therefore pulmonary siderophages.) Hanzlick and Delaney (2000) regarded their experience as supporting the view that significant amounts of pulmonary iron suggested a cause of death other than SIDS.

In the Schluckbier et al. (2002) study all the SIDS cases were category 1 cases. There were six cases in category 2 and four cases in category 3. These are listed below.

Table 19—SIDS categories, Schluckbier et al. (2002)

Category 2	Category 3
Complications of necrotizing enterocolitis	Anoxic encephalopathy*
Undetermined (massive cerebral edema)*	Complications of mechanical asphyxia*

Acute pneumonia	Probable asphyxia (manner undetermined)*
Complications of congenital heart disease (coarctation)	Undetermined
Undetermined (presence of bruises)	
Anoxic encephalopathy*	

* Note the imprecision of diagnostic terminology. These cases, based on these causes of death, are best regarded as “undetermined.”

Schluckbier et al’s (2002) conclusion is that “*if siderophages are present in increased numbers without an obvious explanation, further investigation is warranted.*”

What is the net effect of this work in contributing to the understanding of a particular infant death? We know that a significant percentage (66%) of SIDS infants have intra-alveolar hemorrhage [99]. However, it does seem that SIDS deaths are under-represented in the cases with obvious siderophages visible in the lungs. Where there is contention about the conclusion of smothering, the differential diagnosis will often be SIDS, or the various subtle natural diseases that are capable of causing sudden unexpected death in infants and form part of the differential diagnosis for SIDS itself. Even if SIDS is discounted, the number of unexplained deaths amongst the cases with obvious pulmonary siderophages makes it difficult to conclude that their presence is a marker for previous deliberately imposed airways obstruction. Their presence is perhaps better conceived of as a marker for further investigation. This provokes a philosophical question: Is there a distinction in levels of certainty between what is good enough to merit further investigation and what is good enough to present as a suspicious circumstance, or to present as a conclusion in court? This is a big question.

In a recent paper on the more general issue of pulmonary intra-alveolar hemorrhage, Krous et al (2007) concluded as follows: “*The most severe grade of pulmonary intra-alveolar hemorrhage (PH) occurred in 35% of deaths attributed to suffocation, but in only 9% of the SIDS cases. Age, duration of CPR attempts and post-mortem interval had no effect on the severity of PH in SIDS. Our results indicate that the severity of PH cannot be used independently to*

differentiate SIDS from suffocation death. Each case must be evaluated on its own merits after thorough review of the medical history, circumstances of death and post-mortem findings” [100].

Non Accidental Head Injury

Introduction

This is a particular area where there are special differences between infants and adults. *“The developing human brain presents a unique set of reactions to physical injury, as compared with the adult brain. There are many developmental and bio-mechanical differences that can explain age dependent variations in response [25].”*

During development, the central nervous system, and the brain in particular is closest to the eventual adult weight of any organ in the body except the eye. The brain represents 12-15% of body weight at birth, compared with 2.4% in the adult. The infant brain is 90% water (with a specific gravity of 1.03%) compared with the adult brain which is 76% water and has a specific gravity of 1.15%. The infant brain has a much lower protein content than the adult brain.

The scalp of the infant is much thinner, contains less fat and fewer large blood vessels. It is more elastic and flexible. It cannot dissipate the same amount of force as the adult scalp. Both infant and adult scalp can obscure serious underlying trauma. Underlying injuries are often more extensive than surface injury would lead one to suspect. This may be more marked in infants than adults. Until the age of two years approximately, the infant skull is more malleable and flexible compared with the more rigid, thicker ossified adult calvarium. The consequence of this is that any particular force will cause different injuries. Issues around non-accidental head injuries generally are comprehensively canvassed by Minns and Brown (2005) [101].

It is impossible to do justice to pediatric non-accidental head injury in a few pages. Like other parts of forensic pathology it needs formal systematic review. As an example of what is involved in such a process, Minns and Browns’ 500 page effort[101] was not regarded by one of its reviewers as a systematic review [102]. An additional issue is that most forensic pathologists

are not generally well versed in physics and biomechanics, let alone have the time and inclination to immerse themselves in these aspects of non-accidental head injury. The operational forensic pathologist therefore looks to academic leadership on the issue.

In the absence of a systematic review, much of the discussion around NAHI is characterized by general conclusions which can be difficult to apply to particular cases. For example Duhaime et al. [103] state as follows in relation to the forces required to produce subdural hemorrhage: “...*while controversy still exists as to the exact mechanism, most authors now agree that the forces necessary to cause this type of injury are far from trivial and in fact are considerable.....this sort of injury is **unlikely** (our emphasis) to be inflicted “accidentally” by well meaning caretakers who do not know that their behaviour can be injurious*”. This language is reminiscent of the language used in relation to the fatal potential of short falls. It is also language that stems in part from a clinical diagnostic paradigm, and not a medico-legal paradigm. The likelihood of accident may be slim, but accident needs to be considered in each case individually and sometimes, or even often, needs to be conceded as a possibility, even a reasonable possibility, which cannot be excluded.

We cannot present systematic reviews of the controversies. Our review of short falls is only a brief review, but even so, more comprehensive than we can attempt for other controversies. So it may be of use to consider some of the more general thoughts that inform/govern/influence the way some forensic pathologists approach the interpretive task, the job of coming to conclusions, in a particular case. What follows represents a brief view of an approach, about which others may well differ. The view relates to circumstances where the deceased has died before presentation to hospital, and therefore usually without time to obviously manifest encephalopathy, and without time to have undergone a whole range of investigations and interventions. (Many investigations that are possible clinically are not possible post mortem. For example, many of the tests for hemorrhagic tendencies are not applicable to post mortem

specimens.) This is followed by more specific and referenced commentary picking out some specific points.

Shaking

Even though we are discussing shaking, it necessarily overlaps with the next section, blunt head injury. Since the interpretive approach to be taken is often fact specific, let us consider the following:

A deceased female infant, aged three months, presents to the forensic pathology service as a sudden unexpected death in infancy. The parents objected to autopsy, but autopsy was required by the coroner. It reveals bilateral, obvious, clotted sub dural hemorrhage up to 3 millimetres thick involving the supero-lateral convexities. When the dura over the supero-lateral convexities is reflected, it is separate from the sub-dural hemorrhage and there are areas of yellowish staining apparent on the dura covering both convexities. The brain appears swollen with flattened gyri, but there are no herniation contusions. There is what appears to be prominent cerebellar tonsillar notching, but this attenuates after removal of the brain. The ventricular system appears compressed and smaller than normal. Histology confirms evidence of old sub-dural hemorrhage, appearing possibly of some weeks duration, with obvious haemosiderin (iron) staining. There are no retinal hemorrhages. There are no other injuries or pathology evident. In particular there is no scalp bruising nor neck injuries. When interviewed, the parents explain that the infant was taken from the marital bedroom by the 19 year old unemployed father at about 3 am, having been grizzling and not feeding for the previous two to three hours. The parents had been unable to sleep. When he brings the child back into the bed room the infant is unrousable and dead by the time the ambulance arrives. The father says he was cradling the infant, when he suddenly went limp. He then quickly took the infant into the mother and the ambulance was summoned. They did what CPR they could pending the arrival of the ambulance. The ambulance staff continued with resuscitation attempts, but the infant was dead on arrival at the hospital. The father admits,

during a video-taped interview with police, shaking the baby about a week or so before the death. When asked to demonstrate what he did, he demonstrates what can only be described as a very mild form of shaking.

Some of the matters in the forensic pathologist's mind as s/he approaches this or a similar case might include:

- Notwithstanding possible biomechanical arguments against it, the predominant view, currently, within forensic pathology is that it is **possible** to cause fatal head injury by shaking an infant without it being necessary to invoke the application of blunt trauma to the head, and without there being bruises let alone fractures on the chest or upper arms associated with the gripping of the infant¹⁴. The contrary view, that there **must** be blunt trauma in association with shaking to cause fatal head injury, is not widely held. Uscinski [132] is a neurosurgeon who is sceptical that shaking alone should be regarded as being capable of causing sub-dural hemorrhage.
- The conclusion (in a general sense, not in specific cases) that shaking can cause serious or fatal head injury is based on: the exclusion of other likely causes of the individual clinical observations (sub-dural hemorrhage; retinal hemorrhages; hypoxic ischaemic encephalopathy; possibly chest and/or upper limb bruises compatible with “grip” marks and possible metaphyseal fractures of limb bones); the plausibility of the link; and numbers of confessions. There is, as far as we are aware, no video or other covertly captured recording of a shaking causing a fatal head injury in an infant or child.
- It is possible that non-accidental head injury in infants as a result of shaking has been diagnosed more frequently clinically (as opposed to its rate of diagnosis at autopsy) because scalp bruising which may in fact be present, may not be apparent clinically. We

¹⁴ This is an assertion, and is made without any supporting quantitative evidence.

know that autopsy reveals clinically missed scalp and sub-galeal bruising [104].¹⁵ From a practical point of view, the co-existence of a recent scalp bruise and a recent sub-dural hemorrhage will (in the absence of fractured ribs and “grip” marks) by many if not most forensic pathologists result in a causal inference being drawn that whatever caused the bruise caused the sub-dural hemorrhage. The possibility of shaking can be raised, but the bruise indicates blunt trauma and blunt trauma is the commonest cause of sub-dural hemorrhage. Much of the “problem”, or relying completely upon shaking as the cause of the sub-dural hemorrhage, evaporates. (This leaves to one side the significance attaching to the presence of any retinal hemorrhages, which are generally regarded as being able to accompany blunt accidental head injury, albeit to a controversial degree¹⁶). In the particular case under discussion, however, there is no scalp bruise and there are no retinal hemorrhages. In this case the issues will include:

1. What caused the old sub-dural hemorrhage? Could it be the result of birth trauma?
2. Could the fresh sub-dural be a spontaneous consequence of the old sub-dural hemorrhage?
3. Does this infant have any hemorrhagic tendency rendering more likely a spontaneous re-bleed of an existing old sub-dural hemorrhage.
4. Is this a case of sub-dural hemorrhage consequent upon shaking?
5. If so, what force was required to cause the fresh sub-dural?

¹⁵ This disparity may be responsible for a different quality to the discussion clinically and pathologically about the role of shaking in non-accidental head injury. In relation to this, as an example of relatively weak inter-disciplinary activity within medicine itself, of the 24 contributors to Minns and Brown’s book, pathology is represented by one neuropathologist. No forensic pathologist is included. This is definitely not a criticism of the editors, but simply an observation for which there will be a number of perfectly reasonable explanations, including probably the lack of capacity within forensic pathology to respond to requests to be involved.

¹⁶ This controversy relates both to the incidence of retinal haemorrhages in accidental blunt head injury, and the severity of the accidental trauma required to produce retinal haemorrhages.

- Following from this, it seems that one of, and perhaps the critical issue in particular cases where it is concluded that shaking has occurred is as follows: was the shaking necessarily a “ violent” shaking? Violent in this context means assaultive, or that the person doing the shaking intended serious injury, or must have known or foreseen that serious injury was likely or probable; or if the person did not know, that a reasonable bystander would have known or foreseen this. We are not pretending legal exactitude here, but simply trying to encapsulate an issue. If it was a shaking, must it have been an assault? Just as there are no published accounts of shaking leading to sub-dural hemorrhage and retinal hemorrhages having been captured on film, so also there are no accounts of the kind posited by Caffey, in his original paper [101], of major injury occurring as the result of normal childhood handling, or vibration and noise stemming from domestic appliances.
- A further problem for the forensic pathologist is that many, such as Knight [105], proceed on the basis that a sub-dural hemorrhage might, in some circumstances, result from a blunt trauma that leaves no scalp bruising. This means that blunt trauma will often be there as a consideration, either in addition to or as an alternative to a proposed mechanism of shaking.

To now proceed a little more formally, Shaken Baby Syndrome is a label applied to a constellation of findings, most often in an infant younger than six months, in the brain, eye and possibly skin and skeleton [106]. It is characterized by encephalopathy, intracranial and intraocular hemorrhages often with no evident external trauma. Serious cases may result in death [107].

[Evolution of Shaken Baby Syndrome](#)

The term was coined by Caffey in 1974 [29] and has since been included in the international classification of diseases as a recognised external cause of death [108]. Several

pathologists described the features of Shaken Baby Syndrome prior to Caffey. In 1968 Weston, a forensic pathologist in Utah, reported on 21 children, aged one month to five years, with subdural hematoma. All but one had obvious external injuries such as bruising. Injuries resulted from gross violence in each case, such as beating with fists, sticks or other objects, and being thrown or swung against solid objects. In two cases there was mention of violent shaking, in combination with the head being banged against the floor or the cot sides [109].

In 1971 Guthkelch, a neurosurgeon in Hull (UK), pointed out that not all infants with subdural hematoma had external marks of injury on the head, and hypothesized that these infants had been shaken rather than struck [110].

In 1972 Caffey himself suggested that whiplash shaking was the explanation for cases of subdural hematoma in which there was no sign of external trauma to the scalp; in 1974 he coined the term 'whiplash shaken infant syndrome'. Caffey's hypothesis was that the whiplash shaking might be a component of violence and abuse but might also occur during normal childhood handling such as tossing a baby into the air, 'riding the horse' (baby bouncing on knee of parent), swinging a baby in a circle around the parent or 'skinning the cat' (the child is somersaulted forward while being held by the wrists), or even be caused by noise and vibration from dishwashers, vacuum cleaners and televisions. Belief that shaking is a form of trauma that causes subdural hematoma has led to the name Shaken Baby Syndrome [111].

In 2003 Geddes proposed the "unified hypothesis" following the publication of a pathological study of the dura of 50 intrauterine, neonatal or infant deaths where microscopic hemorrhage was identified in the layers of the dura in 36 cases [112]. The findings of the research suggested that the long held belief that infants who presented with encephalopathy, thin subdural hemorrhages and retinal hemorrhages (the triad indicating SBS) had been subjected to extreme and repeated violence was wrong and that little or no trauma was involved. From this it was hypothesized that subdural and retinal hemorrhage was not caused by traumatic shearing of subdural veins and retinal vessels but by a combination of cerebral hypoxia, raised intra-cranial

pressure from brain swelling, raised arterial pressure, and raised central venous pressure. The findings of the research were used regularly in court proceedings and appeals. In time, the English Court of Appeal (Criminal Division) found itself adjudicating on the scientific merits of the Geddes' Hypothesis and concluded: "*In our judgement, it follows that the unified hypothesis can no longer be regarded as a credible or alternative cause of the triad of injuries.*" [113]

Diagnosis

The term 'Shaken Baby Syndrome' describes a combination of subdural hematoma, encephalopathy (usually manifest in fatal cases by brain swelling) and retinal hemorrhage (commonly referred to as the triad). Of course, it could be applied when violent shaking has damaged the chest wall (e.g. bruising, rib fracture) without causing intracranial injury [109], but it seems to be accepted that the syndrome is essentially an inflicted head trauma.

It has been proposed [113] that injuries could be caused by a single episode of ill-temper and loss of self-control on the part of an adult, rather than always being due to repeated, purposeful, violent actions.

There is wide variability in the clinical presentation, ranging from non-specific symptoms, such as vomiting, to coma or death. In its less severe forms the diagnosis is often missed, being confused with viral illness or gastrointestinal upset [114].

Although often not present, strong indicators that a child may have been subject to violent and abusive shaking are finger or hand mark bruising of the chest wall or arms, sometimes coupled with fractures of the rib cage [109].

Livingstone and Childs [115] describe three components of the Shaken Baby Syndrome: a clinical syndrome, a radiological syndrome, and, lack of a compatible history to explain the first two.

The clinical syndrome of SBS comprises:

- A severe acute encephalopathy with a characteristic clinical course;

- Extracranial features present in up to 70% of cases, including the whole spectrum of physical abuse such as bruises, bites and fractures; and
- Retinal hemorrhages in 65-90% of cases.

The radiological syndrome of SBS comprises:

- Characteristic intracranial hemorrhage with or without an extra-axial fluid collection, this hemorrhage is usually subdural but may in addition be subarachnoid;
- Diffuse parenchymal changes, in particular cerebral edema or contusions;
- Subcortical tears if present are probably pathognomonic; and
- Possible skull fractures and soft tissue swelling.

The mechanism of injury is inferred from a possible triad of signs:

- Severe brain swelling and/or diffuse axonal injury;
- Subdural / subarachnoid hemorrhage; and
- Bleeding in the retina, in the absence of a history of significant accidental injury or other medical conditions sufficient to explain the findings.

There may also be other evidence of abuse, such as rib or long-bone fractures [116].

A large subdural hematoma may act as a space occupying lesion, causing direct compression of the brain and leading to herniation of the brainstem. This, however, is uncommon as a result of shaking alone, and even in fatal cases the quantity of blood in the subdural space may be insufficient to cause a major direct mass effect.

A more usual feature of the syndrome is cerebral swelling, typically due to cerebral edema. If uncontrolled, intracranial pressure can rise to above arterial blood pressure, at which point the brain ceases to be perfused. Cerebral hypoxia may be the consequence of cerebral edema, but a possible additional mechanism is direct damage to the medulla, with axonal injury leading to respiratory arrest and hypoxia. Contusional tears of the brain substance can occur, and are presumed to be the direct result of either impact or shaking.

Disruption of the axons, known as diffuse axonal injury (DAI), is regarded as being sometimes a feature. The older concept of DAI was that shearing of axons occurred as a direct physical consequence of the trauma at the time of injury or very soon after. The newer concept is that axons are not torn by shearing or tensile forces at the moment of injury except in extreme circumstances, but instead focal axonal damage (whether by trauma or hypoxia) impairs axonal transport leading to axonal swelling, disruption and disconnection. DAI is diagnosed by use of histopathological techniques. It is less easy to identify in infants than in older children or adults. Conventional histopathological techniques (e.g. with silver staining) are less sensitive than the newer method of beta-amyloid precursor protein (BAPP) immunostaining. Thus, in one study of 25 subjects with fatal head injuries (all but 3 were adults), DAI was found in 8 cases with Palmgren silver staining but in all 25 with BAPP staining. Moreover, with conventional techniques DAI may not be detectable until 15 hours after injury. With BAPP staining it can be detected within 2-3 hours of injury; thus in a fatal case a negative result with BAPP staining means either that DAI is not present or that the axonal injury, if it occurred, occurred less than 3 hours before death [109].

DAI is important because it is probably responsible both for neurological effects (e.g. unconsciousness) immediately after injury (if it is trauma which has directly led to the DAI rather than hypoxia) and for long-term neurological damage. It is also a cause of cerebral edema, which in turn is potentially damaging to the brain. Whatever the mechanisms, severe neurological damage and handicap are common outcomes in the Shaken Baby Syndrome [109].

[Does shaking cause sub-dural hemorrhage?](#)

Guthkelch (1971) [110] first proposed the role of shaking in the genesis of SDH. However, Bruce and Zimmerman (1989) state that there is no proof that shaking produces the clinical, radiological and pathological findings attributed to it [109]. Punt et al. (2005) also concede that 'in pure experimental terms', this is a correct statement [104]. Biomechanical studies using animals and

models have indicated that the forces required to damage and tear the veins of the brain and cause SDH are enormous, about 20 times those attainable by fit, young adult volunteers [117]. Research by Bruce and Zimmerman (1989) [118] suggests that the combination of shaking and impact is required to produce SDH.

A further mechanism of damage due to shaking is presented by Geddes et al. (2001a, 2001b) [119, 120]. They showed that most infants with inflicted traumatic head injury had suffered lack of blood or oxygen supply to the brain. One third had torn nerve fibres in the part of the brainstem where the respiratory control centres are found. The authors suggest that damage there would cause a baby to cease breathing, leading to a cascade of events resulting in the brain swelling and retinal and subdural hemorrhages. The validity of this hypothesis has been rejected by some [121], and more recently, as mentioned above, by the English Court of Appeal.

Nevertheless, a study of 48 infants conducted by Duhaime (1987) with mean age of 7.85 months who had experienced inflicted head injury showed that 18 had no evidence of blunt impact injury to the head [122].

[Pathogenesis of retinal hemorrhages](#)

The pathogenesis of retinal hemorrhage in the Shaken Baby Syndrome is poorly understood. Two main mechanisms have been proposed. One is a shearing effect on the retina at the junction of the vitreous to the retina, particularly at areas of rigid adherence such as around the optic nerve and at the pars plana.

The other suggested mechanisms include a rise in intraocular venous pressure, due to a rise in intracranial pressure, an increase in central venous pressure or rupture of vessels within the subarachnoid space. All result in pressure within the central retinal vein. [109]. Some biomechanical authors view this with scepticism with Ommaya et al (2002) stating: “*the levels of force required for retinal bleeding by shaking to damage the eye directly is biomechanically improbable*” [123].

Application in legal settings

In April 2006, a hearing was conducted before a judge alone to test the validity and admissibility of proposed medical and scientific evidence in a Kentucky Circuit Court case [124]. A Grand Jury had indicted the defendant for first-degree criminal abuse by violently shaking a child.

The Defendant alleged that the child's medical records indicated that the only significant injuries for the victim were a subdural hematoma and retinal hemorrhaging, and there was no significant bruising, fractures, or evidence of impact. The prosecution's case was based upon the existence of Shaken Baby Syndrome.

The Court after hearing expert testimony and reviewing the evidence, made the following finding:

"The Court can further conclude that based on the medical signs and symptoms, the clinical medical and scientific research communities are in disagreement as to whether it is possible to determine if a given head injury is due to an accident or abuse. Therefore, the Court finds that because the Daubert test has not been met, neither party can call a witness to give an expert opinion as to whether a child's head injury is due to a Shaken Baby Syndrome when only the child exhibits a subdural hematoma and bilateral ocular bleeding. Either party can call a witness to give an expert opinion as to the cause of the injury being due to Shaken Baby Syndrome, if and only, the child exhibits a subdural hematoma and bilateral ocular bleeding, and any other indicia of abuse present such as long-bone injuries, a fractured skull, bruising, or other indications that abuse has occurred."

The trial court's ruling is apparently not considered binding legal precedent. The Commonwealth of Kentucky has appealed the ruling to the state's intermediate appellate court [125].

In July of 2005, the Court of Appeal in the United Kingdom reversed or reduced three convictions of SBS, finding that the classic triad of retinal hemorrhage, subdural hematoma, and acute encephalopathy are not 100% diagnostic of SBS and that clinical history is also important [126].

In their ruling, they upheld the clinical concept of SBS but dismissed two cases and reduced the sentence on a third based on their individual merits. In their words: *"Whilst a strong pointer to NAHI [non-accidental head injury] on its own we do not think it possible to find that it must automatically and necessarily lead to a diagnosis of NAHI. All the circumstances, including the clinical picture, must be taken into account."*

Blunt head injury

Introduction

As mentioned above, consideration of blunt head injury overlaps with shaking.

There is a point at which most forensic pathologists will agree that non accidental injury is the most appropriate conclusion in a particular case of fatal blunt head injury; for example when there is a multiplicity of injuries , often occurring over a period of time, clearly at odds with the provided history. More problematic is when there is a paucity of injuries and plausibility of some degree in the provided explanation. While on the one hand the vagueness of the story can be one of the suspicious elements in a particular case, it works the other way when one is asked in the witness box: *"Well, Doctor, the parameters of the story accommodate sufficient possibilities to account for what was observed, don't they?"* Attempts to say that a clearer account would have been expected if there was truly an accident may or may not be accepted. In addition, in many legal systems, no one is actually obliged to say anything, in which case the pathologist may have to conclude to a level of specificity which is impossible if the findings are few and there are no specific circumstances provided. Of course, how a jury or other decider of fact will view all of this is another matter.

Subdural hemorrhage

Subdural hemorrhage (SDH) results from bleeding into the potential space just beneath the dura, between it and the brain. Subdural hematomata may connect between all subdural compartments [127]. Whereas the majority of SDH cases in infants is caused by trauma, numerous natural and unnatural causes have been described [104, 127]. These include mode of delivery at birth, thrombocytopenia, vitamin K deficiency, hemophilia, hepatic disease, infection and disseminated intravascular coagulation.

Evolution

Classification of subdural hemorrhage into acute and chronic groups is time honoured [128], however the definitions of time have varied considerably. In 1942 Munro [129] defined acute subdural hemorrhage as 'evidence of fresh or unhealed' hemorrhage.

In 1960 McKissock et al. [128] provided the temporal definitions that related the time of presentation to the time of injury. Acute indicated an interval of less than 72 hours, subacute an interval of 72 hours to 21 days and chronic an interval of greater than 21 days. The temporal definition offered by McKissock (1960) presupposes there is accurate and reliable knowledge of the time of the trauma, and is not really applicable to a pediatric population. For this reason Punt et al. (2005) [104] suggested a simple alternative classification of 'recent' and 'old'.

Incidence

A population based study in south Wales and south west England detected an incidence of 12.8/100 000/year in children aged less than two years and an incidence of 21/100 000/year in infants aged less than one year [130]. A more recent study from the British Isles by Hobbs et al. (2005) [131] estimated the incidence at 12.5/100 000 for children between 0 and two years and 24.1/100 000 for infants.

Mechanism

Rutty and Waney Squier (2006) [127] classify mechanisms of traumatic SDH into four categories:

1. Traumatic SDH usually results from tearing of the draining veins as they cross the subarachnoid and subdural spaces to enter the sagittal sinus.
2. SDH may result from tentorial tearing, which is associated with moulding of the head and movement of skull bones, usually during delivery.
3. Thin film SDH may result from oozing of blood from dural and arachnoid blood vessels in conditions where there is hypoxic endothelial damage together with raised intracranial vascular pressure. (There are elements of controversy in this mechanism, echoing as it does the Geddes Hypothesis)
4. Bleeding may occur into the subdural space from a bleed in another intracerebral compartment, after rupture of an aneurysm or following an arachnoid tear.

Punt et al. (2004) broadly describe three causes of SDH.: shaking injury, penetrating trauma (unusual) and impact injury: The significance of impact injury as a cause of subdural hemorrhage is probably underestimated clinically because the incidence of identifiable injury to the head in cases of subdural hemorrhage goes up if an autopsy is performed [119].

How much force causes a subdural hemorrhage?

Punt (2005) argues that there is no evidence that the application of any force that would be regarded as proper by a reasonable, responsible, average carer in the course of everyday childcare might produce SDH [104]. His assertion is supported by Duhaime and Christian (1999) [103] who state ‘this sort of injury is unlikely to be inflicted ‘accidentally’ by well-meaning caretakers who do not know that their behaviour can be injurious.’

In a population study involving 11,466 infants aged less than seven months Warrington et al. (2001) demonstrated that trivial domestic accidents that occur in households resulted in no

serious consequences. These included 3,202 falls which resulted in 375 head injuries, 21 of which resulted in concussion or skull fracture [74]. Shugerman et al. (1996) reinforce this with their finding that minor impacts and injuries in infants typically resulted in extradural hematomas, whereas subdural hematomas are the result of high energy rotational acceleration/deceleration forces [132].

The controversy can be encapsulated thus: there is no evidence that “violent” force must always occur for a clinically significant subdural hemorrhage to occur; versus: There is no evidence that the forces associated with reasonable, responsible ordinary child care produce clinically significant sub-dural hemorrhage in normal infants and children.

[Does birth injury cause subdural hemorrhage?](#)

Whiby et al. [133] prospectively screened 111 healthy newborn infants in their first 48 hours for intracranial abnormality, especially sub-dural hemorrhage. Using MRI scanning, it was found that 9 of the 111 had sub-dural hemorrhage, all of which were asymptomatic. All resolved within 4 weeks. The group of infants who were delivered by forceps after failed vacuum extraction showed a significantly higher incidence of sub-dural hemorrhage. There were no such hemorrhages in those delivered by caesarean section. Of the 9 hemorrhages, 6 were in the posterior fossa only, one was supratentorial, and two were both infra and supra tentorial. A retrospective study in Dallas [134] revealed 26 symptomatic presentations of sub-dural hemorrhage from a population of 50,000 births, producing an incidence of 0.05%. Half the infants had normal vaginal delivery. Only one infant required surgery (for a depressed skull fracture). None required evacuation of the hemorrhage.

Uscinski (2002) has suggested that clinically silent SDH related to birth and delivery might generate subdural hematomas which may be discovered at a later date and be mistakenly attributed to the consequences of inflicted injury, and that membranes arising from such hematomas might result in rebleeding that could be wrongly attributed to a further inflicted injury [135].

[Do chronic subdural hemorrhages spontaneously rebleed and cause death?](#)

Again the controversy can be framed thus: there is no evidence that spontaneous re-bleeding of an existing clinically insignificant healing sub-dural hematoma can cause serious brain injury; versus: there is no evidence that only “violence” (as defined above) can cause serious injury from an existing healing clinically insignificant sub-dural hematoma.

Re-bleeds are believed to occur frequently in resolving subdural hematomas, but the amount of bleeding around the existing hematoma is seldom large. The hypothesis is sometimes advanced that the existence of an old, small, chronic subdural hematoma or effusion can predispose to the development of massive and life-threatening acute subdural hematoma as the result of very minor trauma or normal handling. One mechanism that has been suggested for this putative phenomenon is that excess extracerebral fluid allows the brain more freedom to move about within the cranium, thereby rendering the bridging veins more prone to tearing.

If this mechanism did indeed apply, one might expect to see subdural hematoma in association with other causes of excess extracerebral cerebrospinal fluid, such as post-meningitis subdural effusion, communicating hydrocephalus or cerebral atrophy, but with rare exceptions (some of which have been reported on the internet) infants with these conditions have not developed subdural hematoma.

The controversy centres on whether spontaneous rebleeding of chronic subdural hematomas can cause catastrophic clinical deterioration and death. Studies into the histological evolution of subdural hemorrhages [136, 137] suggest that rebleeding is capillary in origin, under low pressure and would therefore be of insufficient volume to become a space occupying lesion. In contrast, acute subdural hematomas result from tearing of larger bridging veins leading to more rapid hemorrhage and the accumulation of larger volumes of blood before the bleeding stops.

[Other issues](#)

Can vitamin deficiencies cause subdural hemorrhage?

Rutty et al. (1999) state that subdural hematomas may occur in childhood with vitamin C and K abnormalities. Vitamin K deficiencies may arise from either maternal vitamin K deficiency with SDH arising in the developing foetus or post delivery vitamin K deficiency, also known as hemorrhagic disease of the newborn (HDN). HDN may be delayed beyond the immediate post natal period and may present with both SDH and retinal hemorrhages [138].

Can vaccinations cause subdural hemorrhage?

According to recent research by Rutty and Waney Squier (2006) [127], to date no peer reviewed papers have demonstrated a categorical causal link between vaccination and SDH. Clemeston's (2004) hypothesis links vaccination, vitamin C deficiency, abnormal histamine levels and SDH but presents no research based evidence supporting this.

Peri-Partum Forensic Pathology

[What does a pathologist mean when s/he uses the term “infanticide” or “neonaticide”?](#)

“Infanticide” has been used as a term by doctors and pathologists for almost as long as they have been involved in evaluating such deaths. In the early 19th century it was defined as follows: “The violent and premeditated death of an infant either born alive or at the time just previous to its birth, is termed infanticide” [143]. Taylor (1844) defined it as follows: “By infanticide we are to understand in medical jurisprudence, the murder of a new born child” [144]. Some authors (Adelson, 1974) use the term neonaticide interchangeably with infanticide, indicating some continuity with the original use of the word [145].

This usage of the word “infanticide” long precedes the development of law of the same name. *The Infanticide Act* (1922) in England and Wales, it would seem, made the term one of legal art, with a special meaning reducing the culpability of the mother for such deaths if particular criteria were met. Interestingly, this Act used infanticide in its neonatal sense. It was only with revision of the Act in 1929 that the relevant period during which the defence of

infanticide could apply was extended to the first 12 months of the infant's life. The forensic pathology usage of "infanticide" to denote neonaticide continues, with the reputable texts having chapter titles incorporating this word. It is intended, in these forensic pathology usages, to indicate the range of ways in which a newly born infant might meet his/her death at another's hands, usually the mother's. The chapters also canvass the general difficulty of establishing that the infant has survived the birth, compounded by difficulties establishing the cause and circumstances of the death if there was survival. It is difficult to conceive that any forensic pathologist, in a jurisdiction with an English-based legal system, who used the word today, would not also have a reasonable appreciation of its legal meaning. The perseverance of the historical usage in contemporary forensic pathology in the face of almost 100 years of differing legal use is an interesting illustration, perhaps, of the separation of law and medicine.

It is interesting that a search of all deaths recorded in the National Coroners Information System (NCIS) (over 130,000 deaths for 2000–07), but not including deaths from Western Australia, there was not one cause of death including the word "infanticide."

[Can a pathologist tell from the post-mortem examination whether a dead new born baby was born alive?](#)

This is clearly a fundamental question, and it surprises many people to learn that the answer is far from straightforward. The question arises when a dead newly born baby is found abandoned such as in a laneway, a rubbish bin, an incinerator, or in a toilet or the cupboard of a house. Adelson (1974) refers to the perplexities surrounding these deaths being such that it is difficult to obtain reliable statistics.¹⁷ The task for the forensic pathologist is to form a view whether the infant was stillborn, or was born alive and died of natural causes, from birth trauma, an accident during or shortly after birth, or from a criminal act [92].

¹⁷ As a problem for forensic pathologists in the developed world, it would seem to be less frequent than in decades past. This is presumably related to improved provision of reproductive and related medical and social services.

There is no particular medical definition of being born alive. The importance of the determination arises from the law, this being the point at which legal personhood is deemed to exist. Forensic pathologists therefore need to look to the law to discover what the relevant definition is. The detail of the law varies from place to place, but in many parts of the world with an orientation toward English law, there is an emphasis upon the following in concluding whether or not an infant, found in the sort of circumstances mentioned above, was born alive:

- The relevant point at which life starts is the point of complete expulsion from the mother. This is generally taken to mean that no part of the infant's body remains within any part of the birth canal. (This does not mean that the infant's umbilical cord is not still attached to the placenta, which, in turn, is still attached within the mother, to her uterus [or womb]. In other words the attachment of the cord and placenta are not relevant to this decision);
- Having been expelled, the infant achieves an independent existence. Essentially, this means that the infant breathes and establishes its independent circulation. (On occasions, a prior question will arise here: is the infant of sufficient maturity—gestational age—to be capable of achieving a separate existence?)

Now, a practical aspect of the context in which the forensic pathologist is considering this issue is that there is often no antepartum, intrapartum, or postpartum medical records or information available. There is usually no witness to say that the infant was heard to cry. (Interestingly, such information may not be determinative of the question of whether there was a live birth because infants can cry—and this requires an infant to breathe—before being completely expelled, i.e., while still in the birth canal. Some of the complexities may be starting to appear in the reader's mind.)

“Forensic pathology texts list a variety of anatomic features useful in differentiating live births from still births ... The entire situation is beset with anatomic ambiguities and pathologic perplexities” [145]. The ends of the spectrum from stillbirth to live birth are fairly well

delineated. A fetus dying in utero and remaining there for a period such as 24 hours or more acquires a particular appearance referred to as maceration. This appearance is the result of autolysis (i.e., the breakdown of the tissues as a consequence of the cessation of the circulation. The failure to deliver oxygen and nutrients means that the tissues of the body die. Under the influence of existing enzymes, the tissues break down). The appearance is different to decomposition and putrefaction in other deaths. A fetus being, usually, sterile will not undergo putrefaction in utero. There will be little trouble for a pathologist, examining the infant within a relatively short time of its delivery, in identifying maceration. At the other end of the spectrum is the undoubted live birth.

The external signs of a separate existence are few. They are limited in fact to changes in the umbilical cord, and the presence of injuries which cannot be ascribed to labour and delivery.... At 36 hours or thereabouts a zone of reddening of the skin appears around the attachment of the cord.... Internal examination may provide strong, if not unequivocal evidence of a separate existence. It can be shown that extraneous material, which could enter only after complete extrusion of the infant, is present in the air passages or the digestive tract. (careful demonstration of milk beyond secondary bronchi—but beware of artefact; and milk in the stomach/duodenum).... For the rest, the demonstration of a separate existence enters a field of investigation in which a definitive assessment of the value of its criteria has yet to be made.... The main test in the past, that known as the hydrostatic test ... [146]

Examination of the lungs at autopsy will assist in some instances to decide whether the birth was a stillbirth or a live birth. The lungs of the stillborn baby are collapsed and are dark purplish-red in colour. They are in the back of the chest and only a small portion is visible from the front. The diaphragm is high [92]. This appearance may be affected by artificial respiration.

Some infants die during labour, and there are numerous causes for this—malpresentation, pelvic deformity, prolonged labour, prolapse of the cord, pressure on the cord, or the cord wound around the neck. Even if a child in such cases has not breathed, well-marked petechial hemorrhages may be found on the heart and lungs [147].

The lungs of an undisputed live birth are distended, well aerated, fluffy, have a salmon-pink colour, and are visible in the front of the chest when the sternum is removed. The chest is expanded, and the diaphragm is lower than the level of the fourth to sixth rib. When the case is a typical stillbirth or live birth – that is, at either end of the spectrum—the external appearance of the lungs is pathognomonic.

Unfortunately in many cases the signs are not so characteristic and may be somewhere in between. It may then be difficult, or not correct, to decide the question of a live birth or a stillbirth. It is important to keep in mind the different possibilities that may occur during birth and to consider what effect they may have on the lungs of the newborn baby.

For example, the fetus may be born with every expectation of breathing, but the cord may be wrapped around its neck so that it cannot breathe, and cord strangulation occurs during labour or just after it. Under such conditions the lungs are like those of a stillbirth.

Partial aeration of the lungs does not always mean live birth as breathing may start before the infant is completely expelled from the birth canal, and the child may die before it is completely delivered. Conversely, the lungs may be atelectatic and sink in water, even when the infant has breathed after birth.

The histological appearances of the lung may not be of great assistance. *“According to the present level of knowledge ... ventilation of the lungs alone cannot be taken as a certain indication of a live birth. Under various circumstances, lungs originally aerated can become devoid of air; conversely, the lungs of still born neonates can appear aerated. It is not possible to be certain in all cases.... In medico-legal work, though it is quite in order to refer to the usual*

state of affairs, when a criminal trial is in progress ... the well documented exceptions just mentioned ... make it unsafe to be dogmatic over a histological opinion” [148].

Sometimes the birth occurs in the bathtub or in a toilet bowl, so that the newborn child is drowned; the signs of drowning in such cases are often indistinct, especially if there is also an element of post-mortem decomposition present. The diagnosis therefore often cannot be made positively on the evidence discovered at necropsy.

Police investigation producing evidence indicating a crime will usually be needed if there is to be a prosecution in these circumstances. Smothering in the membranes; overlying by the mother, which may be produced accidentally during an unattended birth; or homicidal smothering or drowning will probably all be indistinguishable at autopsy. Table 20 below sets out some related comments from the literature about these issues.

Table 20—Establishing “live birth”

Author	Comments about Establishing “Live Birth”
Gonzales, Vance, Helpert & Umberger, 1954 [147]	The appearance of the lungs can distinguish between an undoubted live birth and a stillbirth. In many cases the conclusion falls somewhere in between. There are problems with concluding drowning in circumstances of recent delivery.
Camps, 1956 [149]	“An experienced pathologist performing post mortem examinations on four babies from a maternity unit came to the conclusion as a result of his examination that two were still born and that two had lived. To his dismay, far from being correct, the clinical notes reported that the two alleged live births had in fact shown no evidence of life, whilst the two alleged still-births had lived for at least 24 hours in hospital.” This case is not referenced in the book so must be treated with some skepticism. However, its inclusion in the book illustrates the importance attached by the authors to the caution that should be exercised in making these conclusions.
Bowden, 1965 [92]	The presence of petechial hemorrhages on the surface of the heart and lungs is not evidence of a live birth.
Polson, Gee & Knight, 1985 [146]	Even if one accepts the signs of asphyxia as real, there could be natural explanations in circumstances of perinatal deaths. The presence of Tardieu’s spots are not signs of a live birth.
Saukko & Knight, 2004 [79]	The expansion of the lungs really must be unequivocal for the pathologist to conclude that there was a live birth. Smothering is almost impossible to prove ... Establishment of the infant having breathed has come to be accepted as proof of a separate existence, although separate existence cannot be established at autopsy. The unequivocal demonstration of breathing in a newborn is fraught with difficulty. Along with the estimation of the time of death, it has probably provoked more discussion, printed words, and controversy than any other topic in forensic medicine.

These are problematic cases, compounded by the fact that they are relatively infrequent. Individual forensic pathologists see few such cases now in the developed world, and many may not see more than one case a year in Australia. This is another example of the importance of forensic pathology actively engaging with another sub-specialty, in this case, neonatal pathology, which may have some experience in examining numbers of perinatal deaths in a hospital setting.

Chapter 4—Establishing the Cause and Manner of Death: The Hidden Controversy

Introduction

The attribution of “cause” in forensic pathology is a fascinating subject about which too little has been written [150]. Pollanen has made a significant recent contribution [151]. It is one particular area, amongst many, of confusion at the dynamic interface of law and medicine. One reason for this confusion is that those involved have to grapple with (at least) two different conceptions of cause: that used in medicine and that used in law. This chapter has been written from a medical perspective, which is clearly all the authors can try to do.

The chapter begins by outlining the terrain covered by forensic pathology and the main features in the landscape. It then briefly looks at the form in which the cause of death is certified. The main aim is to arrive at something like a coherent schema for the attribution of the cause of death following autopsy. While in most cases this is straightforward, in many cases in pediatric forensic pathology it is not, and in those cases there is not a completely uniform approach. This is not surprising as the attribution of cause, particularly in retrospect and in as complex and unpredictable field as biology, is essentially a philosophical question. During the course of this chapter, examples of the issues arising in a number of cases will illustrate the breadth of the forensic pathologist’s contribution and the complexity of the associated causal issues, which, it must be said, are sometimes not appreciated by either pathologists or lawyers.

Forensic Pathology—The Terrain

Forensic pathology is the application of the principles and practice of pathology to the needs of the courts, or more generally, the law. Pathology is the study of disease. Anatomical pathology, its largest subdiscipline, has left its morbid anatomical roots and concentrates in the main on surgical and biopsy pathology. Forensic pathology, closely allied to or even derived from

anatomical pathology, is a mortuary-based specialty that provides the knowledge basis for the performance of autopsies in deaths reported to Coroners, or their equivalents.

It is important to appreciate, at the outset, the scope of the forensic autopsy. The aims of this part of a death investigation are [152]:

1. To discover, describe, and record all the pathological processes present in the deceased and, where necessary, the identifying characteristics of the deceased;
2. With knowledge of the medical history and circumstances of the death, to come to conclusions about the cause and time of death and factors contributing to death and, where necessary, the identity of the deceased;
3. In situations where the circumstances of death are unknown or in question, to apply the autopsy findings and conclusions to the reconstruction of those circumstances. This will, on occasions, involve attendance at the scene of death, preferably with the body in situ; and
4. To record the positive, and relevant negative, observations and findings in such a way as to enable another forensic pathologist at another time to independently come to his or her own conclusions about the case. As forensic pathology is essentially a visual exercise, this involves a dependence on good quality, and preferably colour, photographs.

Encapsulated in this approach to the forensic autopsy are two consequences at odds with a common perception of the specialty. Firstly, forensic pathology could be regarded as the “what happened” specialty (and not the “whodunnit” specialty). It is as part of this that the pathologist is concerned with coming to the best conclusion about the cause of death. However, and this is the second consequence, in pursuit of answers to “what happened,” conclusions about the cause of other findings on or in the body, or at the scene, or of events described in witness statements, may require the pathologist to attribute “cause” in areas other than the cause of death. Provided the pathologist keeps to his or her expertise, this is a quite proper exercise.

Certification of the Cause of Death

There is a preliminary area that needs a little exploration, which is the formality of framing the cause of death.

Table 21—Cause of Death

1	
Disease or condition directly leading to death.**	(a)
Antecedent causes (morbid conditions, if any, giving rise to the above cause, stating the underlying condition last).	(b)
2	
Other significant conditions (contributing to the death but not related to the disease or condition causing it).	

** This means the disease, injury, or complication that caused death, NOT ONLY, for example, the mode of dying such as “heart failure, respiratory failure,” etc.

One needs to understand the form of the cause of death to appreciate what is being conveyed. The cause is divided into 1 and 2. “1” is the direct cause of death or the disease or condition directly leading to death. “1” is subdivided, if necessary, into a, b, and c (and theoretically d and e, etc., if necessary; it very rarely is). “1a” is due to or a result of “1b” and so on. The last listed condition under “1” is the main, central or underlying cause of the patient’s death. “2” represents those other significant conditions contributing to the death but not directly related to the disease or condition causing it.

This is the internationally accepted form that favours singular particular causes of death (which makes coding, classification, and statistics easier) and does not invite multiple interacting causes. There is an ill-defined distinction drawn between cause (“1a, b” above) and contribution (“2” above) that is essentially arbitrary where the latter is some form of lesser cause of death acting independently of the main cause of death.

The cause of death is not often a contentious issue in litigation, and when it is, there will almost always have been an autopsy. It is to these cases that we now turn.

Deciding the Cause of Death following Autopsy

Deciding the cause of death is a fundamental responsibility for all anatomical pathologists after autopsy. The responsibility is greatest for forensic pathologists, yet very little has been written about the criteria that need to be satisfied to make a decision. This issue causes confusion in some court cases because both pathologists and lawyers fail to appreciate something of the philosophy of causation. Leaving aside the minority of cases where the lesion observed at autopsy is incompatible with life (e.g., decapitation), what in fact usually happens in coming to a conclusion is that *a* cause of death discovered at autopsy, which accords with the medical history and circumstances, is elevated to *the* cause of death. In general terms, the pathologist makes a decision that a certain autopsy finding is capable of leading to death, and that as this is consistent with the deceased's medical history and the supposed circumstances of death, and there is no other competing cause, it is the cause of death. Such a conclusion about the cause of death is retrospective and therefore cannot generally be tested. This approach emphasizes the need to discover all the pathological processes present in the deceased before considering them in relation to the medical history and the circumstances of death. A corollary of this is that if there is no autopsy finding discerned that is capable of leading to death, the pathologist will be reliant completely upon the circumstances and medical history. To the extent these are likely to be disputed, or are inherently difficult to corroborate, the pathologist will need to be careful.

Causation: Philosophy and Problems

The authors are not philosophers, but have found the following to be a useful framework for discussion. David Hume (1711–1776), believed that for X to be the cause of Y, X must be both sufficient and necessary for the effect, Y: thus, X is always followed by Y, and Y never occurs unless X occurred. Somewhat differently, John Stuart Mill (1806–1873) thought that the cause was the sum total of the conditions in which an event occurred: it was not correct to isolate *one* of the conditions in which an event occurred as the exclusive cause. To Hume, the statement “*the rising of the sun causes daylight*” would have been reasonable, since the rising of the sun is always followed by daylight, and daylight never comes about unless the rising of the sun has occurred. The statement is, in fact, incomplete because daylight could not occur unless there was an atmosphere. Mill’s approach would include an atmosphere in any statement about what caused daylight because it is one of the conditions in which the event occurs.

The restrictiveness of Hume can be seen in the commonest cause of death in the Western world: coronary atherosclerosis (“hardening of the arteries of the heart,” “heart disease,” “heart attack,” “myocardial infarction”). The development of coronary atherosclerosis is not always followed by death, and death does not occur only when coronary atherosclerosis has developed. Yet clearly it is a reasonable proposition that coronary atherosclerosis has been the pathological basis for an enormous number of deaths. It seems that Hume’s approach is suited more to those cases where the cause of death is incompatible with life, for example, decapitation. This is not to say that Mill’s approach is necessarily the complete answer. Take the example of the heavy smoker who dies of carcinoma of the lung. One of the conditions in which the death occurred is smoking, but there are more: a person may smoke because of the effect of advertising, because of parents’ smoking, because of particular personality traits, and so on *ad absurdum*. Pathologists (and in some cases, courts) have to make a practical decision that cause stops somewhere. In

general, the line is drawn at the “medical” cause of death, but, as the example shows, this may be unsatisfactory: smoking is increasingly noted on death certificates.

[Establishing the cause of death is heavily dependent upon an interpretation of the circumstances of death](#)

This is a particularly important issue in pediatric forensic pathology, all the more so because it may be difficult to clearly establish what the circumstances are. Let us start with an adult case.

Mr. A. was a 29-year-old man with no known previous illnesses, was working with electrical machinery when he suddenly collapsed and died. His workmates thought he had been electrocuted, although others in contact with the same machine had felt no shock. The results of the examination of the machinery were controversial; government inspectors were saying that it was conceivable that the machine had been electrically alive, electrical engineers retained by the factory were saying it was not. At autopsy, there were no marks of electrocution. (It is quite possible to be electrocuted and for there to be no marks.) The only positive finding was appreciable hydrocephalus (“water on the brain,” or dilatation of the ventricles of the brain) but no acute cerebral edema (or brain swelling due to intercellular fluid accumulation). Some basal meningeal thickening suggested the hydrocephalus may have been secondary to meningitis in infancy or childhood. There were no abnormal histological or toxicological findings. If it is assumed that uncomplicated hydrocephalus (as in this case) can cause sudden unexpected death (a matter of some dispute at the inquest), it is easy to see that the cause of death is completely dependent on the assessment of the machinery by electrical experts. Even the assessment of the circumstances contained causal issues because the inspectors, who said it was conceivable that the machine could become electrically live, could not say that it actually had been. For the purposes of discussion, let us consider this real case.

Baby A was 3 months old and left in the care of a local authority nursery. It was windy and snow was on the ground. She was left outside in a pram unattended for

three hours. When she was fetched in, it was found that she was dead. There were no significant pathological findings at autopsy.

The definition of the Sudden Infant Death Syndrome (SIDS) is the sudden unexpected death of an infant during a period of supposed sleep in whom a thorough autopsy, and review of the medical history, scene, and circumstances of the death, fails to find any adequate cause for the death. The absence of any pathological findings in the case above puts this death in the category where SIDS would be considered, subject to a review of the medical history, scene, and the circumstances. It also needs to be understood that death from exposure (or, more technically, hypothermia—low body temperature) may also have no pathological findings. Clearly, the circumstances of the death in this case, having been left outside in the wind on a freezing cold day, mean that hypothermia will have to be considered as a realistic cause of death. One approach would be for the pathologist to conclude that the cause of death was “unascertained,” and then to have discussed in his/her report the extent to which the exposure may have been involved in this death by a consideration of the circumstances. Another pathologist may have concluded that the cause of death was indeed “hypothermia” or was “consistent with hypothermia.” There may have been information that only surfaced months after the death that in fact the nurse at the nursery took the baby’s temperature soon after she was brought inside, and it was 28 degrees Celsius. This observation might be regarded as supporting death from hypothermia, but as a matter of fact cannot exclude death from some other (possibly natural cause) soon after being left outside, and the drop in temperature was simply cooling after death in a cold, windy environment.

Table 22—Possible approaches to the cause of death in the case of an infant left outside on a very cold day

Cause of Death	Comments
1 (a) Sudden Infant Death Syndrome	No. Hypothermia as a realistic possibility, based solely on the circumstances of the death, excludes a diagnosis of SIDS
1 (a) Hypothermia	The pathologist’s opinion is that the cause of death is hypothermia. In his/her experience, the circumstances are such that hypothermia could explain the death, and in the absence of any

Cause of Death	Comments
1 (a) Consistent with hypothermia ¹⁸	<p>competing cause of death, s/he believes it is reasonable to conclude that hypothermia indeed caused the death.</p> <p>As above, except that the pathologist believes that other, perhaps slight, possibilities cannot be excluded. For example, the baby might have had an underlying cardiac arrhythmia (e.g., long QT syndrome, or similar) that manifested itself when the baby was put in a stressful situation, or manifested itself spontaneously within minutes of the baby being left outside.</p>
1 (a) Unascertained (Undetermined, or similar word)	<p>The pathologist is not sufficiently confident that the circumstances can be held responsible for the death, because s/he is slightly surprised that, even though the pram was uncovered, the baby was well wrapped, and was several inches below the upper level of the pram such that the worst effect of the wind would not have been felt. The other slight possibilities assume greater significance in this pathologist's mind, and while hypothermia is discussed in the report as a possible cause of death, in the end the pathologist's opinion is that s/he cannot say.</p>
1 (a) Unascertained in an infant left outside in a pram on a very cold day	<p>Very similar to 1 (a) Unascertained, except that the pathologist wants to indicate formally that s/he believes the circumstances of being left out in the cold are implicated in the death.</p>

¹⁸ The use of the phrase “consistent with” generates its own controversy. The controversy sits between its technical usage by the expert, and the thought that a jury member might interpret the phrase as meaning “highly likely”, “highly probably” or, even “is” hypothermia. Logically, “consistent with” carries with it the inference that the thing could be consistent with something else. It is in this latter sense that it is used here. Its usage in reports and in oral evidence should probably always be qualified by this explanation.

One only has to consider that if this baby had been found dead at home, at 6 a.m. in her cot, with precisely the same autopsy findings, the cause of death would have been correctly given as SIDS. This is an example of dependence upon information about the circumstances. The pathologist in the mortuary, blind to the circumstances, would not be able to tell the difference between the two deaths.

Let us consider another actual case. D.L., a two-year-old boy who died following surgical evacuation of a subdural hemorrhage resulting from a head injury. The defence wanted to establish that after whatever injury caused the subdural hemorrhage there was a 6 hour delay (lucid interval) before the child deteriorated. The neurosurgeon involved did not believe that such an interval was a realistic possibility. (The existence of lucid intervals is a controversy in pediatric forensic pathology that has not been discussed in this paper). Notwithstanding that the accused admitted hitting the child while he was sitting in a car seat, he was acquitted because the pathologist discovered a previously existing old subdural hemorrhage that complicated the assessment of when the acute subdural may have started and the force required to cause the acute bleeding. The formal cause of death given by the pathologist was 1(a): Head injury.

The cause of death does not really address the issues of concern to the court:

1. Was the injury admitted as having been inflicted by the father the injury that caused the death?
2. If so, what sort of force must have been involved?

In another case, a two-and-a-half-year-old boy was left alive and well by his mother in the care of her de facto husband. When she returned one hour later, the child could not be wakened. An ambulance was called and it arrived some 15 minutes later. The child could not be resuscitated. The account of events leading to the child's death came from the de facto husband, who described himself losing control and striking the boy to the back of the head with an open hand some nine times with the child perched prone over his lap. The blows occurred in the one-hour period when the mother was absent and the de facto husband described putting the child to bed after the final blow. No lucid interval was described. At autopsy, there was evidence of

several discrete areas of bruising subcutaneously on the back of the head. There was no significant subgaleal hemorrhage or skull fracture. There was no extradural, subdural, or subarachnoid hemorrhage. The brain was, however, swollen, showing mild tentorial grooving of both hippocampi. Formal neuropathological examination was undertaken including ophthalmological examination. No cerebral parenchymal injury was demonstrated. There was no evidence of axonal spheroids to suggest diffuse axonal injury as a mechanism. There were no retinal hemorrhages. The cause of death was cerebral edema, most likely secondary to trauma. The autopsy findings were relatively subtle and arguably non-specific. The description of malignant cerebral edema or rapid brain swelling following head injury (often trivial) is, however, well described in the literature [25, 153, 154]. Nonetheless the admissions of the accused were essential to the marrying of the relative non-specific evidence of trauma to the back of the head (discoid bruises) to the ultimately determined cause of death. He was found guilty of manslaughter and sentenced to seven years imprisonment. Had the accused asserted his right to silence, the outcome may well have been completely different.

Concluding the Cause of Death: A General Formulation

What general rule, if any, can we formulate following all of this? If the autopsy discovers a finding, a disease or condition that is operating at the time of death and that is capable itself of causing or accelerating death, and in the circumstances of the particular death, its effects apparently exerted themselves, then the disease or condition should be included as part of the cause of that person's death. (This formulation deals, *inter alia*, with the situation where it would be highly pedantic to include coronary atherosclerosis in the cause of death of a man decapitated by a train having been seen by the driver to place himself on the line in the path of an oncoming train.) In addition, if a disease or condition (whether or not a potentially fatal condition) aggravates or complicates another disease or condition (whether or not a potentially fatal condition) such that death occurs, then both diseases or conditions should generally be included as part of the cause of death.

This formulation has its weaknesses; for example, it does nothing to help draw the line in relation to remoteness of cause.

Conclusions as to Manner of Death

It will have been apparent in some of the cases above that conclusions as to the cause of death often carry with them an implication as to the manner of death; but not always. Making conclusions as to the manner of death is a fundamental responsibility of medical examiners in the United States and of many Coroners' jurisdictions around the world, but not all.

Making these conclusions can be a bit like pushing square pegs into round holes. So much so that some jurisdictions have moved away from requiring such conclusions. In Victoria, the Coroner must find who the deceased was, the medical cause of death, and how the deceased died [155]. The last requirement is met in descriptive terms. For example:

“The deceased, who was an intravenous drug addict, unemployed and depressed, was found on the floor in the kitchen of the house he was sharing with friends. Intravenous drug paraphernalia was found on the kitchen table, recent injection marks were identified on his forearms at autopsy, and metabolites of heroin together with alcohol and a benzodiazepine were found on toxicology. The cause of death was ‘Mixed Drug Toxicity.’

“Accidental” overdose is the commonest situation in circumstances of heroin abuse leading to death. In this case the possibility of suicide also exists. The Coroner has chosen to leave the matter open. The word accident or suicide is not used.

The reverse is true in England where Coroners are required to classify the death into one of a number of categories [156]. For example, natural cause, industrial disease, want of attention at birth, dependence on drugs, killed himself (while the balance of his mind was disturbed), accident, misadventure, murder, manslaughter, infanticide, accident, open verdict. Doing so may not be straightforward as some work from the United States has verified.

A study by Hazlick and Goodin (1997) is interesting in demonstrating the high level of disagreement in this area [157]. Twenty-three succinct, well-described classical forensic pathology situations were presented to more than 700 medical examiners/Coroners who were members of the National Association of Medical Examiners, eliciting responses from 198 of them. The manner of death inferred from the ICD Code that was assigned by the (U.S.) National Center for Health Statistics matched the most common response of participants in 18 (78%) of the 23 scenarios. Table 23 shows the percentage of agreement for the most popular conclusion (homicide, suicide, accident, natural, undetermined, other/blank).

Table 23—Percentage of 198 forensic pathologists agreeing on the most popular manner of deaths in 23 scenarios

%	No. of Scenarios (n=23)
41–50	2
51–60	4
61–70	5
71–80	1
81–90	7
91–100	4

In fewer than half the cases, admittedly chosen because they were at the boundary of different manners of death, was there greater than 80% agreement. In other words, in only 11 out of 23 cases did more than 80% of 198 U.S. forensic pathologists agree on the manner of death. There was considerable diversity of opinion amongst the relevant experts as to the manner of death in these examples. This points to different understandings, even on the same facts, of the criteria to establish particular manners of death. Such differences commonly include:

- how “certain” one should be to make a conclusion (e.g., the special consequences of concluding suicide may be such as to mean some will require a higher standard of proof than others); and
- different understandings of intention (e.g., the reckless killing of another may be a homicide whereas the reckless killing of oneself will generally be considered an accident and not a suicide).

The differences amongst pathologists and between them and the health statisticians about the manner of death shows how contentious assigning this form of cause can be, albeit in cases designed to be contentious.

Chapter 5—Case Studies

Overview

This chapter aims to capture some of the challenges of pediatric forensic pathology through the use of case studies: a fatal head injury in a young child; a newly born baby found dead; and a classic “Battered Baby.” Let us imagine the process of the forensic investigation, starting with the history provided by the authorities and the caregiver, the autopsy conducted by the forensic pathologist, and concluding with the forensic pathologist giving evidence in court. The purpose of the exercise is to see what the pathologist says: after all, this is where the limits and controversies are played out with real consequences for justice. This is not to say that equally key opportunities to influence the course of events do not occur very early in the process when the pathologist’s view will be very important to the attitude taken on by police. We have tried to impose the same structure on each case and to frame the questions accordingly. This means that some questions will be less relevant in some cases than others. This schema is one that forensic pathologists and barristers may find useful to contemplate. The questions are not intended to be exhaustive but many of them illustrate recurring themes in pediatric forensic pathology and forensic pathology more generally.

The Structure for Evaluating the Case Studies

1. What is the cause of death?

The cause of death is a basic piece of data. A major aim is to identify whether the cause of death is related to injury. If so, discussion about whether any conclusions can be made about how the injury was sustained, and whether there are alternative explanations, will follow.

2. What are the key physical signs found at autopsy in this case study?

These signs should be explained in relation to the cause of death, i.e., whether they are specifically related to the cause of death or not. This is to explain the difference between general and specific signs that may be observed, and how the signs present may be diagnostic, supportive, or neutral in relation to the cause of death. Are you relying on observed signs at autopsy or on clinical signs during life or other information in witness statements?

3. How did the physical signs found at autopsy in this case study lead you to your conclusion about the cause of death?

Describe a classic presentation, the most common presentation, or the most complex/indeterminate case, i.e., describe how the information in the previous section is thought through.

4. How did you ascertain these features were present?

The pathologist will obtain information by imaging techniques (e.g., plain radiography, CT scan), direct visual inspection, histopathology, and other special tests.

5. Are the described features verifiable?

Explain how the presence of physical signs and other key findings are documented, allowing another forensic pathologist to review them. Are there any known inter-observer differences? Do certain signs require specialized equipment, pathologist experience, or technical expertise to discover them?

6. Are the findings beyond the scope of the individual pathologist? If so, who else or what else is being relied on? Is there a clear chain of custody in relation to samples sent for special testing?

Describe any reliance upon other expertise in reporting the autopsy findings, and the conclusions generated from them. If these findings and conclusions relate to further examination or testing, is the chain of custody accurately documented?

7. Are the observed signs due to artefact, natural disease or injury?

Explain why they could or could not be due to artefact natural disease processes.

8. Are the observed signs of injury due to intentional harm in this instance?

If this requires clinical, witness or scene investigation or knowledge this should be clearly stated and explained. For example, is the information being relied upon to draw conclusions information from direct observation at autopsy, specimen analysis by laboratory, or witness descriptions of the circumstances? The difference between what is observed and what is an interpretation needs to be highlighted.

9. How certain are you, as the pathologist in this case study, that the observed signs of injury are due to intentional harm in this instance?

Have all the possibilities been excluded? Is it feasible or reasonable or appropriate to exclude all possible alternatives? What are the other possibilities?

10. Have the features of this case ever been described in the literature as being non-intentional?

Could this be an exceptional case? What are the exceptions? Discuss and describe the rarity and how ancillary or supporting data is used.

11. Is the evidence/research base relied on in this case unequivocal and definitive?

What are the known limitations of the research these conclusions are based upon?

12. Would your peers come to the same conclusion based on the observed physical signs detected at autopsy?

Explain why or why not.

Purpose

We have tried to impose the above structure onto these different pediatric pathology cases.

Within this structure, when it is transferred to the courtroom, there remains the dynamism of the examination in chief and cross-examination.

There is, as far as we are aware, nothing other than the most general of standards to apply to this part of the forensic pathologist's work. Reduced to its most fundamental component, the forensic pathologist must tell the truth. Put another way, the forensic pathologist must honestly convey his or her opinion. This is assumed, and the structure we have tried to impose stresses the importance of the evidence behind the opinions being disclosed both in the way the evidence was gathered, and the strength of the connection between the evidence and the opinion. These last elements need to be considered by pathologists (and perhaps all givers of expert evidence) as much a fundamental obligation as honesty.

The case studies are not examples of 'best practice' evidence provision by a forensic pathologist. The three pathologists are different people with different experience. All three are well intentioned and want to be constructive and assist the justice system to the best of their ability.

They have no legal training or qualification. We do not believe any of the pathologists are behaving outside the bounds of acceptability. The weakness of the case studies is that none of the authors are barristers - and no doubt the pathologists would have been held to stricter standards by experienced counsel.

Case Study 1: Short distance fall versus inflicted head injury

In this case study a well intentioned and reasonably experienced forensic pathologist is giving evidence in a heavily contested trial. The father claims a short fall was responsible for a fatal head injury. The prosecution says this must be a homicide.

Case History

A deeply unconscious 12-month-old boy (A.B.) arrived by ambulance at the emergency department of a city hospital at 20.30 on a Saturday night. The ambulance had been called at 19:45 by the distraught father, saying the boy had fallen out of his arms and was now unconscious and he did not know what to do. The ambulance had arrived at 19:55 and had found the boy on the floor in the living room. He was unconscious, a large scalp bruise was palpable, and he was intubated. At hospital, a CT scan was performed, indicating a linear parietal skull fracture one edge of which had approximately 4 mm of depression and a large or severe subdural hemorrhage. Bilateral retinal hemorrhages were seen with an ophthalmoscope by the emergency department consultant. The boy died 70 minutes after admission, having been unresponsive to resuscitation attempts by the emergency department staff.

The emergency department consultant interviewed the father. A.B. was at home alone with the father. The mother was working as usual as a barmaid in the local hotel. They were not married, and the family had no contact with social services. A.B. was their first child. A.B. was previously fit and well and had had no need of any significant medical attention during his life. He was being carried in the arms of his father because he was upset at having to sit on the potty in the bathroom preparatory to being put to bed. The father slipped on the ceramic-tiled bathroom floor and the child fell about 3 to 4 feet, hitting the back of his head on the floor and landing on his back. The event was, obviously, not witnessed. A.B. was initially conscious, and after the father picked him up, he was able to waddle in his normal fashion for about a minute or so, but then became obviously unwell and comatose. The father immediately called for an ambulance. After the ambulance came and took A.B. away, he was able to get in touch with the mother. At the end of the interview, the mother arrived at the hospital. Upon being told that A.B. had died, she was inconsolable and shouting at the father, accusing him of hitting A.B. yet again.

A post-mortem examination was conducted the next day. The report indicated the presence of parietal scalp bruising, an underlying 5-cm-long parietal skull fracture, and confirmed the presence of the large unilateral subdural hematoma, cerebral swelling, and uncal herniation, as well as the bilateral retinal hemorrhages. There were a couple of small yellow bruises on the knees and a yellowing bruise on the left forehead 2 cm in diameter. In particular there were no chest or arm bruises and no fractured ribs.

Further investigation by the police revealed that the next-door neighbours had heard the father shouting, as he often did, apparently at A.B. The neighbours could not unequivocally rule out the possibility that the shouting was in fact the distraught father after whatever event occurred. The neighbours had occasionally seen A.B. with one or two bruises on his face. It was also revealed that the family was known to social services. The infant welfare nurse had reported concerns based on the mother telling her that the father was very rough in handling the child. A social worker had visited the family on three occasions—the father had been present at two—and had not thought there were sufficient issues to warrant any further action. The father was charged with a homicide-related offence.

1. What is the cause of death?

Prosecution: Doctor, what was the cause of death in this case?

Forensic Pathologist: Head injury.

Prosecution: Can you be more specific? How did the head injury kill the infant?

Forensic Pathologist: He sustained a fractured skull, the forces associated with which also caused bleeding in the subdural space. There was also cerebral swelling which is really a general response of the brain to many forms of insult. In this case, the swelling could be secondary to the same forces that caused the fractured skull and subdural hemorrhage, it could be secondary to the fact that the brain did not receive enough oxygen following the onset of unconsciousness when the baby's airway was blocked, as is a real danger when anyone is unconscious, or the swelling could be secondary to the effects of pressure on the brain from the subdural hemorrhage. The swelling brain, being in a relatively fixed sized container (the skull), together with pressure from the subdural hemorrhage, probably resulted in the brain stem being squashed through the foramen magnum, causing respiratory depression.

Prosecution: Thank you. In your opinion, what caused the fractured skull?

Forensic Pathologist: I believe the fractured skull was caused by blunt trauma.

Prosecution: Is it possible that the blunt trauma was due to a direct blow to the skull from the accused?

Forensic Pathologist: I believe it was. I do not believe this head injury resulted from a fall in the way described by the father in his statement.

2. What are the typical, classical, or key physical signs found at autopsy in this case?

Prosecution: *Is there anything that would ordinarily be seen in a case such as this which is not present here? In what way is this case unusual compared with a more classical presentation, if there is one, of inflicted non-accidental head injury?*

Forensic Pathologist: *Well, that is a very big question. But essentially, there is a relative paucity of injuries to A.B. compared with classical assaultive non-accidental injury. In the classical situation, there will be a multiplicity of injuries, sometimes concentrated in particular areas such as the head. The multiplicity of injuries exceeds that which would normally be associated with the falling around that toddlers have. In A.B.'s case, he had a bruise to the forehead and a couple of bruises on a knee. To be fair, this is no more than might be seen on an ordinary toddler, but obviously I cannot exclude the possibility of an assaultive origin of the forehead bruise. When there is a multiplicity of injuries, it may be apparent that they have occurred on more than one occasion.*

In addition we need to consider the possibility of a shaking-impact sort of situation. In this scenario, A.B. might be picked up and violently shaken, during the course of which his head struck a hard surface or object, causing the skull fracture and all the consequences described above. In such a scenario, bruises associated with the grip around the chest or the arms can be seen; there were no such bruises in this case. Also, the ribs can be fractured. There were no rib fractures in this case. There can be injuries to the musculature of the neck and to the nerve roots of those nerves arising from the cervical spine. No such injuries were seen here. Also, these cases tend to involve younger children, such as infants in the first few months of life, but it can occur in toddlers and even older children.

In a fall, there may be other injuries associated with the particular fall. There were no other injuries seen in this case referable to a fall at the same time as the occurrence of the head injury.

A linear skull fracture is the most common form of skull fracture, however caused, whether accidental or non-accidental. Distinguishing between the two presents a challenge to the forensic pathologist. Overall probability that a skull fracture is due to abuse is said to be 23%, rising to 37% if the fracture is complex [158]. Such percentages are little help in any particular case. Skull fractures are caused by a deformation of the skull due to impact of some kind. The likelihood that a child will suffer a skull fracture depends on the force, location of the impact, age of the child, and biologic/mechanic characteristics/properties of the skull at the point of impact.

Subdural hemorrhages can be caused by impact injury following falls, inflicted blows, or motor vehicle accidents [59], as a result of bleeding from a congenital malformation in the brain such as an arterio-venous malformation [73], clotting disorders, or vitamin deficiencies [127]. Extensive subdural bleeding can occur in the absence of any observable injury to the scalp visible at autopsy. The characteristics of the bleeding cannot therefore be used to determine the cause of the subdural or the manner in which it occurred.

3. What are the physical signs found at autopsy in this case that led you to the conclusion that this was the cause of death?

The forensic pathologist found the cause of death to be head injury and believes it was not due to a short distance fall. However, it may not be possible to actually determine the manner in which the trauma was caused from the autopsy findings alone. To determine the manner in which the trauma was sustained requires further information; namely, a truthful account of the circumstances in which the injury was caused from the caregiver at the time.

Prosecution: How do you determine whether the injuries were sustained in an accidental or non-accidental manner?

Forensic Pathologist: Feldman and colleagues have proposed a gradient of certainty for determining the mechanism of injury [59]. The spectrum spans from definitely accidental in which there is a single vintage injury; the major event is corroborated by direct observation by a reliable and independent third party— to definite abuse that is a corroborated, witnessed, or confessed event; or there are multiple injuries, incompatible with normal, accidental childhood injury; incompatible with the history of injury vintage(s) or child's development; multiple or patterned bruises; old, unexplained fracture(s); findings of cranial impact without history or with inadequate history of impact trauma[59].

By virtue of my own reading, and presentations I have heard at conferences, I am aware that the incidence of fatal head injury from short falls is exceedingly rare and that falls from very much greater heights than proposed here cause no skull fractures and are not fatal. I am also aware in this case that the father clearly has problems with anger management and has lied about aspects of the past history. What is more, there were no other superficial injuries that might have occurred in the course of a fall from 3–4 feet to corroborate the likelihood of a fall. I concede that there is really only the one significant injury in A.B.'s case, and that this puts the case into the category where an accidental

explanation needs to be considered. But the reasons I rely on for concluding this is an inflicted injury are:

- *The very low incidence of fatal head injuries in short falls;*
- *The generally designated height from which falls need to occur for life-threatening injuries to be sustained is very much greater than is said to have occurred here;*
- *The absence of other injuries that might have been expected in a fall; and*
- *The presence of retinal hemorrhages, which is indicative of an inflicted injury.*

Applying population findings to a single case

This is a major area of controversy because the answer being sought from the forensic pathologist will assist in determining whether the death is intentional or non-intentional.

This controversy arises in the nature of the relevant scientific research and its applicability to a single case rather than arising specifically in the field of forensic pathology.

The nature of this controversy is the interpretation of population-based scientific research and its application to the particular case at hand. Large population studies of childhood injuries on the whole indicate the likelihood of severe accidental head injury from short falls is rare. This is counterpointed by anecdotal case reports that suggest it can happen.

The challenge lies in how and whether this information is applicable to a specific case. For example the death rate from head injury due to short falls is estimated at 1.3 per 100,000 [159]. The difficulty for pathologists, lawyers, and the courts lies in the fact that while the statistical evidence is that fatalities from falls are rare, they do occur. If the phenomenon has occurred once, it can happen again, whatever the statistical weight against its frequency [54].

It could be argued that the role of a prudent pathologist in this situation is to provide a description of the findings and observations, to interpret these and explain the method and limitations of this interpretation.

4. How did you ascertain these features were present?

Forensic Pathologist: *The linear skull fracture and subdural hemorrhage were initially observed in the CT scan. The complete autopsy examination was conducted, confirmed these findings, and found no other significant injuries than those to the head. No significant pre-existing natural disease that could have caused or contributed to death was found.*

Prosecution: *How was the autopsy conducted?*

Forensic Pathologist: *The head was shaved to look for bruises (contusions) on the outside surface of the scalp. The boggy contusion was seen in the area overlying the skull fracture.*

The scalp was incised and reflected to expose the skull and to inspect for hemorrhage and fractures. I made a note of the subcutaneous scalp bruising. The linear fracture was observed to directly correspond to this bruising.

The skull cap was removed, exposing the brain. I directly observed the subdural hematoma. This blood was clotted, there was a small amount of surrounding fluid blood, and had no obvious capsule. It appeared to be an acute subdural hematoma.

The brain was removed and weighed. It appeared swollen.

The eyes were removed and examined following fixation in formalin; I directly observed extensive hemorrhages in the retinas of both eyes.

There was a full dissection of the organs of the chest and abdomen, and these revealed no pathology.

Prosecution: *Was this the full extent of your post-mortem examination?*

Forensic Pathologist: *Yes.*

Prosecution: *Could the presence of any other injuries or disease process have been missed?*

Forensic Pathologist: *It is possible, but given that we are looking to identify all the injuries and disease that are present, I hope that I did not miss any. But I do concede I did not completely dissect under the skin of the arms, legs and back (including the buttocks).*

Prosecution: *Were there any other pathologists present when you were conducting the autopsy?*

Forensic Pathologist: No. The mortuary scientist/technician who assists in the autopsy procedures, together with the police photographer and a representative of the police investigating team, were the other people present.

Prosecution: How did you establish that the subdural hemorrhage was acute?

Forensic Pathologist: There was nothing to suggest it was otherwise. It was a dark red clot with some fluid blood as well, and it showed no signs of healing. I also made a histological section of the subdural hemorrhage and when observed under the microscope I saw only the constituents of blood within it and nothing else. A number of histological sections of the dura showed no signs of healing subdural hemorrhage.

Prosecution: Did you consult a neuropathologist before coming to your conclusion?

Forensic Pathologist: No. In this particular case I did not think it was necessary. Also, it is very difficult to find a neuropathologist who is prepared to undertake these cases because they can be quite disruptive to the normal life of a hospital-based pathologist, which is where most of the small number of neuropathologists work.

Prosecutor: In relation to the retinal hemorrhages, can you describe your technique and what you observed?

Forensic Pathologist: I removed the eyes and, upon dissection, directly observed extensive retinal hemorrhages that extended to the periphery of the retina.

5. Are the described features verifiable?

Prosecution: Do you agree that you should conduct the autopsy in such a way that, as far as possible, another pathologist at another place and time can independently evaluate the critical findings so that s/he can come to his or her own conclusions?

Forensic Pathologist: Yes.

Prosecution: When you conducted the post-mortem examination, what steps did you undertake to ensure the critical findings were reviewable or verifiable?

Forensic Pathologist: The images of the CT scan are available at any time to be viewed electronically. The plain X-rays that were taken prior to autopsy, and which showed no other fractures than the skull fracture, are available for review. When I shaved the scalp I photographed the head and took particular care to photograph the external appearance of the scalp bruise. I also photographed the under-surface of the scalp when it was reflected and the fracture was exposed. In addition, when the top of the skull was removed I photographed the subdural and the brain while it was both in situ and after it was removed to help show the cerebral swelling.

Photographs of the whole body were taken. These showed the bruise to the forehead and the knee, and the absence of other injuries elsewhere, including to the back. All of the histology is available for review.

6. Are the findings beyond the scope of the individual pathologist? If so, who else or what else is being relied on? Is there a clear chain of custody for the samples sent for special testing?

Prosecutor: *Have you relied upon the observations or findings of any other person in making the important observations in this case? (I do not mean in making your conclusions).*

Forensic Pathologist: *Sometimes I do, but in this case, no. If I had relied upon someone else's observations, I would have referred to them in my autopsy report and attached a copy of what they had written to my report.*

Prosecutor: *When you sent the blood and toxicology samples to be tested, how did you ensure the results you received and interpreted related to the samples you sent off?*

Forensic Pathologist: *I followed our usual departmental protocols.*

Prosecutor: *So can you assure the court that you followed the forensic approach where the continuity of the specimen can be assured?*

Forensic Pathologist: *I rely on the system to work after the specimens left my custody at the completion of the autopsy.*

Defence: *Do you have specific training in ophthalmology or ophthalmological pathology?*

Forensic Pathologist: *No. Neither do I know anyone who specializes in ophthalmological pathology. Sometimes neuropathologists have a particular interest in the eyes.*

Defence: *Did you present the findings to a peer for review, or present this case at a meeting of your colleagues to gain the benefit of their views?*

Forensic Pathologist: *No. I work in a solo practice, so this is usually not really feasible, as much as I would like it to be.*

7. Could the observed signs be due to natural disease or injury?

Prosecutor: *Is or are there any normally occurring conditions that would have aggravated the effects of whatever trauma occurred here, or even caused the effects observed here with minimal force?*

Forensic Pathologist: *That, if I may say so, is a very big question. Suffice to say, I saw no signs to suggest to me that this infant was suffering from any tendencies to fracture easily (and as far as I am aware, there is no history of a previous fracture). In addition there is no history of a tendency to bruise easily and there were no obvious signs at autopsy of underlying significant hemorrhagic disease.*

Prosecutor: *What about a mild form of vitamin C deficiency? That might be difficult to test for, and you cannot rule out that this infant might have had that and that would explain a tendency to bleed more easily following any particular force.*

Forensic Pathologist: *As a purely factual matter I can't rule it out, but there is nothing presented to me to rule it in as any sort of reasonable possibility. Vitamin C deficiency of any form would be extremely rare in our developed country. Of course, it does not explain in any way the skull fracture.*

8. Are the observed signs of injury due to intentional harm in this instance?

Prosecutor: *In your report there is mention of a single large bruise on the scalp of the infant and a yellowing bruise on the forehead and two yellowing bruises on the knee. How did you ascertain the absence of any other bruises on the remainder of the body?*

Forensic Pathologist: *I inspected the body thoroughly, paying particular attention to the bony prominences where accidental bruising often occurs as well as all other parts of the body and found no other bruises or contusions.*

Prosecutor: *Did you employ any imaging techniques to identify bruises or trauma that are not visible to the human eye, such as ultraviolet photography, to confirm your findings?*

Forensic Pathologist: *No. Ultra Violet photography is not part of our routine repertoire and I am not aware of it routinely used in any other forensic pathology department, and I am not sure it would be of significant assistance anyway.*

Prosecutor: *If that was the case did you take the step of dissecting in the subcutaneous plane of the skin of the infant to look for any hemorrhage that was not visible on external examination?*

Forensic Pathologist: Yes, as far as I thought was reasonable, because this sort of dissection can create upset. I didn't dissect beneath the skin of the limbs, although I did look beneath some of the skin of the back as a continuation of the process of inspecting the spinal cord.

Cross-examination by defence counsel

Defence: I take it that you accept that it is perfectly possible for bruising to exist that is not visible through the skin. In other words, bruises can be present but you can't see them externally.

Forensic Pathologist: Yes.

Defence: And that this phenomenon might be more significant in relation to the back because the skin is thicker there, and therefore more likely to obscure any subcutaneous bruising.

Forensic Pathologist: Well, I suppose that might be the case. But perhaps the thicker skin also means that bruises are less likely to occur there as well.

Defence: That may be so, but that might also mean it is more important to be sure there are no bruises there rather than just relying on an external examination.

Forensic Pathologist: Mmmm, yes, I suppose.

Defence: So it is quite possible that a bruise that might have occurred to the upper back during a fall of the kind described by the father was present, and not seen by you.

Forensic Pathologist: That might be the case.

Defence: If there had been one or two bruises to the upper back, perhaps on the back of one or both shoulders, and all the other findings were the same, would that change your view about the likelihood of a fall, such as described by the father having occurred? I would like to alert you, before you answer that question, that we have instructed Professor Bloggs, Chief Medical Examiner of Big City, who I am sure is known to you, in this case.

Forensic Pathologist: I must say, if that is so, that I am quite disappointed that Professor Bloggs has not communicated with me. But leaving that aside, I think I might have to agree with you and say that if I had seen one or two bruises to the upper back, perhaps around the back of one or both shoulders, I would have to accept that that would increase in my mind the likelihood of a fall having occurred. Of course one would have to consider the possibility that that was some other fall and not one connected with the head injury.

Defence: *But doctor, you are now splitting hairs. In the circumstances of this case and this trial, because you cannot say there are no bruises to the back, indeed we are entitled to proceed on the basis that there might well have been bruises on the upper back, this would mean that you cannot now rely on their absence, as you did at the beginning of your evidence, as supporting an inflicted nature to the head injury in this case. Is that true?*

Forensic Pathologist: *Yes, you are correct.*

Defence: *Thank you. And I do applaud you for your courage in making the concession you have. The whole justice system relies upon expert witnesses being prepared to change their mind when the need arises.*

Forensic Pathologist: *That is kind of you. I still believe however that the literature is with me in my overall view about this being an inflicted injury.*

Your honour, may I just say that if it is true that Professor Bloggs was going to put the view that I have just conceded, I am disappointed he did not contact me as it would have cut short an unnecessary thread in the case.

Defence: *I can understand you saying that doctor, but Professor Bloggs was under strict instructions not to communicate with you.*

Forensic Pathologist: *Well then, you are lucky that I agreed with you and conceded because if I had not, you would be left with a simple disagreement between experts, albeit a bigger one on your side and that then would have been your responsibility. And, if I conceded now, I would certainly have conceded before the trial and the whole course of the matter may have been different. If I had not conceded, and Professor Bloggs was involved, we may still have been able to resolve the matter.*

Defence: *Moving on now to another topic, what steps did you undertake to exclude any coagulopathy disorders, drug or vaccine reactions?*

Forensic Pathologist: *Post-mortem tests for these are generally not very reliable. I am not aware of any medical history of these problems. In any event, even if there was a bleeding disorder, of which there was no evidence as I saw no other significant bruises (bearing in mind the above discussion, and I do concede that a subdural hemorrhage may be the first manifestation of an underlying bleeding disorder), there remains the issue of the skull fracture. There was no medical history of easy fracturing, and no radiological evidence of any condition predisposing this infant to spontaneous fractures or fractures occurring after minor trauma. There were no fractures of any other part of the skeleton seen. The results of toxicology analyses and microbiology tests revealed no findings of significance. No drugs were detected and no organisms of pathological significance were identified.*

Defence: *Did you check for specifically for vitamin K or C deficiencies?*

Forensic Pathologist: *No, there was insufficient blood left to do any test other than what is considered standard. These deficiencies are extremely rare in otherwise well infants or children.*

Defence: *Then you cannot rule out the presence of some underlying pathology, not limited to the above conditions that could have exacerbated the traumatic injury that you observed?*

Forensic Pathologist: *I saw nothing to positively indicate the existence of relevant natural disease that could have exacerbated the trauma this baby sustained to the head.*

Defence: *That is not an answer to the question I asked.*

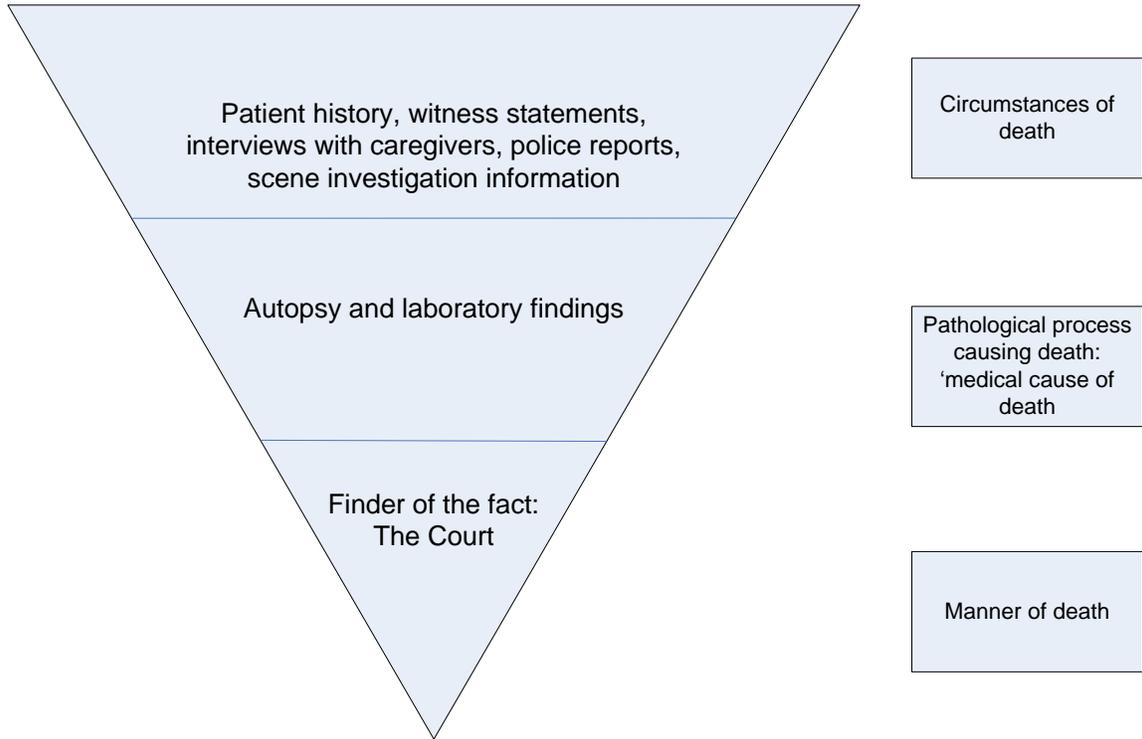
Forensic Pathologist: *I rarely completely rule things out, but such conditions are very rare, especially in children who are otherwise well, and I do not see any particular reason to believe that they have played any part in this death.*

Based simply on the presence of the linear skull fracture, acute subdural hemorrhage, and retinal hemorrhages, and in the absence of any account of the circumstances of the death, a forensic pathologist would be unlikely to be able to differentiate between intentional and unintentional harm.

If there was evidence of multiple other injuries, incompatible with normal, unintentional childhood injury; incompatible with history of injury vintage(s) or child's development; multiple or patterned bruises; old, unexplained fracture(s); findings of cranial impact without history or with inadequate history of impact trauma [59]—that is, the pathological manifestations of abuse—it lends weight that the harm may have been intentional but does not necessarily mean that the fatal injury itself could not have occurred as claimed.

In a criminal matter, most forensic pathologists in English-derived legal systems would agree with the following schema:

Figure 2—Legal system schema



9. How certain are you as the pathologist that the observed signs of injury are due to intentional harm in this instance?

Prosecution: How certain are you, Doctor, that what you saw in this case was the result of inflicted injury?

Forensic Pathologist: Well, based on my reading and understanding of the literature, the likelihood of a short fall causing the fatal injury I observed is extremely small. I am comfortable with my view that this infant died of non-accidental injury.

Prosecution: Comfortable? That's an interesting word, but not, I'm afraid, very useful for the judge or the jury in helping them with the burden and quantum of proof in this case. Can you be a bit more specific?

Forensic Pathologist: I appreciate that, but I have sworn to tell the truth and the word "comfortable" reflects my state of mind. I suppose I could say that the statistical chance in my mind is so small that I really do not have to worry about the possibility of a short fall causing fatal injury.

Prosecution: Well, Doctor, we also have an expert on the literature in this subject; so thank you for your evidence so far today. We will however need to recall you for a few more questions shortly.

Defence: We have an expert too.

10. Have the features of this case ever been described in the literature as being non-intentional?

Prosecution calls an expert witness on falls

Prosecution: Professor Strong, could you please tell us the position you hold?

Witness: I am the Director of the Accident Research Centre of the University of Big City. I have a doctoral background in engineering and statistics.

Prosecution: Could the injuries seen in this infant be due to a short distance fall?

Witness: No— in my opinion these injuries were not the result of a short distance fall such as described by the father.

Numerous studies provide data to suggest death from short distance fall does not occur. In 2006 Pitone and Attia reviewed the records of 787 patients, which included 326 children aged less than 4 years [69]. Falls from chairs and beds were a common cause of injury with infants aged 2 years or less predominantly sustaining head injuries. None of

these infants had intracranial hemorrhages or required neurosurgical intervention. She concluded that routine household falls generate little significant injury and that falls from stairs and furniture are relatively low risk.

Lyons and Oates' review of bed falls [65], studying 207 children aged 5 years and younger who had fallen from bed during a hospital stay, found 31 cases of injury; 29 cases resulted in contusions and small lacerations, and 2 resulted in fractures (clavicle and skull). However, loss of consciousness was not reported in any cases. They concluded that falls from short heights do not typically produce clinically significant injuries.

Helfer's [41] seminal study on hospital falls in children aged 5 years and younger found a total of 85 children who had fallen approximately 0.9 metres. In 57 incidents there was no apparent injury, 17 had small cuts, 20 had a bump or bruise, and 1 child sustained a skull fracture with no serious or apparent sequelae. Their study found a low incidence of fracture and no serious head injuries. The authors conclude that physicians should be suspicious of child abuse if they examine a child with a serious head injury when the cause is reported to be a fall from a bed or sofa.

Defence cross-examines expert witness

Defence: *A couple of points, Professor. As you have just said, the authors simply conclude that physicians should be suspicious of child abuse if the cause is a short fall. Why do you conclude more definitively than the authors of these papers who carefully craft their conclusions to leave open the possibility of exceptions? In addition, the three papers you quote refer to beds, chairs, and falls in hospitals. This was a fall onto a ceramic floor from the father's arms. Doesn't that make a difference?*

Witness: *I think the court could rest quite easily on the conclusion that this is an inflicted injury. It is based on the literature, evidence of the incidence of serious head injury from short falls. Yes, there will be exceptions, but they are very rare.*

Defence: *Professor, you have not answered my second question.*

Witness: *Well, I am not a doctor or a pathologist so it probably would not be right of me to talk about the difference between the conditions of particular falls as to their effect on the outcome in a particular case.*

11. Is the evidence/research base relied on in this case unequivocal and definitive?

What are the known limitations of the research these conclusions are based upon?

Defence calls an expert witness on falls—Professor Wary, Head of the Neurosurgical Unit at the Big City Children's Hospital

Defence: *Could the injuries seen in this infant be due to a short distance fall?*

Witness: *It is possible. The height from which children must fall to sustain fatal head trauma is a question that lacks a single, easy answer [40]. The scientific community has been engaged in a 30-year debate on whether short distance falls can cause serious injury or death. Scientific journals are punctuated by letters to the editor, rebuttals and responses [159-166] contesting the premise that, in rare instances, it can happen. Today it still remains a matter of controversy.*

The factors that may influence the severity of the injury following a fall may include[42]:

- *The distance fallen*
- *The nature of the surface onto which the child falls*
- *Forward or sideways protective reflexes*
- *Whether the fall is in some way “broken”*
- *Whether the child has been propelled or ejected*
- *The mass of the body and the head*
- *The proportion of total energy absorbed in deforming the skull, the brain, and the rest of the body*
- *Whether or not energy is dissipated in causing fractures*
- *Whether the contact with the ground is concentrated to one point or onto a flat surface*

Review papers summarising what is known on these subjects generate conflicting conclusions [40, 43, 44]. Experiments on monkeys and in some cases on deceased infants suggest that a short distance fall is capable of causing serious harm [45-48]. Simulation testing with life-size dummies and computer modelling also assert that serious injury is possible[49-53], although both these study types come with the caveat of not being able to replicate real-life circumstances[43]. Studies of hospital-, community-, and household-based falls in infants have also failed to provide a conclusive answer. Large population studies of childhood injuries on the whole indicate the likelihood of severe head injury is rare. This is counterpointed by anecdotal case reports that suggest it can happen.

The literature shows that manifestations of intentional injuries can almost all occur unintentionally, albeit in rare circumstances. The challenge lies in translating population incidences of these rare events to the particular case at hand. For example, the death rate from head injury due to short falls is estimated at 1.3 per 100,000 [159]. Small population incidences belie the difficulties facing pathologists confronted (in all likelihood) with the one in a million; after all, if it was an unusual or unexpected death, it would most likely be subject to a post-mortem examination.

The difficulty for pathologists, lawyers, and the courts lies in the fact that while the statistical evidence suggests that the potential for fatalities from falls are rare, they do occur. If

the phenomenon has occurred once, it can happen again, whatever the statistical weight against its frequency [54].

Krous and Byard (2005) conclude as follows: “... *it seems quite clear that with the exception of extraordinarily unusual circumstances, short falls rarely cause the death of infants and young children*” [36].

Conflicting Scientific Findings

It could be argued that the role of a prudent pathologist in this situation is to provide a description of the relevant findings and observations only. Accurate interpretation of the autopsy findings requires additional information that is gathered by field investigators and considered by the court. The accepted method for resolving conflicting scientific research is by having a transparent and explicit process that evaluates the strengths and limitations of the studies and their applicability to the question posed. The adversarial arena may not be the best place for such a resolution. In the absence of conclusive evidence a consensus position established by recognized experts is usually required.

12. Would your peers come to the same conclusion based on the observed physical signs detected at autopsy?

The forensic pathologist is recalled.

Prosecution: *Doctor, you mentioned earlier that no other suitably qualified pathologist was present when you were conducting the autopsy*

Forensic Pathologist: *That is correct.*

Prosecution: *Did anyone review your findings?*

Forensic Pathologist: *No.*

Prosecution: *How often has your practice been audited?*

Forensic Pathologist: *Auditing has not been a common practice in forensic pathology, although I understand that a number of centres have introduced this as a requirement. I would like to be involved in such an exercise, but it is time-consuming and it delays even further the release of reports. I am already so snowed under with work that I do not relish the thought of yet another imposition on my time.*

Case Study 2: A newborn baby found dead

In case study 2 the forensic pathologist is very recently qualified and works in isolation in a provincial center. The question at hand is whether the infant was live born, a critical threshold issue in some pediatric forensic pathology cases.

Case History

A 35-year-old woman, separated from her husband and alone at home, delivered a male infant at 16:00 hours on the 10th of October 2007. The mother placed the infant into a plastic bag shortly after birth. She was visited by a friend at approximately 21:00 hours the same day. She told her friend that an ambulance had taken the baby. At approximately 01:00 hours on the 11th of October, the friend found the deceased infant in the laundry. The friend contacted the ambulance service, who attended and located the deceased infant inside a bag within the laundry of the house. Death was confirmed.

The mother told the ambulance staff that she thought she was between 36 and 46 weeks (sic) gestation. She lived alone and cut the cord herself. The mother said the baby made no noise and that she placed it in a plastic bag. The mother said she cut the cord with scissors. The cord was not around the baby's neck. The mother stated that a doctor informed her in August that her baby was dead in utero then. Sometime following the birth, she found the baby amongst the blood, fluid, and feces on the bathroom floor. She did not check for signs of life and did not look at the baby closely but wrapped the baby inside a plastic bag and placed him in the laundry. She had not felt any fetal movements in the days and weeks leading to birth.

At autopsy the infant had no signs of external injury or maceration and showed vernix caseosa over the skin. A short length of the umbilical cord was still attached, the base of which appeared slightly reddened. Petechiae were present on the surface of the heart and lungs. The left and right lungs were mainly pink and floated when placed in water. Histology of the lungs showed variable expansion of the alveolar spaces. For the purpose of discussion, in this particular

jurisdiction, an inquest can also form the function of being a Committal hearing if the Coroner believes a properly instructed jury could convict a named person of a homicide-related offence. Consequently, the mother is represented at the Inquest.

1. What is the cause of death?

Counsel assisting the Coroner: *Doctor, what was the cause of death in this case?*

Forensic Pathologist: *The cause of death was probably asphyxia. The infant may have been smothered, asphyxiated by being placed in the plastic bag or in and amongst blood, fluid, and faeces.*

Counsel assisting the Coroner: *So you are quite satisfied that this baby was born alive?*

Forensic Pathologist: *Well, I think he was probably born alive.*

Counsel assisting the Coroner: *And it is your conclusion that this baby was killed.*

Forensic Pathologist: *Yes, asphyxiated.*

Mother's Counsel: *Before we go too much further, I would like to talk about whether the baby was ever alive in the legal sense of the word.*

Forensic Pathologist: *By which I take it that you mean the baby was completely expelled from the mother and had established respiration and circulation of his blood independently of the mother.*

Mother's Counsel: *Yes, that's what I mean.*

Forensic Pathologist: *Well this is historical territory that pathologists and lawyers have been talking about for more than 100 years. I think the baby breathed because the lungs were pink, fluffy, and floated in water. It is also possible they may have floated due to putrefaction, but I believe this was not the case in this instance. I do not know, and no one can know whether this baby had a foot or a leg or even more of its body in the birth canal when it expired. If it did, then this was not a live birth but a stillbirth. Mother could well be exhausted, lying on the floor, and baby is lying face down in the fluid, blood, and feces and the physical obstruction to breathing causes his death. The lungs are pink and fluffy because he breathed for some minutes but his legs are still in the birth canal. Clearly however, it could have been a live birth and a range of causes of death are possible.*

The key issue here is whether the child was born alive and if so, whether he was killed, perhaps smothered by the mother, or died following placement in the plastic bag. If the baby has been killed, the mother could be charged with murder or infanticide.

The signs of live birth are few. They include changes to the lungs and the umbilical cord; injuries that cannot be ascribed to labour and appear to have occurred during life; the presence of material in the airways or digestive tract (such as milk) that could only happen after birth.

A live birth requires, in many jurisdictions:

- Complete expulsion from the mother; and
- A separate existence from the mother, usually taken to mean an established circulation and respiration.

2. What are the typical or classical or key physical signs found at autopsy in this case?

Counsel assisting the Coroner: *How have you come to this conclusion?*

Forensic Pathologist: *The lungs of the infant were inflated indicating the infant had inhaled air and the stump of the umbilical cord showed signs of redness, both suggesting she was born alive. There were small petechial hemorrhages on the surfaces of the lungs and heart, consistent with the child dying from asphyxia.*

In addition, the mother did not inform the authorities of the birth of the child and lied to her friend as to the whereabouts of the child, suggesting she had something to hide.

Mother's Counsel: *Are you aware that some pathologists do not accept that the petechial hemorrhages you refer to are even a sign of live birth let alone represent a sign of "mechanical asphyxia"?*

Forensic Pathologist: *No I must admit I had not thought of that, but now that you say it I can accept that. What is the reference for that?*

Counsel assisting the Coroner: *I am sure Counsel will provide you with that in due course. Is the behaviour of the mother after the event of any relevance in how the infant died?*

Forensic Pathologist: *Not specifically. But her behaviour was definitely suspicious and it did focus my mind at the time of the autopsy. I was particularly careful to look for signs indicative of compression of the mouth and nose or perhaps even of blunt trauma.*

Bias and decision making

The controversy is recognition and understanding that bias exists in how a forensic pathologist approaches, conducts, evaluates, and presents the cause of death and whether this is due to intentional harm.

Bias is not unique to forensic pathology but affects all human decision making. There is extensive literature on cognitive, behavioural, and social decision making. Much of the literature in medical sciences has concentrated on clinical practice. There is no reason to suppose that forensic pathology would be immune from biases. One would reasonably expect that similar biases might apply in pediatric forensic pathology and forensic pathology generally.

Different types of bias can affect each stage of the forensic pathology process. Prior to autopsy the provision of information may create a framing bias[167], because too narrow a description of the circumstances of the death was presented to the pathologist. For example, a previously healthy child from a middle-class, church-going family who are known for their community volunteer work have their 4-month-old child die suddenly. The family express concerns about a full autopsy, the police state there are no suspicious circumstances, external examination shows no injuries; subsequently the pathologist is persuaded to limit the extent of his/her investigation, knowing that subdural hemorrhage could be present for example, with no external sign.

Selection bias occurs when a conclusion is drawn from selective sampling [167]. This may occur in an autopsy setting by limiting the extent of the autopsy or limiting the amount of tissue obtained for examination, for example, one histological section for a large bruise is taken and a conclusion made about the age of that bruise from that section. Another example of selection bias is when complex or intentional cases are always assigned to a particular pathologist. This creates a history that the majority of cases are intentional injury and may become self-fulfilling.

Confirmation bias is defined by the tendency to interpret information so that it confirms one's preconceived ideas [168]. For example a pathologist who is conducting an autopsy where he or she suspects the infant was abused, who has injuries that may be related to resuscitation, may be more likely to interpret these injuries as being intentional rather than due to resuscitation attempts. The reverse confirmation bias can also occur with the same fact situation.

The halo effect is defined by the assumption that a person's positive or negative traits transfer from one area to another[169]. For example a jury may be persuaded to believe a highly technical and specialist pediatric forensic pathology opinion from a doctor who is a respected member of the community with limited expertise in pathology.

This controversy arises in the decision-making process faced by the pathologist. The nature of this controversy is that, although decision making is known to be flawed and this can occur in situations of high stress, heavy workload, and unfamiliar circumstances,

this is not formally or explicitly recognized. Many people have proposed that information should be kept from the pathologist so that unbiased opinions are provided. This is to conceive of forensic pathology wrongly and fails to understand the nature of bias. Such a withholding of information would, at the very best, introduce another bias or biases that may be more elusive. In the vast majority of cases, the pathologist's findings can only be interpreted within the context of the medical and circumstantial history. It could be argued that the role of the pathologist in this situation is to ensure that they, their peers, the court, and community are aware of bias and steps have been taken to mitigate these as best as one can.

Forensic pathology is a problem-orientated exercise. It exists to produce answers, and unless there are questions, and these questions derive from the particular circumstances, forensic pathology will be of relatively little use.

3. What are the physical signs found at autopsy in this case that led you to the conclusion that this was the cause of death?

(These are implicit in the remaining sections.)

4. How did you ascertain these features were present?

Counsel assisting the Coroner: Please tell the court the steps you took to confirm your findings and how you conducted the autopsy.

Forensic Pathologist: Unfortunately our CT scanner was out of commission at the time and we performed a babygram (a single X-ray image of the infant) that confirmed the expansion of the lungs with air and no obvious fractures. The autopsy was performed to identify any underlying disease and to try to ascertain the cause of death.

The scalp was reflected and showed a caput succedaneum, such as is commonly seen in newborns. This indicates that the fetus was indeed alive during labour at the time the head was engaged with the pelvic floor and there was a rim of dilated cervix around the presenting or lowest part. There was no evidence of any damage to the skull or subgaleal hematoma (cephalhematoma). The skull was removed and there was no evidence of hemorrhage on the surface of the brain.

The torso was opened in the usual way. The soft tissues in front of the chest were then reflected back. The breastbone and attached rib cartilages are removed exposing the heart and lungs. The internal organs were then individually removed and weighed [170]. The subcutaneous tissues were inspected to check for signs of bruising or hemorrhage.

Counsel assisting the Coroner: How can you be sure the infant didn't die in utero as the mother has suggested or during delivery?

Forensic Pathologist: A fetus dying in utero and remaining in utero for a period such as 24 hours or more acquires a particular appearance referred to as maceration. This

appearance is the result of the breakdown of the tissues as a consequence of the cessation of the circulation. The failure to deliver oxygen and nutrients means that the tissues of the body die. Under the influence of existing enzymes, the tissues break down. The appearance is different to decomposition and putrefaction in other deaths.

When I conducted the autopsy I saw no evidence of maceration.

Counsel assisting the Coroner: *How can you be sure the infant was born alive?*

Forensic Pathologist: *The lungs were pink and inflated and floated when I applied the hydrostatic test, and the stump of the umbilical cord showed what I believe were signs of redness. When a baby is born alive the lungs are distended, well aerated, fluffy, have a salmon-pink colour, and are visible in the front of the chest when the sternum is removed. The lungs of the stillborn baby are collapsed, have the consistency of a flaccid spleen, and are dark purplish-red in colour. They are in the back of the chest and only a small portion is visible from the front [92].*

Counsel assisting the Coroner: *You mentioned the area around the attachment of the umbilical cord was reddened. How did you determine this?*

Forensic Pathologist: *Well, it appeared reddened. But in preparation for this inquest, I now realize that this is a separate sign of survival that appears after a day or so after birth and I would not want to be thought of as suggesting that. It is probably not of significance in relation to the issues we are discussing here.*

5. Are the described features verifiable?

Counsel assisting the Coroner: *Doctor, we do not appear to have any photographs of the lungs.*

Forensic Pathologist: *Yes, I am sorry about that, but I am in no doubt about my findings. I did not think it was necessary to take the photos. Actually as I recall, I think we did take some photos but for technical reasons they did not develop properly.*

Counsel assisting the Coroner: *I suppose then we have to proceed on the basis that we will not be able to get any independent view about the appearance of the lungs.*

Forensic Pathologist: *Well, I am afraid so, except that there is the histology which is consistent with what I saw. But the histology alone, I know, cannot wear the weight of the conclusion that the baby had some form of established respiration.*

6. Are the findings beyond the scope of the individual pathologist? If so who are/what is being relied on? Is there a clear chain or documentation of how the specimens were handled?

Counsel assisting the Coroner: *How long have you been a forensic pathologist?*

Forensic Pathologist: *I have been doing autopsies for the coroner for five years, the last two as a qualified forensic pathologist.*

Counsel assisting the Coroner: *So you've only been a specialist for two years. How many neonatal autopsies have you done where the issue has been whether the baby was born alive or whether it was killed?*

Forensic Pathologist: *This is the third or fourth I think.*

Counsel assisting the Coroner: *So you have effectively had very little experience in such deaths.*

Forensic Pathologist: *Well you could say that but you need to realize that such deaths are quite uncommon and no one pathologist has lots of experience these days in such deaths.*

Secondly, becoming a specialist forensic pathologist means that we learn about these deaths in our training and this is an absolutely classic situation that every forensic pathologist is expected to know about.

Thirdly, we apply a basic set of standard approaches to all cases so the analogy really is like that of a driver driving for the second time up a particular bit of tricky highway. He does not know the particular highway very well, but he knows how to drive and in the circumstance, drive carefully. The same applies to me in doing an autopsy.

Defence: *Did you discuss this with a senior colleague?*

Forensic Pathologist: *No, that is not possible. There is only one forensic pathologist in this city.*

Defence: *Could you not have discussed this with colleagues in the capital?*

Forensic Pathologist: *I suppose so but I didn't think it was necessary. The Coroner was happy with my findings. Indeed, it was the Coroner that asked me to do the autopsy in the first place. I think in future it would be a good idea to involve a pediatric or a neonatal pathologist in an autopsy such as this. We could produce a joint report that would combine both the forensic and more specialist neonatal issues.*

The determination of cause of death was not assisted in this case by the input of other specialists. What is critical is that a comprehensive and thorough approach to the case is

undertaken. One might argue that an experienced pediatric (forensic) pathologist should have been involved because the final determination of the cause of death has such significant implications.

7. Could the observed signs be due to natural disease or injury? Please explain why it could or could not be due to natural disease processes.

Mother's Counsel: *Is it true that some lungs may inflate or float when the child has not been born alive?*

Mother's Expert Forensic Pathologist: *Yes, the child might have breathed before being completely expelled from the birth passage, or there might be some putrefaction, or even some forms of handling the body have been reported to possibly introduce air into the lungs.*

Mother's Counsel: *The mother reported not hearing the baby cry. Does this have any significance?*

Mother's Expert Forensic Pathologist: *The baby being heard to cry would raise the serious possibility of the baby being born alive; however, technically speaking, an infant can cry prior to complete expulsion and therefore may have, in the process of being born, breathed and then died before complete expulsion or died subsequent to being born by being smothered or overlaid accidentally by an exhausted mother.*

Mother's Counsel: *Can there be any alternative explanations for the expansion of the lungs?*

Mother's Expert Forensic Pathologist: *The slightest degree of decomposition or resuscitation or it is said even ordinary handling of the deceased may aspirate some air into the lungs. The problem is complicated further by a minimally respired lung, where the changes are intermediate. There are too many recorded instances when tests have shown that stillborn lungs may float and the lungs from undoubtedly live-born infants have sunk to allow it to be used in testimony in a criminal trial. I believe any doubts must be resolved in the direction of no breathing and, even in doubtful instances when the pathologist decides on balance that respiration has occurred, he should convey this uncertainty in the body of his report.*

Absence of definitive scientific research

The nature of this controversy is in the absence of definitive scientific research. Individual pathologists may interpret their findings in either direction. Historically the scientific research suggested the ventilation of the lungs and the floatation test of lung tissue was the basis for establishing the infant had breathed and therefore had an existence independent of the mother. However, according to the present level of knowledge, ventilation of the lungs alone cannot be taken as a certain indication of a live birth. One needs to know the legal definition of a live birth to assist in a proper evaluation here. In addition, under various circumstances, lungs originally aerated can become devoid of air; conversely, the lungs of stillborn neonates can appear aerated. Therefore it is not possible to be certain in all cases [171].

It could be argued that the role of the prudent pathologist in this situation is to provide a description of their findings and observations only and to provide general technical information that is readily available from the accepted international body of forensic knowledge. To answer the question of whether or not an infant had a definitive and separate existence, i.e., was live born, requires pre-autopsy information from field investigators and interpretation of the veracity of that information by the court, not the pathologist. If matters of criminal culpability are at stake, this is a function of the court.

8. Are the observed signs of injury due to intentional harm in this instance?

Counsel assisting the Coroner: In your opinion what happened here?

Forensic Pathologist: Based on the autopsy findings I cannot determine whether the infant was stillborn or was legally born alive and died of natural causes, or from birth trauma, or an accident at birth, or subsequently from a criminal act.

The only way to determine the manner of death would be some form of evidence that indicates the probability of a crime and a police investigation proving that the death was intentional[92]. Medicine and forensic pathology cannot provide you with this conclusion.

9. How certain are you as the pathologist that the observed signs of injury are due to intentional harm in this instance?

(Not relevant following response above.)

10. Have the features of this case ever been described in the literature as being non-intentional

(Not relevant following the response above.)

11. Is the evidence/research base relied on in this case unequivocal and definitive?

Counsel assisting the Coroner: *It strikes me that you have had previous little forensic pathology research to guide you in this case.*

Forensic Pathologist: *Well, I think you are right. There is quite a lot in the textbooks over the years, but not much in the contemporary forensic pathology literature that I could see.*

12. Would your peers come to the same conclusion based on the observed physical signs detected at autopsy?

Counsel assisting the Coroner: *Doctor, what was the cause of death in this case?*

Forensic Pathologist: *Well, on reflection and taking into account our discussion and what else I have heard today I think forensic pathologists would coalesce around the following formulation: From the autopsy findings I could not say if the baby who was found dead, died during, or shortly after birth. Because of this, assigning a particular cause of death becomes near impossible. Formally, I gave the cause of death as “unascertained in a newly born baby found wrapped in a plastic bag.”*

Case Study 3: A battered baby

In case study 3, the pathologist is very clear in his own mind as to the cause of death and is well experienced in the court room. He works in a team environment and has performed a good reviewable autopsy. The question is whether the defacto father had inflicted the immediately fatal injuries, complicated by the presence of much older injuries for whom there were a number of possible assailants.

History Provided by Caregivers

From 13:00–16:00 hours, C.D., a 4-month-old boy, was in the sole care of his mother. At 16:00 she went to work, handing the care of the child to the de facto father, who had arrived home. The father reported the boy had been hot, not feeding, vomiting, and crying on and off after he took care of the boy, and he put C.D. down in the cot at 17:00. When the mother arrived home from her part-time work at 21:00, she found the father desperately trying to resuscitate C.D., thumping his chest, since he had apparently shortly before just stopped breathing. The mother called the emergency number and was given instructions for resuscitation. Ambulance services were dispatched and arrived at 21:15. C.D. was unconscious. Emergency services staff continued administering resuscitation and transported the infant to the hospital. He was announced dead on arrival. Parents reported C.D. had been unwell, not taking his food, and vomiting for the past three days.

The Coroner ordered an autopsy, which was performed the next day despite the mother and de facto husband formally objecting to autopsy, saying they did not want C.D. to suffer any more. They did not pursue their objection to a higher court.

Autopsy

The post-mortem report indicated the presence of a healing skull fracture but no detectable residuum of intracranial trauma, recent anterior rib fractures with little or no hemorrhage

associated and two old healing rib fractures posteriorly, recent rupture of the small bowel at the junction of the duodenum with the proximal jejunum with purulent peritonitis, old trauma to the liver, and numerous bruises to the face, trunk, and buttocks

Post-Autopsy Enquiries

After the autopsy the forensic pathologist was shown photographs of C.D.'s home. The house was very untidy and the kitchen was filthy. There were many empty bottles of alcohol around, as well as cigarette butts all over the place. The pathologist then made notes of a briefing provided by police: *“Police informed me that the de facto father initially claimed no knowledge of the bruises, the skull or rib fractures or abdominal injury, or any idea of how they might have been caused. The mother also claimed no knowledge of any injuries. She herself apparently had a number of bruises to her arms and an obvious black eye which were noted by the attending police.”*

Later, the parents each stated to police that the rib fractures and facial bruising may have occurred during resuscitation attempts. The fractured skull must have occurred, said the de facto father, when mother dropped the baby a month or so previously during a heavy drinking party at their house. They also stated that quite a lot of babysitting was undertaken by friends and relatives in the weeks preceding the boy's death.

There had been one hospital visit four weeks previously, and appointments for follow-up and for various tests had not been kept. Unopened letters from the hospital were found at the house. The hospital had not felt the need to inform the child protection agency because it did not feel the child was at any particular risk. The medical file stated the child had presented at the hospital with sniffles and lethargy.

When the police told the parents that the boy had died from blunt trauma to the abdomen, which must have occurred in the hours before death, they each denied any knowledge of how that

might have occurred. Mother became very upset and the de facto husband started shouting at the mother, saying what a hopeless mother she was.

Two weeks later, the police charged the de facto father with homicide. The following transcript is of proceedings from the Committal hearing. This type of hearing is attended by the forensic pathologist and other witnesses. There is a prosecution and defence and the hearing is presided over by a Magistrate, in order to determine if there are sufficient grounds to proceed with a trial involving a jury.

Forensic pathologist's role in scene investigation

The controversy is whether a forensic pathologist should seek scene information and be provided with witness information prior to, during, or after the autopsy to assist in determining the cause and contributing to the reconstruction of the circumstances of death.

This is an area of controversy because some argue that the opinions being sought from the forensic pathologist may be biased if s/he has information that may wrongly taint his/her perception of the death.

The controversy arises because the opinions required from the forensic pathologist are expected to be objective, based on verifiable observations, and confined to the boundaries of their skills and training.

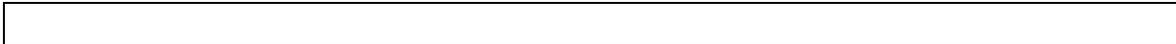
One possible bias would be evident by simply seeking information to support a particular view rather than actually seeking all information.

It could be argued that the prudent forensic pathologist would seek training and endeavour to become skilled in scene examination and would limit him/herself to obtaining witness information from trained police and other investigators.

1. What is the cause of death?

Prosecution: Doctor, what was the cause of death in this case?

Forensic Pathologist: The boy died from septic shock following purulent peritonitis, which followed the spillage of bowel contents into the peritoneal cavity from a traumatically ruptured small bowel. The purulent peritonitis is an infection that quickly spread to his bloodstream, and his body went into septic shock, which can be thought of as the body being overwhelmed by the infection, from which the boy succumbed.



2. What are the typical or classical or key physical signs found at autopsy in this case?

Prosecution: *In your opinion what was the cause of the purulent peritonitis?*

Forensic Pathologist: *The traumatically ruptured small bowel caused the purulent peritonitis. The ruptured small bowel was due to blunt trauma, probably the result of the bowel being caught forcefully and suddenly between the sternum at the front and the vertebral column behind.*

Prosecution: *What could have caused the rupturing of the small bowel?*

Forensic Pathologist: *It could have occurred in a number of ways, say if the boy was involved in a motor vehicle accident, if he had fallen very forcefully on his abdomen against a raised object or had received a blow to the abdomen, such as with a fist. But that is not to say these are the only ways.*

Prosecution: *How do you think the injury was inflicted?*

Forensic Pathologist: *The classic cause for purulent peritonitis from ruptured jejunum in an infant is a blow, or repeated blows to the abdomen, with a fist. I think the same probably occurred here.*

Prosecution: *What made you come to that conclusion?*

Forensic Pathologist: *The parents apparently gave no history of the boy being involved in a motor vehicle collision or any other major abdominal trauma for that matter. The blow to the abdomen must occur in such a way that the carer would be aware that something serious had happened to the baby, because a 4-month-old baby is more or less immobile.*

In addition, the child had several different injuries of different ages. This looks like a textbook case of a battered baby. This child's death was obviously the result of abuse as anyone can see with all these injuries in a 4-month-old baby who is incapable of any significant movement on its own accord [172].

Magistrate intervenes

Magistrate: *We have to be careful here, doctor. We are having this hearing to see if this man should be sent for trial. Part of my job is to work out what injuries are relevant in considering this boy's death. If I understand you correctly, the old injuries such as the fractured ribs at the back and the virtually healed skull fracture have nothing to do with the boy's death. If so, they may be distracting to a jury and prejudice them against the accused and distort their approach to the fatal injuries, which is what the accused is charged with having inflicted.*

Forensic Pathologist: *I can see the logic of what you say, but you need to know that as far as I am concerned this child is a battered baby. The presence of multiple injuries of varying age in circumstances where there is no satisfactory explanation for how they might have occurred is classic of a battered baby and I believe this should be given due weight. Strictly speaking though, you are correct. The fatal injury from a purely biological point of view is simply and solely the ruptured bowel.*

Did the baby die from or with the injuries?

When considering if a child died from abuse a forensic pathologist will come across four distinct possibilities. Distinctive examples are provided for the sake of clarity, although in practice the distinction between the four categories is less obvious.

A: The child did not die of abuse and had no history of abuse in life. For example the child dies from a bleeding disorder. Some bleeding disorders cause bruising and subdural hemorrhage. The role of the pathologist is to distinguish the natural disease process (which mimics the symptoms of abuse) from actual abuse.

B: The child dies from injuries that were caused by abuse, but had no previous history of abuse in life. For example an otherwise healthy child dies after a single episode of abuse at the hands of a perpetrator. In these cases the role of the forensic pathologist is to describe injuries, evaluate them from the point of view of possible abuse, and estimate their timing (bearing in mind serious imprecisions with this) to assist investigators to identify/exclude potential suspects.

C: An abused child with burn injuries and fractures dies from a non-abuse cause of death, for example, a motor vehicle collision. The pathologist's findings that the child was abused can be distressing and may have implications for the carers both in a criminal and family court setting but are unrelated to the cause of death.

D: A chronically abused child dies from injuries that are the result of abuse. This could include complications resulting directly from the abuse. These can occur at different grades of association. The most obvious is a child who dies from internal bleeding as a result of being beaten. A second example is the child who dies from sepsis due to the caretaker's failure to seek medical attention for inflicted burns. The third, less obvious example, is the child who dies from an unrelated infection that was the result of being relatively immune-depressed as a consequence of general neglect.

In this case study, the infant clearly falls into this final category. He had a history of abuse and died as a direct result of receiving a blow or blows to the abdomen.

3. What are the physical signs found at autopsy in this case that led you to the conclusion that this was the cause of death?

Defence: Doctor, my client states that he has no knowledge of the previous injuries to the infant. Both he and the child's mother have given sworn statements that they have not intentionally inflicted injuries to the child. They also state that they often left the child in the care of friends and relatives who were, to use their own words "rough with the kid." Could you establish a time frame for the previous injuries the infant had sustained?

Forensic Pathologist: On external examination there were several bruises on the face around the lips and cheeks. A large bruise was observed on the buttocks with the outline shape of a large handprint. Bruising was also present on the abdomen and the upper arms. The boy was below the 10th percentile for height and weight for a boy of his age.

The skull fracture was resolving and almost healed so I estimate the time of the initial injury was of the order of four to six weeks previously. It could have been more, or even slightly less. The liver trauma and healing fractured ribs were probably more than two weeks old.

Defence: What about the bruising? Can you give us an age of the bruises?

Forensic Pathologist: All the bruises (other than the abdomen), including the apparent spanking injury, although its seriousness is such as to render the word spanking a serious understatement of the forces involved, showed elements of aging histologically (either well-established acute inflammation and/or granulation tissue and fibrosis) so certainly the latter group probably occurred in a time frame different to the time associated with the rupturing of the bowel and the course from there to death.

Multiple injuries of different ages, incompatible with normal, unintentional childhood injury or the history of injury, is regarded as definitive abuse [59]. However, definitively establishing the age of injuries is a controversial topic in forensic pathology.

The age of a bruise cannot be established based on an assessment of the colour of the bruise with the naked eye. The accuracy of observers who estimate the age of a bruise visually is no better than 50%. A practitioner who offers a definitive estimate of the age of a bruise in a child by assessment with the naked eye is doing so from their own experience without adequate published evidence [173].

In addition to the age of the injuries in identifying child abuse, forensic pathologists also draw on the pattern of injuries in determining whether the bruise occurred accidentally or not. Pattern means, in this context, the number and distribution of injuries. Bruises of uniform appearance, especially on the face and neck, are suggestive of abuse[174]. Abusive bruises often occur on “softer” parts of the body, such as the abdomen, back, and buttocks. Clusters of bruises are a common feature in abused children. These are often on the upper arm, outside of the thigh, or on the trunk. Abusive bruises can carry the imprint of the implement used or the hand [175]. Rib fractures have been reported in the pediatric literature to be a rare complication of cardio-pulmonary resuscitation (CPR), occurring in 3 out of 923 children studied. The fractures caused by CPR were all multiple and anterior, with no posterior fractures reported [176]. Rib fractures in the absence of a history of major trauma, birth trauma, or underlying bone disease has a high specificity for abuse, with a positive predictive value of 84% [176].

4. How did you ascertain these features were present?

Prosecution: *Can you tell me the steps you took to identify the injuries present in the infant?*

Forensic Pathologist: *I made an incision into the scalp and reflected it to expose the skull and to inspect for hemorrhages and fractures. The skull fracture was observed. The fracture was not separated, displaced, or depressed. The skull was opened by incising along the sutures. The brain and dura appeared normal with no obvious subdural or subarachnoid hemorrhage.*

I removed the brain and placed it immediately into formalin. The eyes were removed and examined following fixation in formalin; there were no abnormalities seen.

The skin of the neck and face was then reflected back. Hemorrhages were observed, directly corresponding to the bruises seen externally on the skin, and an additional fresh 0.5 cm area of bruising of the chin was also seen.

The chest and abdomen were then opened in the usual way. The infant had four fractured left anterior ribs showing no healing and with little in the way of hemorrhage, and two old healing fractures on the left posteriorly adjacent to the midline with callus formation and no hemorrhage evident. There was no injury to the lungs or heart, which were normal.

In the abdomen there were extensive fibrino-purulent bands between loops of bowel themselves, and between the bowel, liver, and abdominal wall, as well as what appeared to be bowel contents.

Upon dissection, there was hemorrhage around the duodeno-jejunal junction and about 20% of the circumference of the bowel wall in this region was ruptured. Hemorrhage extended into the mesentery in this region.

I reflected the skin of the arms, legs, and back, including the buttocks. There were bruises to the trunk and buttocks, which varied in age histologically from recent to old.

Prosecution: *Were there any other pathologists present when you were conducting the autopsy?*

Forensic Pathologist: *Yes, the pediatric pathologist who specializes in child and infant autopsies assisted me with the autopsy.*

5. Are the described features verifiable?

Prosecution: *Are your observations independently verifiable?*

Forensic Pathologist: *Yes, the following records exist:*

- Full skeletal survey;
- Whole body CT scan;
- Photographs taken externally and of the important internal findings; and
- Histological slides.

6. Are the findings beyond the scope of the individual pathologist? If so who are/what is being relied on? Is there a clear chain or documentation of how the specimens were handled?

Prosecution: *Are your findings beyond the scope of an individual forensic pathologist such as yourself?*

Forensic Pathologist: *In this case, I do not believe so, but it was very useful to have a pediatric pathologist present. You never know what you are going to come across, so the comfort of having a pediatric pathologist at least readily available, if not already present, is considerable. In our institution it isn't always possible for him to give up the time and there is only the one pediatric pathologist in our town. If he cannot actually attend, I can usually work around it with photos organizing to retain organs or tissue, checking the histological findings, and discussing the case with him. He, of course, is delighted that I am happy to deal with the police and lawyers and take the lead in court,*

although he knows you might want him to give detailed evidence too, I suspect if you do he will defer to my opinion in matters of core forensic pathology just as I would defer to him in areas of pediatric anatomical pathology.

7. Could the observed signs be due to natural disease or injury?

Prosecution: Doctor, could these injuries be due to natural disease processes?

Forensic Pathologist: There was no evidence radiologically of any disease rendering fracturing more likely. There was no indication of any underlying bleeding disorder to render the bruises more likely to occur from lesser degrees of trauma.

8. Are the observed signs of injury due to intentional harm in this instance?

The multiplicity of injuries and their varying age, in the absence of any understanding of how these might have occurred from the caregivers, is diagnostic of abuse. Even if one allows that the babysitter eight weeks previously may be responsible for both the skull and rib fractures and the old liver injuries, one is left with both the bruising to the buttock and the ruptured small bowel. The caregivers, one or both of them, must know how these occurred since the 4-month-old is barely mobile. The pathologist's view is that even taken alone, these are inflicted injuries until s/he is presented by someone with a plausible alternative.

9. How certain are you as the pathologist that the observed signs of injury are due to intentional harm in this instance?

The pathologist's opinion is that until s/he is presented with a plausible alternative, the ruptured bowel and bruising to the buttock represented non-accidental injury.

10. Have the features of this case ever been described in the literature as being non-intentional?

The pathologist is not aware of literature describing ruptured bowel, in this location in an infant, with mesenteric hemorrhage and overlying bruising presenting as purulent peritonitis being due to anything other than trauma.

11. Is the evidence/research base relied on in this case unequivocal and definitive?

(Not relevant.)

12. Would your peers come to the same conclusion based on the observed physical signs detected at autopsy?

Yes.

Conclusion to the Case Studies

These case studies are designed to draw out different perspectives and illustrate the complexities of pediatric forensic pathology. They do not necessarily reflect best practice or the norms. The cases studies and dialogue in the questions and responses are a composite based on research literature and experience.

The twelve questions are a framework to assist in an analysis of the cases. The questions are sequenced to lead the reader into areas of ever increasing complexity. The questions are also designed to dramatically and directly address the key issues and decisions faced by a forensic pathologist. The three case studies were designed individually and collectively to illustrate the limits of forensic pathology. For example, Case Study 1 demonstrates the limitation of scientific research, the nature of conflicting research studies and the limits in applying studies about populations to individual cases. Case Study 2 demonstrates the limitations of the forensic pathologist, the limitations of the autopsy to determine the cause and manner of death and illustrates how bias is inherent in this work. Case Study 3 demonstrates the issues of the aging of injury, the need for information from the scene and about the circumstances of the death, and raises the question of whether a baby died because of or with evidence of intentional harm.

The tables in Appendix 4 summarise the challenges related to the practice of pediatric forensic pathology compared to adult forensic pathology. Appendix 5 sets out some of the controversies not touched on in this paper.

Appendix 1—Medical Specialist Practice and Colleges

Field of Specialist Practice	Subspecialties	Specialist College Responsible for Assessment
Anaesthesia	- Pain Medicine	Australian and New Zealand College of Anaesthetists
Dermatology		Australasian College of Dermatologists
Emergency Medicine		Australasian College for Emergency Medicine
General Practice	Note: Not recognised by all State or Territory Medical Boards	Royal Australian College of General Practitioners
Intensive Care Medicine and Pediatric Intensive Care Medicine		Joint Faculty of Intensive Care Medicine/Royal Australasian College of Physicians and Australian and New Zealand College of Anaesthetists
Internal Medicine	<ul style="list-style-type: none"> - General Medicine - Cardiology - Clinical Genetics - Haematology - Immunology and Allergy - Clinical Pharmacology - Endocrinology - Gastroenterology and Hepatology - Geriatric Medicine - Infectious Diseases - Medical Oncology - Nephrology - Neurology - Nuclear Medicine - Rheumatology - Sleep Medicine - Thoracic Medicine 	Adult Medicine Division, Royal Australasian College of Physicians
Medical Administration		Royal Australasian College of Medical Administrators
Obstetrics and Gynaecology	<ul style="list-style-type: none"> - Gynaecological Oncology - Maternal-Fetal Medicine - Obstetrics and Gynaecology - Obstetric and Gynaecological Ultrasound - Reproductive Endocrinology and Infertility - Urogynaecology 	Royal Australian and New Zealand College of Obstetricians and Gynaecologists
Occupational Medicine		Australasian Faculty of Occupational Medicine Royal Australasian College of Physicians
Ophthalmology		Royal Australian and New Zealand College of Ophthalmologists
Pediatrics and Child Health	<ul style="list-style-type: none"> - Community Child Health - Neonatology and Perinatology - Pediatric Emergency Medicine - Pediatric Rehabilitation Medicine - Pediatric Subspecialties (as for Internal Medicine) 	Pediatrics and Child Health Division, Royal Australasian College of Physicians
Palliative Medicine		Australasian Chapter of Palliative Medicine Royal Australasian College of Physicians

Field of Specialist Practice	Subspecialties	Specialist College Responsible for Assessment
Pathology	<ul style="list-style-type: none"> - General Pathology - Anatomical Pathology (including Cytopathology, neuropathology, pediatric pathology, neonatal pathology) <u>Forensic Pathology</u> - Clinical Chemistry - Genetics - Haematology - Immunology - Microbiology 	Royal College of Pathologists of Australasia
Psychiatry		Royal Australian and New Zealand College of Psychiatrists
Public Health Medicine		Australasian Faculty of Public Health Medicine Royal Australasian College of Physicians
Radiology	<ul style="list-style-type: none"> - Diagnostic Radiology - Diagnostic Ultrasound - Nuclear Medicine - Radiation Oncology 	Royal Australian and New Zealand College of Radiologists
Rehabilitation Medicine		Australasian Faculty of Rehabilitation Medicine Royal Australasian College of Physicians
Surgery	<ul style="list-style-type: none"> - General Surgery - Cardiothoracic Surgery - Neurosurgery - Orthopaedic Surgery - Otolaryngology—head and neck surgery - Pediatric Surgery - Plastic and Reconstructive Surgery - Urology - Vascular Surgery 	Royal Australasian College of Surgeons

Source: Adapted from the Australian Medical Council.

Appendix 2—Fractures—Welsh Child Protection Systematic Review Group

Fractures are a normal part of growing up. This information is based on a systematic review of all the quality work in the world literature about fractures in children prepared by the Welsh Child Protection Systematic Review Group.¹⁹

Accidental fractures are common in children: up to 66% of boys and around 40% of girls will sustain a fracture by their 15th birthday. An estimated 85% of accidental fractures are seen in children over five years of age. However, they can also be indicative of abuse. Abusive fractures indicate a serious assault on a child.

What do we know about fractures in child abuse?

Fractures occur in up to 25% of physically abused children; 80% of these fractures are in children under 18 months. Any bone in the body can be broken as a result of child abuse. Many abusive fractures are not clinically obvious unless X-rays are taken, especially in infants under two years. Fractures, particularly rib fractures, may not be accompanied by bruising.

How do you know if a child has a fracture?

Fractures in very young children may present with non-specific symptoms and may only be revealed by X-ray or other radiological tests. Fractures may not be obvious even on X-ray immediately after the injury; they are easier to identify once the bones show some signs of healing.

¹⁹ The Welsh Child Protection Systematic Review Group (CORE) is a systematic review for the diagnosis of physical child abuse. It is not specifically aimed at pathologists.

How do you find the fractures?

Abused children frequently have multiple fractures and these may be of different ages. Where physical abuse is suspected, specialized X-rays should always be taken of children under two years and may need to be taken of some older children. Two types of X-ray may be used.

1. Skeletal survey: This is a series of plain X-rays of all the bones in the body.
2. Radionuclide bone scan: A radionuclide bone scan uses a radioisotope to identify a hot spot, a healing reaction, at the site of a fracture. It is a specialized X-ray that is particularly good at detecting recent fractures and may show additional fractures not evident on the skeletal survey. However, a bone scan will miss skull fractures and may miss metaphyseal fractures. As either test may miss different fractures, consideration should be given to performing both.

Can you tell how old a fracture is?

Although a recent fracture can be distinguished from an old fracture, radiologists can estimate the age only in weeks, not days. Despite fractures showing predictable X-ray features over time as they heal, dating of fractures in abused children can be difficult if no accurate description of the cause or timing of the injury has been given, further injury to an already broken bone occurs, or the bone has not been immobilized, which may alter the rate of healing.

When should you be concerned that a child may have been abused?

The following apply in the absence of organic bone disease:

- Rib fractures are highly indicative of abuse in children who have not been in a major accident.
- A femoral fracture in a child who is not walking can be suggestive of abuse.

- A spiral fracture is the commonest fracture of the femur in abused children younger than 15 months; in all other age groups, a transverse fracture is the commonest accidental or abusive femoral fracture.
- Metaphyseal fractures of the femur in very young children are more likely to be due to abuse than accidental causes.
- A humeral fracture has a one-in-five chance of arising from abuse, but a supracondylar fracture is highly suggestive of accidental injury.
- Up to a third of complex skull fractures may be as a result of abuse.
- Multiple fractures are frequently seen in abused children; these may show different stages of healing.

Implications for practice

A fracture, like any other injury, should never be interpreted in isolation. It must always be assessed in the context of the child's medical and social history, developmental stage, and explanation given. Any child with unexplained signs of pain or illness should be seen promptly by a doctor.

In the following situations there should be a careful evaluation to exclude child abuse:

- children under 18 months with a fracture;
- children whose fracture is inconsistent with their developmental stage;
- multiple fractures, particularly of different ages, in the absence of an adequate explanation;
- rib fractures in children with normal bones and no history of major accidents; and
- a fractured femur in a child who is not yet walking.

Appendix 3—Bruising—Welsh Child Protection Systematic Review Group

Bruising is the most common injury to a child who has been physically abused. This information is based on a systematic review of all the quality work in the world literature about bruising in children prepared by the Welsh Child Protection Systematic Review Group.

What is known about bruising?

Bruising is strongly related to mobility. Once children are mobile they sustain bruises from everyday activities and accidents. Bruising in a baby who is not yet crawling, and therefore has no independent mobility, is very unusual. Most children who are able to walk independently have bruises. Bruises usually happen when children fall over or bump into objects in their way. Children have more bruises during the summer months.

Where would you expect to see bruising from an accidental injury?

The shins and the knees are the most likely places where children who are walking, or starting to walk, get bruised. Most accidental bruises are seen over bony parts of the body, e.g., knees and elbows, and are often seen on the front of the body. Infants who are pulling to stand may bump and bruise their heads, usually the forehead. Fractures are not always accompanied by bruises.

When should you be concerned?

There are some patterns of bruising that may mean physical abuse has taken place.

Abusive bruises often occur on soft parts of the body, e.g., cheeks, abdomen, back, and buttocks. The head is by far the commonest site of bruising in child abuse. Clusters of bruises are a common feature in abused children. These are often on the upper arm, outside of the thigh, or on the body. As a result of defending themselves, abused children may have bruising on the forearm, face, ears, abdomen, hip, upper arm, back of the leg, hands or feet. Abusive bruises can

often carry the imprint of the implement used or the hand. Non-accidental head injury or fractures can occur without bruising.

Can you age a bruise accurately?

No. Estimates of the age of a bruise are currently based on an assessment of the colour of the bruise with the naked eye. The accuracy of observers who estimate the age of a bruise visually is no better than 50%. The evidence is that we cannot accurately age a bruise from an assessment of colour—from either a clinical assessment or a photograph. A practitioner who offers a definitive estimate of the age of a bruise in a child by assessment with the naked eye is doing so from their own experience without adequate published evidence.

Implications for practice

A bruise should never be interpreted in isolation and must always be assessed in the context of the child's medical and social history, developmental stage, and explanation given. Bruising that suggests the possibility of physical child abuse include:

- bruising in children who are not independently mobile;
- bruises that are seen away from bony prominences;
- bruises to the face, back, abdomen, arms, buttocks, ears, and hands;
- multiple bruises in clusters;
- multiple bruises of uniform shape; and
- bruises that carry an imprint—of an implement or cord.

Appendix 4—Pediatric Forensic Pathology Is Challenging for Forensic Pathology Generally: Why?

Issue in Pediatric Forensic Pathology	Analogue in Adult Forensic Pathology
<p>Natural disease presenting as trauma. Hemorrhagic disease of the newborn may have, as its first presentation, subdural hemorrhage. Differential diagnosis in infants of course will include Inflicted Head Trauma, especially Shaken Baby Syndrome.</p>	<p>Not such an issue. For example, no analogue of shaking, for all practical purposes, in adults. Occasionally, natural hemorrhagic disease presents as sudden unexpected death, but usually these cases are not difficult to distinguish from trauma and there will usually be other manifestations of the underlying disease than the bruising. However, the pathologist must be wary—occasionally someone with hemorrhagic disease will be assaulted.</p>
<p>Common reference to conditions on the margins of medical understanding is also a feature of these cases. For example, subclinical vitamin C deficiency; temporary brittle bone disorder; vaccination-induced sudden unexpected death/tendency to bleed.</p>	<p>These claims exceptional in adult cases.</p>
<p>Technical difficulties, for example, removing the brain in a way that causes minimum damage to what is a very soft structure in infants is challenging, especially if any time has passed between death and autopsy.</p>	<p>The size of adults makes the technical issues of a different kind.</p>
<p>Single fatal injury, often a head injury, with an accidental explanation.</p>	<p>Not such an issue, because many assaults involving adults are witnessed, and with the exception of stabbings and shootings, single fatal injuries are not particularly common. Often “punch and fall” cases.</p>
<p>Multiple injuries:</p> <ol style="list-style-type: none"> 1. Their timing; 2. The causal relationship between old injuries and death, and formulating a proper cause of death statement; 3. “Multiple injuries” may be inadmissible as the cause of death if they involve both recent and old injuries. Although possibly a true cause of death statement, the accused may only be charged with the recent injuries resulting in death because of difficulties in knowing who caused the older injuries. If so, it would be prejudicial to the accused to introduce the older injuries, and left with the recent injuries only, the pathologist may be hard pressed to conclude that, on their own, they must necessarily be the result of an assault 	<p>Not such an issue. Occasionally can be with “battered wives,” or with distinguishing between multiple falls, in, for example, a chronic alcoholic, and an assault.</p>
<p>Often more than one carer at the relevant time. This can be a problem, but is not an issue for the pathologist. It simply means that pathology obviously cannot do much to help distinguish between two potential assaulters when each is saying the other did it.</p>	<p>This can occasionally be an issue in elder abuse, but again is not really an issue for pathology.</p>

Issue in Pediatric Forensic Pathology	Analogue in Adult Forensic Pathology
<p>Occasional conflict between medical specialties involved during the deceased's hospitalization:</p> <ul style="list-style-type: none"> - Radiology; - Neurosurgery; or - Pediatrics <p>where conclusions made in a therapeutic context are transferred into the court room, a different environment that will have difficulty making sense of the conflicts. For example, the aging of a subdural hemorrhage.</p>	<p>Again this seems to be less of a problem with adults.</p>
<p>Resuscitation injuries can be an issue, but because more subtle injuries can be of greater significance in babies/children, resuscitation as a cause for such injuries, in circumstances where resuscitation has been attempted, can be difficult to rule out. Inexpert resuscitation in children, where there are claims of inadvertent use of resuscitative force, can also be a problem in particular cases, even if it is known that as a general rule injuries from resuscitation are few and of minor severity.</p>	<p>Resuscitation injuries are referred to not infrequently as possible explanations in a number of cases where neck compression and/or smothering have been factors that have been concluded as causing the death.</p>
<p>Controversy over Shaken Baby Syndrome; the categorical assurance the court occasionally requires that the findings in a particular case (not necessarily of SBS) are indeed those of an assault</p>	<p>No analogue in adults. One case has been described at the hands of security forces. Occasionally the possibility of an accidental (or self-inflicted) explanation arises especially in relation to single gunshot wounds or single stab wounds.</p>
<p>Major legal issues.</p> <p>Was the force used such that the person inflicting the injuries must have known that serious harm would result or was likely to result? This is not meant to be a legal formulation, but an indication of some understanding that issues in this area are important for the decision maker in criminal trials, and that the court will often want some indication of the sorts of forces required to cause particular injuries. Often these issues can only be addressed in the most general way.</p>	<p>This is a recurring theme in adult cases, especially single-stab-wound homicides not involving bone where there can be quite a lot of discussion about the degree of force required.</p>

Notwithstanding that many of the issues of pediatric forensic pathology are issues of forensic pathology more broadly, there are a number that are peculiar to the former. Because of the small number of cases, these are therefore correspondingly more challenging issues.

Appendix 5—Some Controversies Not Examined

Fractures

- Conclusions about the cause of the fracture from its appearance (often its radiological appearance). Advent of CT and MRI should immeasurably improve research capability into mechanisms of injury to cause particular fractures.

Head Injury

- How much force causes a SDH?:

- i. Terminological confusion: does a severe SDH necessarily mean a severe degree of force?
- ii. Assessment of the degree of force is not objective and has a significant subjective element; degree of force is critical to the law because if severe, the inference will be drawn that the person inflicting it must have intended serious injury or was aware that serious injury was likely. These states of mind are sufficient to find a person guilty of murder in many jurisdictions. What is being asked is, if there is an accused:
 - Could it have occurred in the way indicated by D.?
 - If D. does not know, or is not saying, and the child is of an age where it could not have sustained such an injury without the involvement of another person, was the force such that the person responsible must have known that harm would or was likely to result?

- With what degree of accuracy can the age of a SDH be ascertained?

- Does the presence of subdural hemorrhage in more than one compartment of the cranium necessarily imply greater degrees of force? Can and does subdural blood easily travel from one compartment to another?

- What significance does the presence or absence of a lucid interval have in deciding between inflicted and accidental trauma?

Asphyxia and Petechiae

- How many petechiae are significant?
- Does number matter?
- How are petechiae detected?
- Are there any limitations in the methods of detection?

Injury Determination

Aging of Injuries

- Adults vs. children
- Rates of healing in different tissues
- Rates of healing in circumstances of serious illness

Resuscitation

- The incidence and significance of resuscitation injuries in pediatric forensic pathology.

Starvation

- Best practice exclusion of all natural causes and cachexia in an apparently starved child?

Munchausens Syndrome by Proxy (Fabricated or Induced Illness)

- Does this exist?
- Can a child be force fed a fatal dose or doses of salt?
- If so, how can this be diagnosed?

Specific Injuries

- Accidental versus sexually assaultive injuries in children.
- Injured frenulum has been traditionally regarded as pathognomic or diagnostic of non-accidental injury. What is the evidence for this?

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