

© Maya2008 | DollarPhotoClub.com

Are Multiple Rib Fractures in Infants Always Due to Abuse?

hen he left the hospital a week ago your child was fine," police tell the newborn's mother. "Now he's back here and his ribs are fractured." The officer's tone takes a turn and becomes accusatory: "You know, rib fractures like these just don't occur spontaneously. With a child this small, we know something or someone had to have caused these injuries. We need you to tell us exactly how this happened." After noticing her child wincing in pain and crying, mom had brought the newborn back to the hospital of birth. X-rays were taken, child service workers were summoned, and police took an innocent mother into custody for abuse. What was supposed to be the happiest time in a new mother's life has now turned into the worst.'

The story of a parent accused of a crime she did not commit is not a myth; it is a true story with an ending that is still unwritten for many defendants. The way these stories end will be determined largely, if not exclusively, by criminal defense lawyers. They must be willing to expose the whole truth about the current research on infantile rib fractures due to metabolic bone disease.

Much has been said over the last few years about the use of faulty science in the courtroom. The National Academy of Sciences report, *Strengthening Forensic Science in the United States: A Path Forward*,² found that much courtroom "science," with the exception of properly applied DNA testing, is junk science and nothing more than conjecture. In the closely associated "shaken baby syndrome," the current and commonly accepted view may have no firm scientific foundations.³ Many innocent parents and caregivers — victims of misapplied and faulty science — are being convicted in infantile multiple rib fracture cases.

Where do these prosecutions go wrong? Where does current science in infantile rib fracture cases overstep its bounds? What can a defense attorney do about it?

I. Fractures

How is evidence of bone fractures commonly misused in wrongful child injury prosecutions? The story begins to unfold when a mother notices that her infant is flinching, wincing, and experiencing bouts of colic. Like most parents, she waits a week or two to see if it resolves itself. When she seeks medical care for her infant, doctors find multiple rib fractures, some healing and some new. Unknown to mother, doctor, or hospital staff, the child is suffering from a metabolic bone disease. The bone disease, not child abuse, caused the rib fractures.

The parent can offer no explanation for how any of these fractures occurred. The matter seems urgent to

BY JOHN CANNELL AND BRANDON SANCHEZ

hospital staff; they know of no conclusion to draw other than abuse. Furthermore, the parent may offer damning conjecture when confronted with the injuries, such as saying maybe a sibling was responsible or recounting the time the child slipped slightly during a bath and was jostled a bit. These explanations do not account for the extensive fractures seen on x-ray, but they do serve to make the parent look suspicious in the eyes of law enforcement and eventually, perhaps, a jury.

Given these facts, pediatric child abuse specialists would probably say this was a case of Multiple Unexplained Infantile Rib Fractures in Various Stages of Healing (MUIRFVSH). They would say rib fractures like these do not occur spontaneously and are characteristic of "nonaccidental injury," which means child abuse. The Department of Family Services (DFS) would remove the infant from the home, charge the parents with child abuse, and place the family's other children in foster care. But the parents are innocent, so where did the investigation go wrong?

A. MUIRFVSH

Multiple unexplained rib fractures can occur in the absence of child abuse. In fact, the idea that parental abuse could cause these x-ray findings is, as will be shown, a flimsy premise at best that leaves more questions than answers. The common belief motivating these prosecutions is that "something" traumatic had to happen to cause these types of fractures; they do not just happen by themselves. Child abuse specialists see one unexplained fracture as possible. However, child abuse is the only valid explanation for many pediatric child abuse specialists when they see two or more fractures (some healing and some new) and the parents cannot explain how they got there. When a helpless infant is involved, the presumption is that intentional child abuse caused MUIRFVSH. This presumption puts a life-changing set of events in motion.

The truth of the science, however, is that researchers do not know how common metabolic bone disease with multiple fractures is in nonabused infants. AAPCOCAN (American Academy of Pediatrics' Committee on Child Abuse and Neglect) specialists say that these fractures always indicate abuse. However, Wei et al. reported eight cases of posterior rib fractures in hospitalized infants x-rayed before they left the hospital, which excludes the possibility of the parents abusing them.⁴ A large group of otherwise normal infants has never been sequentially imaged for subclinical rib fractures. Consequently, no one knows how common MUIRFVSH is.

By the time MUIRFVSH was first reported, in the 1960s, interest in child abuse was growing due to a seminal article by Dr. C. Henry Kempe et al.⁵ In the 1960s and 1970s, child abuse specialists simply speculated that MUIRFVSH was due to child abuse and repeatedly published that conjecture.⁶ No firm scientific evidence supported the conjecture.

Child abuse specialists largely ignored other causes of MUIRFVSH, such as known causes of metabolic bone disease, in the rush to judgment about child abuse. In their rush to judgment, child abuse specialists refused to add the "idiopathic metabolic bone disease" category. "Idiopathic" means medicine does not know the cause. Dr. David Ayoub, a radiologist who may be the world's leading expert on healing infantile rickets, reports some of the radiographs depicted in the Kempe article actually showed rickets — the metabolic bone disease not child abuse. One of the radiographs in the Kempe article even shows classic bowing of the legs, which is diagnostic of rickets.7 This means the seminal paper that started the medical profession on its current course of always diagnosing MUIRFVSH as child abuse was fatally flawed.

Rarely does modern medicine admit its ignorance. An exception is sudden infant death syndrome (SIDS), which was first reported before child abuse allegations became common. SIDS, by definition, means modern medicine does not know why some infants just suddenly die. Thus, in SIDS, modern medicine admits its ignorance. With metabolic bone disease, however, modern medicine, in effect, claims it knows every cause. This is simply not possible. Modern medicine does not know everything about any medical disorder.

Medical science cannot say that MUIRFVSH is always child abuse. However, child abuse experts, usually pediatricians, rely on criteria formulated by AAPCOCAN. Those guidelines clearly imply, despite the lack of scientific evidence, that the finding of MUIRFVSH always means child abuse.⁸ Most pediatric child abuse experts belong to AAPCOCAN. Some child abuse experts are pediatricians who take an extra three-year residency training to become certified child abuse experts. Such experts are invariably members of AAPCOCAN. However, what is not often known is that AAPCOCAN members almost always appear as expert witnesses for the state. Seldom do they appear as expert witnesses for the defense, and almost never in MUIR-FVSH cases because a subcommittee of AAPCOCAN has issued clear guidelines stating MUIRFVSH means child abuse.⁹

A large sequential bone imaging study of normal infants needs to be done before more innocent parents are accused of the horrendous crime of child abuse. It may be that MUIRFVSH fractures are present in nonabused, otherwise healthy-appearing infants due to idiopathic metabolic bone disease. That is, MUIRFVSH, like SIDS, may be due to a cause not yet discovered by modern medicine.

B. MUIRFVSH, but No Organ Damage

Consider a scenario in which law enforcement officers accuse parents of abusing their child in such a way as to cause multiple unexplained rib fractures (some new, some healing), but there is no organ damage present. How can traumas severe enough to break ribs leave no internal organ damage or soft tissue injury? AAPCOCAN experts often give testimony that defies common sense: Multiple traumatic rib fractures in infants can occur with no internal injuries and no soft tissue injury.

In the trauma literature, Victor Garcia et al. reported that 100 percent of children with multiple rib fractures also suffered internal injuries.¹⁰ Other trauma experts report multiple rib fractures in children due to trauma sufficient to cause death or internal injuries over 90 percent of the time.¹¹ In 91 children who suffered chest trauma of the type the states' experts often hypothesize, the vast majority had internal injuries.¹² Yet in cases of MUIRFVSH without organ damage, the presumption is child abuse, and counsel representing the accused must challenge this presumption.

C. Nondisplaced Rib Fractures

Why are the rib fractures in MUIRFVSH cases usually perfectly aligned (non-displaced) or minimally displaced? Does this make sense given the numerous traumatic beatings often alleged in these prosecutions? Displacement means that at the site of the fracture the ends of the bones are either angulated or the ends do not align with each other. A nondisplaced fracture means the ends of the two frac<u>~-</u>.

tured bones exactly line up with each other. An extreme example of a displaced fracture is a bone that has ripped out of the skin after a break — a compound fracture. Clearly one part of the bone is "displaced" from the other end of the break in that instance. On the contrary, in a nondisplaced rib fracture the two ends of the fractured rib would appear on an x-ray as still aligned with each other.

Rib fractures are usually not displaced in metabolic bone disease. What about after trauma? Wouldn't one expect to see displacement after abusive trauma? In case reports of posterior rib fractures secondary to the normal birthing process, most of the rib fractures were displaced.¹³ Thus, normal childbirth can cause displaced rib fractures, but repeated and savage beatings at the hands of an angry parent do not? Similar to the absence of internal organ damage when there are rib fractures, nondisplacement brings the claim of intentional trauma into question. The absence of a displaced break in these cases is suspicious and should be the subject of cross-examination by defense counsel.

D. Rib Fractures With No Soft Tissue Injury

How often are the infants in these MUIRFVSH abuse cases entirely free of bruising? It is difficult to understand how beatings severe and frequent enough to cause MUIRFVSH do not leave a bruise or any soft tissue swelling. While fractures from falls often do not bruise,¹⁴ repeated savage beatings should surely leave some bruises or swelling.¹⁵ Studies of allegedly abused children found that bruises over the fracture sites were uncommon,¹⁶ but such studies are problematic because of selection bias (the innocent being included with the guilty), as discussed below.

How does one know for certain that abuse occurred in such studies? If the thesis of this article is correct and innocent parents are being held guilty, current "scientific" studies on child abuse shed little light on the issue. State experts too often claim repeated savage beatings caused bruise-less fractures, but it is still hard to explain how fragile, tiny infants can be repeatedly and savagely beaten without leaving a single bruise.

E. Fractures Stop When The Child Is Removed From the Home

Another oft-repeated maxim of AAPCOCAN experts is that abuse must have occurred because no new fractures arise while the infant is away from the parents. The implication is that it was the parent or caregiver that caused the injuries to the child. As discussed in Section III, one cause of these unexplained injures could be rickets. Studies cited in Section III show infantile rickets heals spontaneously, and thus no new fractures would be expected after CPS removes the infant from the home. This is especially true when breast-fed infants are removed from the home and started on vitamin D-rich formula because human breast milk contains almost no vitamin D. Therefore, the idea that the lack of fractures being present after a child is removed from a caregiver's home means authorities know the cause of the original fractures is just not true and should be challenged.

F. Lack of Abusive Parental Behavior

Do potential eyewitnesses, such as friends and relatives, say they saw abusive behaviors in the parents? How can abuse severe enough to cause MUIRFVSH go undetected by friends and relatives? Common sense dictates that at least one witness would have seen some evidence of abusive parental behavior.

Wouldn't parents who savagely beat their tiny infant severely enough to cause MUIRFVSH show evidence of psychological abnormalities? Dr. Kempe's influential 1962 child physical abuse article,¹⁷ more than any other, led to a widespread medical belief that MUIRFVSH fractures always mean child abuse.

Since Kempe and his colleagues published their work and a subsequent book-length treatment on child abuse issues, a number of others have weighed in on the psychological and psychiatric factors, as well as other matters, bearing on persons who are involved in intimate partner violence, domestic violence, and violence against children.18 Attorneys are advised to consult some of the latest literature on these subjects. Several researchers have devoted considerable time to examining various factors including developmental history, medical issues, psychological issues, biochemical and neuroanatomical issues — that have all been thought to play a part, in varying ways, in scenarios that have involved child abuse. In addition, a number of publications devoted to the study of violence against children and violence in families may be helpful in given cases.19

II. Known Metabolic Bone Diseases Causing MUIRFVSH

When allegations of child abuse do not make sense, are there any known alternatives that the diagnosing physician might have missed? The conditions listed below can be overlooked or mistaken for child abuse.

A. Vitamin D Deficient Rickets

In a modern-day study, fractures in young children have not been associated with vitamin D deficiency.²⁰ Old medical textbooks, however, including *Brenner's Practice of Pediatrics*, have repeatedly stressed that fractures are a complication of rickets.²¹ In 1921, Park and Howland found spontaneous multiple rib fractures in 22 children with rickets, the majority also exhibiting multiple extremity fractures.²²

Few dispute that rickets is becoming more common due to increasing vitamin D deficiency arising from sun avoidance and the use of sunblock.²³ Recently experts have pointed out that healing infantile rickets gives a much different radiological appearance than does classic rickets.²⁴ Little is known about healing infantile rickets in the modern age; according to Ayoub et al., radiologists in training no longer study it. In 2012, radiologists Kathy A. Keller and Patrick D. Barnes wrote that infantile rickets is common and readily misdiagnosed as child abuse.²⁵

However, a National Library of Medicine search for "healing infantile rickets" revealed no useful references except for the recent paper by Ayoub et al. in which four experts stated that healing infantile rickets might be quite common. Through the use of uterine ultrasound, it was discovered that approximately one-third of modern-day fetuses in Southampton, England, have rickets, as evidenced by the classic finding of widening at the end of the femur.²⁶

Despite taking prenatal vitamins, 45 percent of Caucasian women residing in the northern United States are vitamin D insufficient when they give birth.²⁷ At most, only 35 percent of American infants consume the daily intake of vitamin D recommended by the American Academy of Pediatrics.²⁸ Paradoxically, breast milk, nature's perfect food, has almost no vitamin D in it unless the lactating mothers are sunbathing or taking high doses of vitamin D3.²⁹ In modern medicine, a wide divergence exists between expert opinion and standard

38

MEMBERSHIP APPLICATION

SPECIAL INTRODUCTORY OFFER: 15 months of NACDL® membership for the price of 12! (New members only)

YES! Sign me up as a new member of NACDL® and start my subscription to The Champion® today!

Applicant Name:			
Referred By:			
Firm Name:			
Address:			
City:	_ State:	Zip:	
Date of Birth:	Gender	:	
Phone:			
Fax:			
E-mail:			
State Bar(s) & Admission Date(s):			
Bar Number(s):			
I certify that I meet the criteria for the membership category to which I am applying.			
Attornouse lam a member of t	he har in good star	ading and Lam not subject to suspension or	

disbarment in any jurisdiction. I understand that prosecutors are not eligible to be NACDL members. _ Date:_

I qualify for the following membership category (Please check one)

Membership Categories:	Annual Dues:
Regular	\$319
New Lawyer*	\$179
Law Professor	\$165
Fedl. or State Public Defender**	\$135
🗖 Judge	\$189
🗖 Military	\$165
🗖 Law Student	\$65
Associate	\$189
International	\$179
Sustaining	\$465
President's Club	\$689
Life Member***	\$5,000

* New Lawyer Membership – for members of the bar for less than 5 years ** Public Defender Membership – for full-time, salaried employees of non-profit or government agency *** Life Membership - a one-time contribution; or 5 installments of \$1000 each over 5 consecutive years

Credit Card #:		
Expiration Date:	Card Type:	
Billing Address:	<i></i>	
Name on Card:		
Signature:		

www.nacdl.org / memberservices@nacdl.org Membership Hotline: 202-872-4001

Return by FAX to: (202) 872-8690, Attn: NACDL® Membership Director Or Mail with Check Payable to NACDL®: 1660 L St. NW, 12th Fl. Washington, DC 20036 clinical practice in preventing and treating vitamin D deficiency.³⁰

In 1942, F.W. Clements performed the largest known prospective analysis of healing infantile rickets.³¹ Nearly half of all of the full-term, otherwise normal infants that Clements studied developed subclinical rickets. The prevalence increased after the first month, peaked in the third month, and disappeared by eight months. This means that infantile rickets goes away by itself as the infant ages. In 1952, American pathologists found that 83 percent of three-monthold infants who died from other causes had rickets.³² As recently as 1958, even in sunny Israel, rickets was present in 17 percent of infants at autopsy, the majority of cases occurring in infants younger than six months.33 The above studies stand in sharp contrast to a recent study by AAPCOCAN members showing that in infant fatalities when abuse is suspected, rachitic changes appear to be rare on biopsy.34

B. Failure to **Diagnose Rickets**

What happens when there is a failure to diagnose rickets? Ask Chana al-Alas and Rohan Wray of the United Kingdom.35 In 2012, it was only an autopsy and bone biopsy of their infant that cleared the couple after they had been charged with murdering the child. Chana al-Alas, who was just 16 when she became pregnant, and her partner Rohan Wray, then 19, were acquitted in December 2011 of murdering their infant, Baby Jayden. The autopsy revealed that Baby Jayden had metabolic bone disease due to infantile rickets that had caused multiple fractures. This disease, infantile rickets, went undetected by radiologists and pediatric child abuse experts when Jayden was alive. If their infant had lived, an autopsy and bone biopsy would not have been done, and the charges against the parents would have proceeded.

The modern medical diagnosis of rickets is based entirely on x-rays. However, a recent study of infants dying of sudden infant death syndrome found that only seven percent of bone biopsyproven rickets was picked up by pediatric radiologists on x-rays.³⁶ That is, two pediatric radiologists missed over 90 percent of biopsy-proven rickets when looking at x-rays.37 Radiologists do not appreciate that the gold standard for the diagnosis of rickets is a bone biopsy, not an x-ray, and that the pathologist reading the bone biopsy must be knowledgeable about rickets. In writing about

Signature:

≻

metabolic bone disease, such as rickets, a current textbook of orthopedic pathology states, "In subtle cases absolute certainty requires tissue examination."³⁸ The authors add, "A bone biopsy is indicated in every patient in whom a cause of fracture is unexplained."³⁹

How often is rickets overlooked in modern testing? Normal vitamin D levels in infants fall somewhere between 30 and 50 ng/ml. A recent study of a group of 52 children who died of various causes reported rickets in 10 children with vitamin D levels of less than 10 ng/ml, but x-rays missed the rickets 70 percent of the time.⁴⁰ In the same study, eight infants with vitamin D levels between 10 and 20 ng/ml had rickets, but radiology was normal in 100 percent of these children. Three of the infants had fractures. Again, pediatric radiologists missed biopsy-proven rickets 84 percent of the time. These studies suggest that modern day pediatric radiologists misdiagnose the vast majority of cases of infantile rickets.

C. Rickets Overlooked

Several experts have written that child abuse and healing infantile rickets are readily confused,⁴¹ but this is not the position of AAPCOCAN.⁴² In a recent review, four experts reported that a particular type of bone x-ray finding, classic metaphyseal lesion, thought to always mean child physical abuse by AAPCOCAN, is in fact healing infantile rickets.⁴³

How can this massive oversight occur? Other than the shortcomings of radiology, defense counsel will often find that the examining doctor or pathologist who concludes abuse occurred did not review the child's prior medical records, including birth records, pediatrician visit records, and other available medical data. This means that medical personnel are putting forth conclusions without all the facts. Therefore, as a matter of course in these cases, defense counsel must engage in the process of obtaining all known medical records and, with the help of a qualified expert, examine whether all the pieces of the puzzle put forth by the state fit together. In the case involving Chana al-Alay and Rohan Wray - wrongfully accused of causing the death of their child — the physician who ordered the bone biopsy that eventually exonerated the young couple reportedly faced harsh pressure from law enforcement to avoid this inquiry. This is undoubtedly because in the mind of law enforcement officials, the conclusion of abuse was already proved and further medical testing did not fit with the conclusion they had already reached.

At the very least, the state should test the vitamin D levels of both the infant and mother at the time the state begins to entertain the diagnosis of MUIRFVSH due to abuse. A recent autopsy study of biopsy-proven rickets found that rickets can occur in infants with vitamin D blood levels above 20 ng/ml.44 In MUIRFVSH cases, it is crucial to employ the services of a board certified radiologist who is educated on the appearance of infantile metabolic bone diseases (and particularly in the appearance of infantile rickets). Defense counsel should cross-examine on the lack of complete medical investigation by the physician diagnosing abuse when MUIRFVSH is present.

When an infant presents with MUIRFVSH due to rickets, pediatric child abuse experts usually diagnose child abuse based on criteria developed by AAPCCAN.⁴⁵ Those guidelines clearly imply that the finding of MUIRFVSH is almost always due to child abuse. Those guidelines also say that vitamin D deficient rickets is "uncommon" and rarely cause fractures when it does exist. Most radiologists trained within the last 30 years have been taught to think of child abuse, not metabolic bone disease, when they see MUIRFVSH. In fact, infantile rickets is often a cause of MUIRFVSH and is often mistaken for child abuse. Defense attorneys should use the data in this article and challenge the state experts' erroneous abuse diagnosis prior to trial, during trial, and if there is a conviction, on appeal.

The infant needs to have blood tests, ideally before being taken out of the home — a vitamin D test [25(OH)D], alkaline phosphatase, phosphorous, calcium, and parathyroid hormone. If these blood tests were not administered when the infant was removed, defense counsel should request that they be administered as soon as possible. However, as noted earlier, these blood test results may revert to normal after the infant is placed on vitamin D-enriched formula. If that is the case, the defense should obtain a vitamin D blood test of the mother because the infant's 25(OH)D level at birth can be calculated from the mother's 25(OH)D level.

D. Ehlers-Danlos Syndrome

Joint hypermobility syndrome, also called Ehlers-Danlos syndrome, is a group of mainly autosomal dominant



National Advocacy Calls on Developing Legislation (NACDL)

Angelyn Frazer hosts this monthly conference call to inform advocates of legislation and litigation that impact criminal justice issues. The calls generally feature a presentation by an expert and a question and answer segment with listeners.

To listen please visit http://www.nacdl.org/ scjnadvocacycalls/



inherited disorders that affect connective tissues — primarily the skin, joints, bones, and blood vessel walls. The disease is much more common than previously thought.⁴⁶ In one study, 50 percent of the parents in contested child abuse cases had evidence of joint hypermobility syndrome.⁴⁷

Connective tissue is a complex mixture of proteins and other substances that provides strength and elasticity to the underlying structures in the body. Joint hypermobility syndrome can predispose to metabolic bone disease and fractures.48 It may be that this syndrome explains many cases of MUIRFVSH, but this is speculation because there has been no scientific study on the prevalence of fractures in infants with hypermobility syndrome. All infants involved in child abuse allegations should be seen by a physician experienced in diagnosing adult and infantile cases of Ehlers-Danlos syndrome to rule it out as a cause of fragility fractures. The parents, not CPS, should take the infant to a physician with experience in this area.

E. Osteogenesis Imperfecta

Osteogenesis imperfecta (OI) is a rare congenital bone disorder characterized by brittle bones that are prone to fracture. Infants with OI are born with defective connective tissue, or without the ability to make it, usually because of a connective tissue deficiency. At least eight genetic types of OI can be distinguished. Most cases are caused by genetic mutations. Diagnosis of OI is based on the clinical features and may be confirmed by DNA testing.

For this reason, an expert pediatric endocrinologist should be consulted in MUIRFVSH cases. The parents should accompany the infant on the visit, not CPS personnel. This will allow the endocrinologist to form his or her own opinion concerning whether these parents would be likely to savagely and repeatedly beat their infant. Too often, children with undiagnosed OI are initially said to be victims of child abuse.⁴⁹ Kazuya Ojima et al. presented a case involving parents who were prosecuted for murder until autopsy findings indicated OI.⁵⁰

F. Scurvy

Studies show that both breast-fed and formula-fed preterm infants can have low vitamin C blood levels.⁵¹ The classical form of infantile scurvy, with bruises, broken bones and sores that will not heal, is still seen today, but may be made more evident in infants with borderline vitamin C depletion.52 Moreover, scurvy (also called Barlow's disease) can be mistaken for child abuse.53 Some researchers think subclinical scurvy is more common than previously believed.54 Scurvy in children may have metabolic bone disease with characteristic x-ray findings that can cause MUIRFVSH.55 Ideally, white cell vitamin C levels or vitamin C saturation tests should be ordered before the infant is removed from the home and the infant's diet is changed.

G. Copper Deficiency

Although rare, infantile copper deficiency may cause fractures and the significant x-ray changes that are associated with metabolic bone disease.⁵⁶ Unless serum copper and ceruloplasmin levels are measured, these cases will be missed.

H. Genetic Forms of Metabolic Bone Disease With Rickets

Multiple rare forms of inherited or genetic rickets with metabolic bone disease exist.⁵⁷ Unless the proper blood tests are preformed, these cases will be missed.⁵⁸

I. Other Rare Causes of Metabolic Bone Disease

Other rare causes of metabolic bone disease have been identified, such as Menkes' syndrome, biliary atresia, proprionic acidemia, myofibromatosis, congenital syphilis, and congenital cytomegalovirus infection.⁵⁹ Two percent of premature infants have subsequent rib fractures.⁶⁰

J. Idiopathic Bone Disease

It is important to reiterate that, even if there is no evidence of any of the above known causes of metabolic bone disease, defense counsel should consider arguing that the infant has an idiopathic metabolic bone disease. If the common sense indicators of abuse are lacking - no organ damage, nondisplaced fractures, no soft tissue injury, no bruises, no motive, and a psychologically healthy parent — then the infant may simply have an idiopathic form of metabolic bone disease, similar in rational to SIDS. In support of this argument, counsel should consider researchers Paterson and Monk, who described a very controversial syndrome of metabolic bone disease

with fractures that they called "temporary brittle bone disease" in 104 infants who were the subject of child abuse allegations.⁶¹ In this syndrome, infants were found to have fractures and brittle bones with fragility fractures for unknown reasons, which indicates, by definition, "idiopathic metabolic bone disease."

Paterson lost his medical license in the United Kingdom after he frequently testified for the defense in MUIR-FVSH cases. However, Paterson also reported on five newborn infants who suffered their MUIRFVSH fractures before they ever left the hospital, which excludes the possibility of the parents abusing them.⁶² If the parents had taken any of these five newborn infants home and later returned to the hospital, they would have been charged with child abuse.

III. Infantile Abuse In Statistics

Is there evidence that infantile child abuse is being overdiagnosed? According to the U.S. Department of Health and Human Services, in 2012 there were 584 reported deaths due to abuse of infants less than one year of age in the United States.⁶³ However, there were almost 300 percent fewer (202) reported abusive deaths of toddlers between the ages of one and two.

The same study reported the prevalence of physical abuse in the United States was 22/1,000 for infants less than one year of age, but that number falls to 11.8/1,000 for toddlers between the ages of one and two. Why is there such a sharp decline in child abuse with the onset of toddlerhood? Wouldn't tempers be easier to lose with a mischievous toddler? Shouldn't child abuse be higher among toddlers? If frayed nerves due to incessant crying is the motive in infantile abuse, wouldn't beating an infant lead to even louder crying? One possible explanation for these discrepancies is that child physical abuse is overdiagnosed in infancy. AAPCOCAN policy is that infants found dead to have MUIRFVSH on autopsy are almost always diagnosed with child abuse.64 If the thesis of this article is correct, this policy by itself will artificially inflate infantile deaths attributed to abuse.

Current medical and governmental studies on the occurrence of infantile physical abuse are problematic due to selection bias, i.e., bias that occurs when innocent caregivers are included in the

·-v

guilty group. When the state cites studies showing the prevalence of infantile abuse, defense attorneys must understand the concept of selection bias. In government and "scientific" studies, abuse is usually determined by the opinion of Child protective Services (CPS) or legal outcome.

In evaluating "scientific" child abuse studies produced by the state, the defense should consider the following questions to find out if infantile abuse numbers were inflated by wrongfully including innocent caregivers in the "guilty" group. What per-cent of studies of "guilty" abuse cases involve CPS or law enforcement telling innocent parents that if they admit to the abuse and take parenting classes, then their infant will be immediately returned and they can avoid further costly litigation? What percentage of "guilty" abuse cases involve CPS allowing innocent parents to plead guilty to the lesser charge of neglect to have their infant returned and avoid further litigation? In what percentage of "guilty" abuse cases does the district attorney or CPS give a mother the "Sophie's Choice" of not being prosecuted and getting her infant back if she will testify against her innocent husband? What percent of "guilty" abuse cases involve an innocent parent accepting a plea bargain after the defense attorney concludes a trial will result in conviction? For these reasons, virtually all governmental and "scientific" medical studies on infantile child abuse have selection bias and include an unknown number of innocent caregivers in the abused group, making the studies scientifically unreliable.

IV. Conclusion

MUIRFVSH cases can stem from idiopathic metabolic bone disease or a known bone disease, but child abuse is not a valid default conclusion in a society interested in protecting the relationship between innocent parents and their children. Defense attorneys in MUIRFVSH cases need to obtain a variety of expert witnesses, including a radiologist, a forensic pathologist, a pediatric endocrinologist, a pediatrician, and a forensic psychiatrist. The state may try to use expert medical testimony about x-rays, based on AAPCOCAN guidelines, to provide all the elements of a crime. However, this is not enough evidence to responsibly allege abuse. When there is no additional evidence of abuse in

MUIRFVSH cases, such as internal organ damage, bruising, displaced rib fractures, parental psychopathology or soft tissue injury, there is not enough information to responsibly allege abuse.

Notes

1. Based on an account from Colin R. Paterson, *Temporary Brittle Bone Disease: Fractures in Medical Care*, 98(12) ACTA PAEDIATR. 1935-38 (2009).

2. NATIONAL ACADEMY OF SCIENCES, STRENGTHENING FORENSIC SCIENCE IN THE UNITED STATES: A PATH FORWARD (2009).

3. Deborah Tuerkheimer, *The Next Innocence Project: Shaken Baby Syndrome and the Criminal Courts*, 87 WASH. U. L. REV. 1 (2009); R.H. Kelly & Z.M. Bravos, *A Critical Look at the Shaken Baby Syndrome*, 97(4) *ILLINOIS BAR J.* (2009); *Shaken Baby Syndrome Faces New Questions in Court*, N.Y. TIMES, Feb 1, 2011; J. Pishko, *How Can Doctors Be Sure a Baby's Been Shaken*? THE ATLANTIC, Nov. 24, 2014.

4. C. Wei et al., *Fractures in a Tertiary Neonatal Intensive Care Unit in Wales*, 101 ACTA PAEDIATR. 587-590 (2012).

5. C.H. Kempe, et al., *The Battered-Child Syndrome*, 181 JAMA 17-24 (1962).

6. D.L. Griffiths & F.J. Moynihan, Multiple Epiphyseal Injuries in Babies (Battered Bay Syndrome) 2 Br. MED. J. 1558-61 (1963); C.T. Teng, et al., Skeletal Injuries of the Battered Child, 6 AM. J. ORTHOP. 202-207 (1964); H.N. Krige, The Abused Child Complex and Its Characteristic X-ray Findings, 40(21) S. AFR. MED. J. 490-93 (1966).

7.http://www.vitamindcouncil.org/wp -content/uploads/2013/04/Ayoub-D.-Critique-of-Kempe-et-al-1962.pdf (last visited May 24, 2014).

8. E.G. Flaherty, et al., *Evaluating Children With Fractures for Child Physical Abuse*, 133(2) PEDIATRICS 477-89 (2014).

9.*Id*.

10. V.F. Garcia, et al., *Rib Fractures in Children: A Marker of Severe Trauma*, 30 J. TRAUMA 695-700 (1990).

11. B.T. Flagel, et al., *Half-a-Dozen Ribs: The Breakpoint for Mortality*, 138(4) SURGERY 717-23 (2005).

12. S.J. Crankson, et al., *Pediatric Thoracic Trauma*, 22(2) SAUDI MED. J. 117-20 (2001).

13. R.R. van Rijn, et al., *Birth-Related Mid-Posterior Rib Fractures in Neonates: A Report of Three Cases (and a Possible Fourth Case) and a Review of the Literature*, 39(1) PEDIATR RADIOL. 30-34 (2009).

14. M.L. Peters, et al., *The Presence of Bruising Associated With Fractures*, 162(9) ARCH. PEDIATR. ADOLESC. MED. 877-81 (2008).

15. F. Dedouit, et al., Child Abuse: Practical Application of Autopsy, Radiological, and Microscopic Studies, 53(6) J. FORENSIC SCI. 1424-1429 (2008).

16.T.J.Valvano, et al., *Does Bruising Help Determine Which Fractures Are Caused by Abuse*? 14(4) CHILD MALTREAT. 376-81 (2009).

17. C.H. Kempe, et al., *The Battered-Child Syndrome*, 181 JAMA 17-24 (1962).

18. Ben Cosway et al., Occult Rib Fractures: Defining the Cause, 24 CHILD ABUSE REVIEW 6 (2015); S.T. Azar, A Cognitive Behavioral Approach to Understanding and Treating Parents Who Physically Abuse Their Children, in CHILD ABUSE: NEW DIRECTIONS IN PREVENTION AND TREATMENT ACROSS THE LIFESPAN 79-101 (D.A. Wolfe, et al., eds. 1997); J.S. Milner & C. Chilamkurti, Physical Child Abuse Perpetrator Characteristics: A Review of the Literature, 6 J. INTERPERSONAL VIOLENCE 345-366 (1991); D.A. Black, et al., Risk Factors for Child Psychological Abuse, 6 Aggression AND VIOLENT BEHAV. 189-201 (2001); A.L. Hazen, et al., Female Caregivers' Experiences With Intimate Partner Violence and Behavior Problems in Children Investigated as Victims of Maltreatment, 117 PEDIATRICS 99-109 (2006).

19. CHILD ABUSE & NEGLECT: THE INTERNATIONAL JOURNAL (http://www.journals. elsevier.com/child-abuse-and-neglect); CHILD ABUSE REVIEW (http://onlinelibrary. wiley.com/journal/10.1002/(ISSN)1099-0852); CHILD MALTREATMENT (http://cmx. sagepub.com).

20. S. Schilling, et al., Vitamin D Status in Abused and Nonabused Children Younger Than 2 Years Old With Fractures, 127(5) PEDIATRICS 835-41 (2011).

21. A. Hess, Rickets, Including Osteomalacia and Tetany 250-270 (1930); M. Eliot & E. Park, *Rickets*, in I. McQuarrie (ed.), Brenner's Practice of Pediatrics, Vol. One 1-110 (1948).

22. E.A. Park & J. Howland, *The Dangers* to Life of Severe Involvement of the Thorax in Rickets, 32 BULL. JOHNS HOPKINS HOSP. 102-115 (1921).

23. M.F. Holick, *Resurrection of Vitamin D Deficiency and Rickets*, 116(8) J. CLIN. INVEST. 2062-2072 (2006); P.M. Rowe, *Why Is Rickets Resurgent in the USA*? 357 LANCET 1100 (2001).

24. D.M. Ayoub, et al., *A Critical Review* of the Classic Metaphyseal Lesion: Traumatic or Metabolic? 202(1) AJR AM. J. ROENTGENOL 185-96 (2014).

25. K.A. Keller & P.D. Barnes, *Rickets vs. Abuse: A National and International Epidemic*, 38(11) PEDIATR. RADIOL. 1210-1216 (2008).

26. P. Mahon, et al., *Low Maternal Vitamin D Status and Fetal Bone Development: Cohort Study*, 25(1) J. BONE MINER. RES. 14-19 (2010).

27. L.M. Bodnar, et al., High Prevalence

<u>~-</u>. ш S 0 ш Δ ഗ ALWAY S ⊢ Z ₹ ₹ Ζ Ζ S ш ≃ CTU < ≃ <u>.</u> ഫ ≃ ш ULTIP Z ш ~ <

of Vitamin D Insufficiency in Black and White Pregnant Women Residing in the Northern United States and Their Neonates, 137(2) J. NUTR. 447-52 (2007).

28. C.G. Perrine, et al., Adherence to Vitamin D Recommendations Among U.S. Infants, 125(4) PEDIATRICS 627-32 (2010).

29. B.W. Hollis, Vitamin D Requirement During Pregnancy and Lactation, 22 J. BONE MINER. Res. (2007).

30. M. Findlay, et al., *Treatment of Vitamin D Deficiency Divergence Between Clinical Practice and Expert Advice*, 88 POSTGRAD. MED. J. 255-60 (2012).

31. F.W. Clements, *Rickets in Infants Aged Under One Year; Incidence in Australian Community and Consideration of Aetiological Factors*, 1 MED. J. AUST. 336-46 (1942).

32. R.H. Follis, et al., *The Prevalence of Rickets at Autopsy During the First Two Years of Age*, 91(6) BULL. JOHNS HOPKINS HOSP. 480-97 (1952).

33. B. Griffel & S.T. Winter, The Prevalence of Rickets at Autopsy in a Subtropical Climate; A Pilot Study in Israel, 4(1) J. TROPICAL PEDIATRICS 13-16 (1958).

34. J.M. Perez-Rossello, et al., *Prevalence* of Rachitic Changes in Deceased Infants: A Radiologic and Pathologic Study, 41 PEDIATR. RADIOL. S57 (2011).

35. http://www.theguardian.com/society/2012/apr/20/children-safety-jaydenrickets-misdiagnosis; http://www.familylawweek.co.uk/site.aspx?i=ed97208.

36. M.C. Cohen, et al., Vitamin D Deficiency and Sudden Unexpected Death in Infancy and Childhood: A Cohort Study, PEDIATR. DEV. PATHOL. (2013).

37. D. Ayoub, *Limitations of Radiology in Rickets*, 16(5) PEDIATR. DEV. PATHOL. 397 (2013).

38. V. VIGORITA, ORTHOPEDIC PATHOLOGY 104 (2d ed. 2007); see Chapter 3, Metabolic Bone Disease.

39. Id. at 127.

40. I. Scheimberg & L. Perry, Does Low Vitamin D Have a Role in Pediatric Morbidity and Mortality? An Observational Study of Vitamin D in a Cohort of 52 Postmortem Examinations, PEDIATR. DEV. PATHOL. (2014).

41. K.A. Keller & P.D. Barnes, *Rickets vs. Abuse: A National and International Epidemic*, 38(11) PEDIATR. RADIOL. 1210-1216 (2008); C.R. Paterson, *Vitamin D Deficiency Rickets and Allegations of Nonaccidental Injury*, 98(12) ACTA PAEDIATR. 2008-2012 (2009); N.M. Clarke & J.E. Page, *Vitamin D Deficiency: A Paediatric Orthopaedic Perspective*, 24(1) CURR. OPIN. PEDIATR. 46-49 (2012).

42. Flaherty, supra note 8.

- 43. Ayoub, supra note 24.
- 44. Ayoub, *supra* note 37.
- 45. Flaherty, *supra* note 8.
- 46. A. Fikree, et al., Joint Hypermobility

Syndrome, 39(2) RHEUM DIS. CLIN. NORTH AM. 419-30 (2013).

47. C.R. Paterson & P.A. Mole, Joint Laxity in the Parents of Children With Temporary Brittle Bone Disease, 32(9) RHEUMATOL. INT. 2843-2846 (2012).

48. O. Bar-Yosef, et al., Multiple Congenital Skull Fractures as a Presentation of Ehlers-Danlos Syndrome Type VIIC, 146A(23) Am. J. MED. GENET. 3054-3057 (2008); A.J. Carr, et al., The Clinical Features of Ehlers-Danlos Syndrome Type VIIB Resulting From a Base Substitution at the Splice Acceptor Site of Intron 5 of the COL1A2 Gene, 31(4) J. MED. GENET. 306-311 (1994); A.L. Dolan, et al., Assessment of Bone in Ehlers-Danlos Syndrome Ultrasound by and Densitometry, 57(1) ANN. RHEUM. DIS. 630-633 (1998); J.L. Yen, et al., Clinical Features of Ehlers-Danlos Syndrome, 105(6) J. FORMOS. MED. ASSOC. 475-80 (2006); http://www.wmtw.com/health/couplehopes-diagnosis-will-reunite-family-endchild-abuse-allegations/29901002.

49. T. Aakhus, et al., Are Prosecuted Parents Allowed the Benefit of the Doubt in Cases of Child Abuse? 110(5) TIDSSKR. NOR. LAEGEFOREN. 627-28 (1990); M.S. Kocher & L. Dichtel, Osteogenesis Imperfecta Misdiagnosed as Child Abuse, 20 J. PEDIATR. ORTHOP. B. 440-43 (2011).

50. K. Ojima, et al., An Autopsy Case of Osteogenesis Imperfecta Initially Suspected as Child Abuse, 65(2) FORENSIC SCI. INT. 97-104 (1994).

51. I. Arad, et al., *The Vitamin C Status of Formula-Fed Preterm Infants*, 3(2) AM. J. PERINATOL. 104-106 (1986); K. Heinonen, et al., *Plasma Vitamin C Levels Are Low in Premature Infants Fed Human Milk*, 43(6) AM. J. CLIN. NUTR. 923-24 (1986).

52. C.A. Clemetson, Barlow's Disease, 59(1) Med. Hypotheses 52-56 (2002).

53. C.A. Clemetson, *Child Abuse or Barlow's Disease*? 45(6) PEDIATR. INT. 758 (2003).

54. A. Bursali, et al., A Case of Infantile Scurvy Treated Only With Vitamin C: A Forgotten Disease, 75(3) ACTA ORTHOP. BELG. 428-30 (2009).

55. S. Ratanachu-Ek, et al., *Scurvy in Pediatric Patients: A Review of 28 Cases*, 86 J. MED. Assoc. THAI. S734-40 (2003); O. Fain, *Musculoskeletal Manifestations of Scurvy*, 72(2) JOINT BONE SPINE 124-28 (2005).

56. M. Grünebaum, et al., *The Radiographic Manifestations of Bone Changes in Copper Deficiency*, 9(2) PEDIATR. RADIOL. 101-104 (1980).

57. R.V. Thakker & J.L. O'Riordan, Inherited Forms of Rickets and Osteomalacia, 2(1) BAILLIERES CLIN. ENDOCRINOL. METAB. 157-91 (1988).

58. H.F. Deluca, Vitamin D-Resistant

Rickets: A Prototype of Nutritional Management of a Genetic Disorder, 8 CURR. CONCEPTS NUTR. 3-32 (1979).

59. C.R. Paterson, *Bone Disease and Fractures in Early Childhood*, in CHILD ABUSE (R.A. Turner & H.O. Rogers eds., 2012).

60. A. Lucas-Herald, et al., *Prevalence* and *Characteristics* of *Rib Fractures* in *Ex*-*Preterm Infants*, 130(6) PEDIATRICS 1116-1119 (2012).

61. C.R. Paterson & E.A. Monk, *Clinical* and Laboratory Features of Temporary Brittle Bone Disease, 27 J. PEDIATR. ENDOCRINOL METAB. 37-45 (2014).

62. Paterson, supra note 1.

63.http://www.acf.hhs.gov/sites/defau lt/files/cb/cm2012.pdf#page=66 (accessed 1/4/2015).

64. K.P. Hymel, American Academy of Pediatrics, *Distinguishing Sudden Infant Death Syndrome From Child Abuse Fatalities*, 118(1) PEDIATRICS 421-27 (2006). ■

About the Authors

John Cannell, M.D., is the founder and



executive director of the Vitamin D Council, a nonprofit dedicated to educating the public and professionals about vitamin D. He is a board certified psychiatrist with

extensive forensic experience.

John Cannell, M.D.

Vitamin D Council 1411 Marsh Street, Suite 203 San Luis Obispo, CA 93401 805-439-1075 E-MAIL jjcannell@vitamindcouncil.org

Brandon M. Sanchez, J.D., is a trial lawyer



from Missouri. He is a graduate of Gerry Spence's Trial Lawyers College and the National Criminal Defense College, and he regularly lectures to attorneys on

topics related to criminal trial work.

Brandon M. Sanchez

Law Office of Brandon M. Sanchez LLC 565 Virginia Avenue New Madrid, MO 63869 573-748-6222 E-MAIL Sanchezlawofficellc@gmail.com WEB SITE www.sanchezattorney.com

46