Journal of Research in Philosophy and History
Vol. 6, No. 2, 2023
www.scholink.org/ojs/index.php/jrph
ISSN 2576-2451 (Print) ISSN 2576-2435 (Online)

Original Paper

Identifying the Failure to Recognize, Acknowledge, and Correct the Epidemic of Vitamin D Deficiency/Infantile Rickets (VDD/IR) and Its Role in False Accusations of Abuse in Infants

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Received: March 1, 2023 Accepted: March 13, 2023 Online Published: March 19, 2023

doi:10.22158/jrph.v6n2p1 URL: http://dx.doi.org/10.22158/jrph.v6n2p1

1. Introduction

Infantile rickets (IR), caused by vitamin D deficiency (VDD), is pervasive. Without vitamin D (VD), its function to deposit available calcium in the blood, into new cartilage, the precursor to bone, to form strong calcified bone, is severely diminished. Calcification of cartilage lags behind cartilage formation. Too much softer cartilage and less solid and abnormally structured bone, results. With 3 million plus newborns born per year with VDD (Note 1) per year, out of 4 million being born in the US, it is the most common form of metabolic bone disease (MBD), by a wide margin. VDD has been shown in many studies, to persist into the child-rearing years for the majority of birthing mothers (up to 80% of mothers (Note 2)). These mothers pass the deficiency to their fetuses during pregnancy and during this time, VDD causes fetal and infantile rickets (IR), the bony manifestation of VDD. At birth, the bony impacts of gestational VDD weaken the skull and during labor and delivery, with extreme deformation during contractions, almost 2 million cases brain bleeding referred to as of perinatal subdural hemorrhage (PSDH) occur. Pressure on the weakened skull structure causes deformation and compressive damage to the covering around the brain, just under the bone, the dura, which then bleeds forming perinatal subdural hemorrhage (PSDH).

Vitamin D Deficiency and infantile rickets (VDD/IR) are synchronous and can be regarded as parts of a unified entity. VDD/IR is easily recognized on plain x-ray films. Any degree of VDD, along a spectrum, causes a commensurate degree of IR and with VDD at epidemic levels, IR is present to some degree in at least 60%-90% 3(Note 3) of all newborns in the U.S. Virtually all cases of IR coming to medical attention via accusations of child abuse are caused by VDD (Note 4).

VDD/IR, being pervasive, is proposed herein to be the root of a majority of false accusations of abuse. With readily available supplements and widely available testing, the intentional failure to correct this

easily correctable condition and address its impact on false accusations of abuse, is inexcusable. It is a stain on pediatrics, obstetrics and all of medicine.

Maternal VDD, woefully under tested for (Note 5), sets up fetal IR, which in turn, sets up rickets related birth trauma and complications for the fetus and then the newborn. The downstream complications from perinatal rickets result in a broad spectrum of impacts (from mild to severe) on the fetal skeleton, the brain, and in too many cases, the wellbeing of families impacted by false accusations of postnatal abuse based on the misdiagnose of infantile rickets.

The consequence is that well-researched and documented rickets findings, known for centuries (Note 6), are being misused or disregarded, to misdiagnose abusive head trauma (AHT), including abusive shaking, previously shaken baby syndrome (SBS). Separate from SBS, supposed abusive fractures, actually rickets findings, are being used to misdiagnose bodily physical abuse (PA) to augment abuse accusations.

After the AHT or a fracture misdiagnosis is made, the findings are then inaptly alleged, against many innocent caregivers, to be the result of felonious abuse. These misdiagnoses, buttressed by abuse of academic power, unsupported false certainty statements asserting an abusive etiology, are powerful drivers within the legal system. The entire process is an insidious, and unrecognized, form of iatrogenic child abuse perpetrated by false accusers. Unwitting or not, we can all agree that any pattern of generating false accusations should be exposed, acknowledged, and corrected immediately.

1.1 The Impact of VDD/IR

The impact of VDD/IR on children begins at conception with VDD pregnant women. Unknowingly, and in conflict with maternal instincts to give birth to healthy babies, mothers pass to their babies a slightly worse deficiency, which is about 80% of their vitamin D (VD) levels (Note 7). These babies are then born, unbeknownst to these deficient mothers, with IR already established in the fetal skeleton.

The degree of IR seen in each newborn also defines a potential for complications in the birth period and during the first year of life, all related to VDD. The complications of fetal VDD/IR include birth related damage from under mineralized fetal bones in the skull, appendicular, and axial skeleton. This undermineralization (when measured) and sub-therapeutic VD levels are constant and reliable findings, and indicators, of infantile rickets.

After birth, infant VDD/IR causes the development of structurally weakened bones in the context of undermineralization and abnormal bone structure. This manifests as abnormal metaphyseal growth plates, underdeveloped cortexes (outer layer of solid bone), and malformed trabeculae (the inner structure of the marrow cavity) of long bones, all amplified by rapid growth and occasional non abuse, accidental fractures. These findings related to bone density can potentially be measured by DEXA scanning, but it is not in use.in infants. It is used regularly in postmenopausal women and older patients to quantify bone density/strength.

1.2 The Two Prongs of VDD/IR Related Misdiagnoses of Abuse

The first VDD/IR track (prong) related to false accusations of AHT, starts with intracranial bleeding at birth caused by a VDD weakened skull. The second prong is misdiagnosis of VDD based rickets as physical abuse (PA).

1.3 The AHT Prong

PSDH occurs in about half of all deliveries judged to be a "normal" (46% of 4.0 million newborns per year, at least 1.8 million VDD newborns). The lack of structural integrity of the skull due to VDD, allows for extreme deformation of the skull during labor contractions. The intrusions that occur, mixed with normal fetal hypoxic stress during contractions, result in intracranial damage to the capillary layer in the dura that causes bleeding which can be called perinatal subdural hemorrhage (PSDH). PSDH, if problematic, remains subtly detectable, and more clearly evident after birth with the later identification of its complications during the first year of life. This includes unresolved cases of PSDH (Note 8), the formation of a chronic subdural hemorrhage (CSDH), rebleeds, varying degrees of pathologic increased intracranial pressure (ICP), and accelerated skull enlargement leading to macrocephaly (enlarged head). In such cases, at almost any stage postnatally, any degree of identifiable blood in the head, old or new when discovered, under almost any circumstances, seemingly reflexively, will invoke the misdiagnoses of SBS and AHT.

1.4 The Physical Abuse Prong

The second prong of false accusations with VDD/IR is related to the dysmorphic bones and pathologic fractures seen with IR, then being misdiagnosed as "physical abuse" (PA). Osteoporotic, under calcified, rachitic bones when deformed or damaged, are regularly being misdiagnosed as abusive fractures and designated as PA, often defying logic and common sense with the intent of amplifying the abuse allegations.

Both of these prongs of misdiagnoses of abuse: (AHT) and infantile rickets (IR/PA) are directly related to preexisting vitamin D deficiency.

Thankfully, and ironically, for those capable and willing to recognize what was happening, in an evidenced, and probability based framework (Note 9), the increased number of skeletal surveys allowed, in short order, first, the validation of the pervasive nature of IR (Note 10) and secondly, understanding how VDD/IR were being misused to create false accusations of abuse on those two tracks. That is the subject of this article.

1.5 The Campaign of Disregarding VDD/IR to Portray Rickets as Abuse

This campaign, IR as PA, was first embraced to amplify an abuse narrative by creating a false sense of a '1-2 punch' in advancing abuse. Without the obvious, but ignored or suppressed, linkage to VDD, rickets findings were portrayed to the unknowing, as an entirely separate, second set of abusive behaviors. This is in addition to the already alleged AHT, which was based on extravasated (leaked) blood in the head. Given that more than half of all babies have rickets findings due to the high incidence of VDD, almost all alleged AHT cases will have concurrent IR findings that can and will be

misdiagnosed as physical abuse. When IR/PA findings are added to accusations of AHT and the cumulative misdiagnosis of abuse is more devastating and more likely to end in a false conviction.

1.6 Academic Failure

When a child abuse workup yields no external, or only minor, head or bodily trauma, along with the nonappearance of neck imaging findings (no neck soft tissue swelling or hemorrhage), both physical impact (Note 11) and abusive shaking (Note 12), as causes of brain bleeding, are effectively ruled out (Note 13). Biomechanical analysis has shown that neck damage in trauma occurs at $1/10^{th}$ the force that causes brain bleed (Note 14). Why these rule outs are ignored, has no basis in probability or evidence based science, and competent academics have a duty to acknowledge that. The proven biomechanical reality (Note 15) regarding the insufficient forces when analyzing the biomechanics of shaking and impact is ignored by CAPs, radiologists, and other pro-abuse providers, for no valid reason. The lack of appendicular tenderness, swelling, discoloration, decreased range of motion, or any history of conventional fracture related clinical findings supports IR as a common form of asymptomatic metabolic bone disease (MBD), not abuse.

1.7 The History of VDD

Understanding, and eradicating rickets, a centuries old malady, was one of the first focused massive efforts of modern medicine, and went on for 50 years (1900-1950). During that time, x-ray came into use (circa 1900) and sun exposure was identified as preventative of rickets by the US government (1931); all before Vitamin D was discovered in 1937. The earliest efforts to decrease rickets were first promoted in that timeframe (early 1930's) to parents in what now seem like quaint pamphlets (see Figure 1).

Figure 8. Brochure of the US Department of Labor promoting sensible sun exposure in children in 1931.

Figure 1. Dept. of Labor pamphlet, circa 1931

Quantum advances in pharmacology were occurring in that period as well, which lead to the discovery and manufacture of supplemental vitamin D. Later, the integration of pathologic findings that had been seen at autopsy for centuries into modern medicine (see Figure 2), collectively created a usable understanding of VDD/IR, and a path to eradicate VDD/IR emerged.

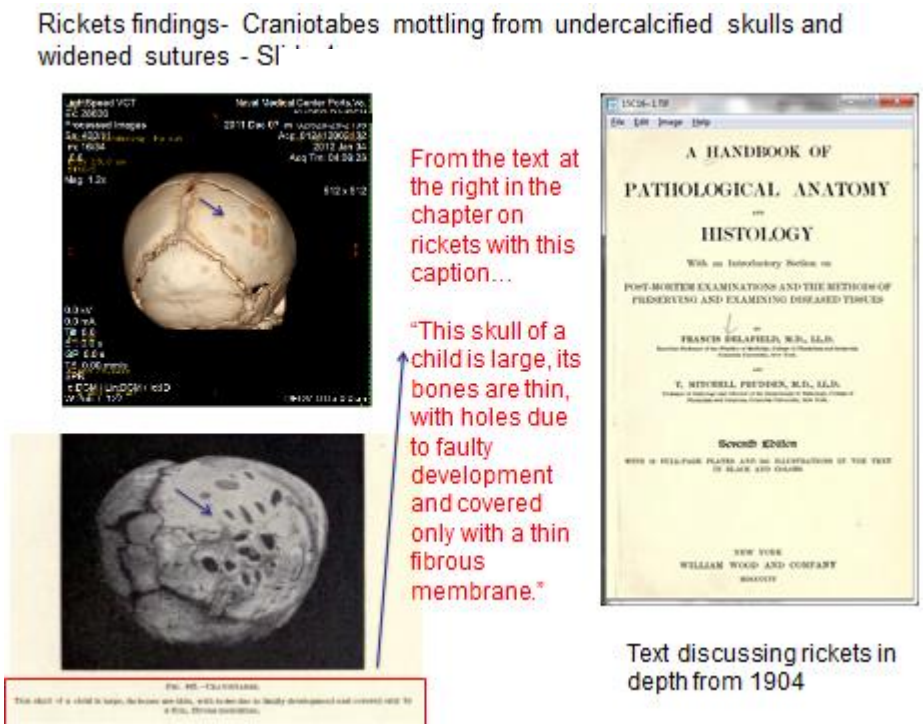


Figure 2. The contrast/similarity that connects the past to the present diagnosing rickets in the head. Mottling of bone with modern 3D CT imaging (left upper image) and at autopsy in ~1904, (just below), with each delivering essentially the same clear picture of severe rickets. The 3D CT image is from a case of accusations of abuse in which VDD/IR was disregarded.

By 1950, vitamin D supplements were being put into use and the scope of IR imaging findings had been cataloged (Note 16). It was easy to identify IR then (and now) via plain x-ray films and later, VD testing. Every radiologist of the era could identify mild to moderate IR, on plain films routinely, and many articles were written to catalog the imaging findings and clinical course of fetal, perinatal, infantile, childhood, adolescent, and adult rickets (Note 17).

So overwhelming was the evidence that VDD caused IR that the government was able in 1950, (now seemingly impossible), to mandate the addition of VD to all commercial milk sold (and it still is!). It was thought that the end of the scourge of VDD/IR was set into motion. The role of sunlight, supplements, and calcium in milk generated the parental calling cry "drink your milk for healthy bones", and it worked! For a generation of Americans from 1950 to ~1970, rickets was recharacterized as a disease of the past. Training in the recognition of VDD/IR decreased and its well documented imaging findings, especially in mild to moderate cases, were no longer a meaningful part of radiology training. The ability to recognize the bony manifestation of mild to moderate rickets in the skull, chest, appendicular, and axial skeleton, faded into the past.

1.8 The Reemergence of Rickets, The Failure to Recognize It, and the Impact on the Fetal Head

Within a few years of 1970, an unexpected and unrecognized decrease in VD acquisition habits by parents for their children, occurred. The result was that VD levels in the general pediatric population incrementally started to decrease and known findings, specifically related to mild to moderate VDD/IR, began to resurface in imaging specifically skeletal surveys, almost exclusively done after any allegation of abuse. This was the beginning of a new phase of the impact VDD. The initiative of the 1950's, which was successful in decreasing VDD/IR, was being progressively impacted and eroded in the minds of mothers by an emerging aversion to sunlight and cow's milk. By the 80's, VDD was on the rise and the trends that exacerbated the aversion to sun and VD fortified milk, were on the rise as well.

During these years, sun exposure was being overly promoted as a cause of skin cancer, and the use of enhancing pharmaceuticals and growth hormones in milk cows, raised fears in mothers of a variety of poorly defined negative impacts. Both aspects of this emerging thinking negatively impacted VDD levels across large swaths of the population. Sun exposure and milk consumption were decreasing dramatically. Thus, the two key sources of Vitamin D and calcium in modern society, sun exposure and vitamin D fortified milk, were falling out of favor. Compounding these trends was an increasingly inside lifestyle and widespread use of sunscreen that blocked UV rays from making VD in the skin of children, even when playing outside. These trends, combined, fueled a progressive resurgence of VDD/IR that incredibly went unrecognized and unstudied for nearly 4 decades (1970 to 2006 (Note 18)). During these years, as you would expect, mild to moderate rickets findings in the skull and long bones, seen on imaging, especially in the context of an increasing number of abuse allegations and skeletal surveys, were seen more frequently and in a more advanced state.

1.9 The Impact of CT Scanning on False Accusations

With advent of modern child abuse pediatrics (CAP), defined herein as after 1975, there was an intersection of the resurgence of VDD/IR, and the beginning of CT scanning, which became available at that time (~1975). At that point, pediatric child abuse pediatricians were readily able, for the first time, to see intracranial hemorrhage caused by VDD/IR in newborns (and how frequently it occurred), This perinatal subdural hemorrhage (PSDH) with the newly available advanced imaging (CT) was seen in a subset of almost ½ of all babies from birth trauma. Even without external findings after the birth period, there were many potential complications to come. Regardless of later developments, PSDH became the first prong (of VDD/IR findings) leading to widespread false accusations of shaking abuse under the rubric of the fabricated SBS hypothesis. When this began SBS was nothing more than a hypothetical and unstudied fantasy that relied solely on SDH and a fabricated link to shaking promoted by CAPs.

Regarding the importance of the structural integrity of the skull, it was placed on a long list of rickets findings cataloged by Ayoub (Note 19) and others (Note 20). The most impactful findings in this period was, in fact, a small group of findings in the skull, collateral to PSDH, that collectively weakened the structural integrity of the skull via undermineralization and lead to traumatic brain damage at birth.

This group of impacts from IR was collectively referred to as craniotabes. This rickets finding and others are easily seen, but not identified as rickets related on imaging of the head and skeleton of VDD newborns~~,~~ Craniotabes is defined by under-calcified, egg shell thin, skull bones coated with thick cartilage on both sides, that should be calcified, but is not due to VDD. Below are lateral and anterior posterior x-rays of the skull of VDD infant showing thin calcification of skull bones or areas with absent calcium. (See Figure 2b).

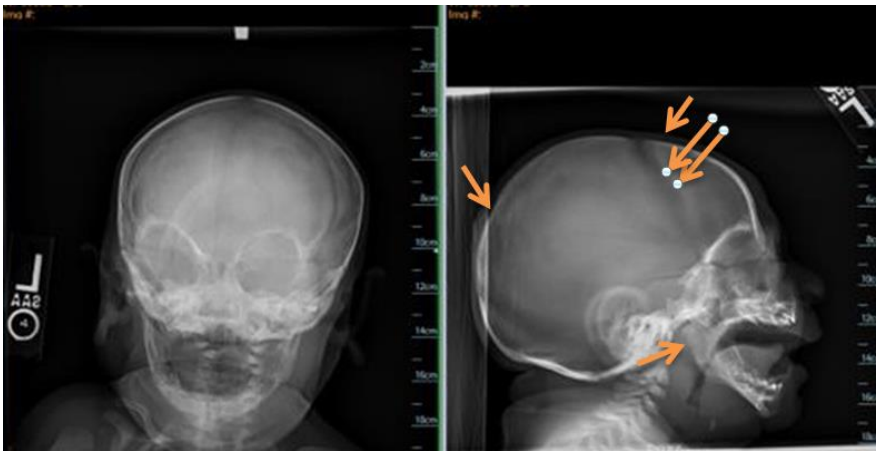
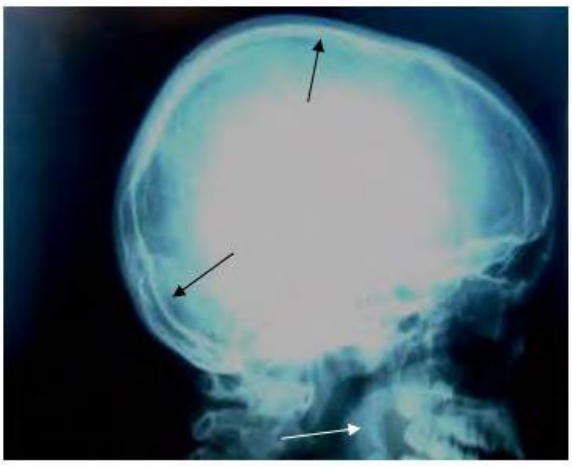


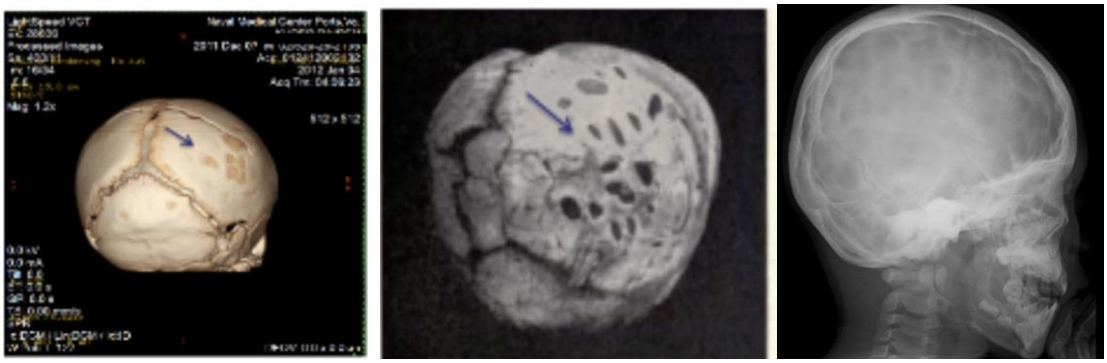
Figure 2b. Right image is a lateral view with no calcium in skull between single arrows at top. Where there is calcium, it is thin and minimal. Enlarged sutures (the space between bones), is another rickets finding seen in right image at double arrows. Absence of calcification of the angle of the jaw at lower single arrow is noted as well. This is another sign of retarded calcium deposition caused by VDD. A normally calcified skull of a 2 month old below (see Figure 2c)



Figurere 2c. Here is a normally calcified skull of a 2 month old for comparison. Note differences all around the skull with respect to thickness and density of the skull bones and the calcified jaw is highlighted as well for comparison (white arrow)

What is seen in severe craniotabes is extreme undercalcification and an excess or abundance of uncalcified cartilage that forms a weak structure of the newborn skull. The skull in this state, functions more like a leather football helmet than a non-deforming modern professional football helmet. The skull, exposed to the compressive forces of labor contractions in an undermineralized state, is unable to protect the brain and its covering, the dura, and the dura's delicate capillary bed. What results is birth related deformation, intrusion, overlapping of bones, and other physical damage the dural capillary bed which is the source of blood for the PSDH and residual areas of damage that cause the complications when acute SDH (PSDH) turns chronic as discussed below.

The undercalcification can be so extreme that some areas of the skull result in areas that show no identifiable calcification on plain films. This results in the appearance of uncalcified pothole-looking like mottling, and in some cases, the irregular deposition of the calcium that is deposited in skull bones on plain films (called Luckenschadel), and a hammered metal appearance. These are diagnostic markers of craniotabes and infantile rickets. (See below Figure 3 a, b, and c.)



a b c

Figure 3a, b and c. Figure a (left, shows 3D reconstruction of a skull CT). Figure b (middle) we see an older autopsy case showing a photo of a mottled, moth-eaten skull from rickets similar to 3a. Figure 3c (right) the image shows Luckenschadel (hammered metal appearance) in an older child with infantile rickets. Note variation in density with lighter and darker gray areas appearing on x-ray simulating hammered metal.

MRI imaging added more detail to the events that occur. MRI had done to a woman in labor shows changes in shape of a baby's head during normal labor contractions. What was seen is overlapping skull bones, extreme deformation of the head, transient vascular venous and arterial occlusion, and direct pressure on the brain (Note 21) with compression and intruding skull bones into the brain and brain space. When this occurs, damage to the dura and bleeding can be found on CT and MRI after birth in about ½ of all newborns (46%) (Note 22). The bleeding seen is PSDH and is highly likely to be the beginning of a sequence that leads to complications.

1.10 The Frequency of Complication with PSDH

The frequency of complications is reported as 0% by pediatricians. This is clearly false and misleading. 3 established studies (Note 23, Note 24, Note 25), in addition to Rooks original study (see Rooks 2008 above), show complication rates in the 40's percentile in the 3 studies of premies, full term newborns, and the elderly. These percentages convert into more than ½ million babies with complications of PSDH every year. It is the common complications being misdiagnosed AHT and that form a large percentage of false accusations of AHT.

At birth, the dural hemorrhage and occult brain damage that occurs resulting in visible PSDH and is almost never looked for, yet is present in about 1,800,000 out of near 4 million, "normal" deliveries (Note 26) in the United States (46% of 4 million births per year in the US). Any successful efforts to identify PSDH in the neonatal period would lead to increased neurological monitoring, special handling, early treatment if needed, and decreased false accusations of abuse related to that knowledge (Note 27).

So what happens to the almost 2 million perinatal subdural hematomas?

The statement, that all PSDH cases return to normal, though, delivered to the unknowing by authoritative figures in white coats, allows in legal proceedings, for birth related trauma to be inappropriately removed from the differential diagnosis of nonabusive SDH in the first year of life.

Squier (Note 28) showed a range of end points with CSDH formation, some visible on imaging and some only at autopsy. The multiple studies across an array of patient's ages (infant to elderly), show a consistent conversion rate of ~40% for the acute SDH (ASDH) to become an identifiable chronic SDH (CSDH) (Note 29, Note 30, Note 31).

These four peer reviewed studies define and support a spectrum of frequencies for CSDH formation after ASDH from a maximum of 40% of 1.8 million: 720,000 cases, to 1% of 1.8 million: ~18,000 cases each year (the low end of Rooks' findings, and a likely underestimate due to incomplete follow-up protocols in her study).

Ironically Rooks', even with what is touted as the minimal 1% conversion rate and 18,000 cases that will result, is cited incongruously by CAPs as "proof" that all children get better. Getting better was defined incorrectly as PSDH not being visible on imaging. Squier clearly showed that other occult damage is the dominant form of residual damage/complication. If Rooks' actual findings, recurrent ASDH and CSDH in 1 of the only 18 patients that had follow-up (5%), when using 18 as the denominator: 1/18 = 5%). 5% would convert to about 200,000 cases of PSDH to CSDH (5% of 4 million). This makes the notion that they all get better, false by a wide margin.

The conversion of ASDH to CSDH takes about 3 weeks to manifest. By far, almost all of the newborns are asymptomatic (99.9%+). What we do know, however, is that consistently each year about 1000-1500 PSDH to CSDH babies with complications, will have apparent life threatening events (ALTEs). ALTE's (Note 32) are a class of events that motivate parents to seek medical encounters that lead to child abuse pediatricians. Furthermore, when CSDH findings and complications are present, the assumption, and misdiagnosis of the findings as abuse, is almost certain.

1.11 Common Clinical Events in the Neonatal Period Related to PSDH

Below are some, but not all, common events that are ignored or misused to allege abuse. When the actual calculated probability of abuse is done, the probability of abuse is miniscule, as explained below using Bayes Theorem of Probability. Analysis shows that misjudgments and mistakes in these areas ruin lives. It can be calculated in almost all cases, that the weight of evidence for nonabuse dwarfs the unreliability of the unsupported beliefs of accusers being misused to generate false accusations of child abuse in case after case.

Here are the common clinical events in the neonatal period that are nonabuse indicators of complications of PSDH.

1) **Caput secundum** is molding (reshaping of the head from labor), and extreme molding and instrumentation, are indicators for concern of PSDH. Instrumentation, including forceps and definitely, vacuum extraction increases the probability of severe PSDH to near 100%. Below are images of common complications.

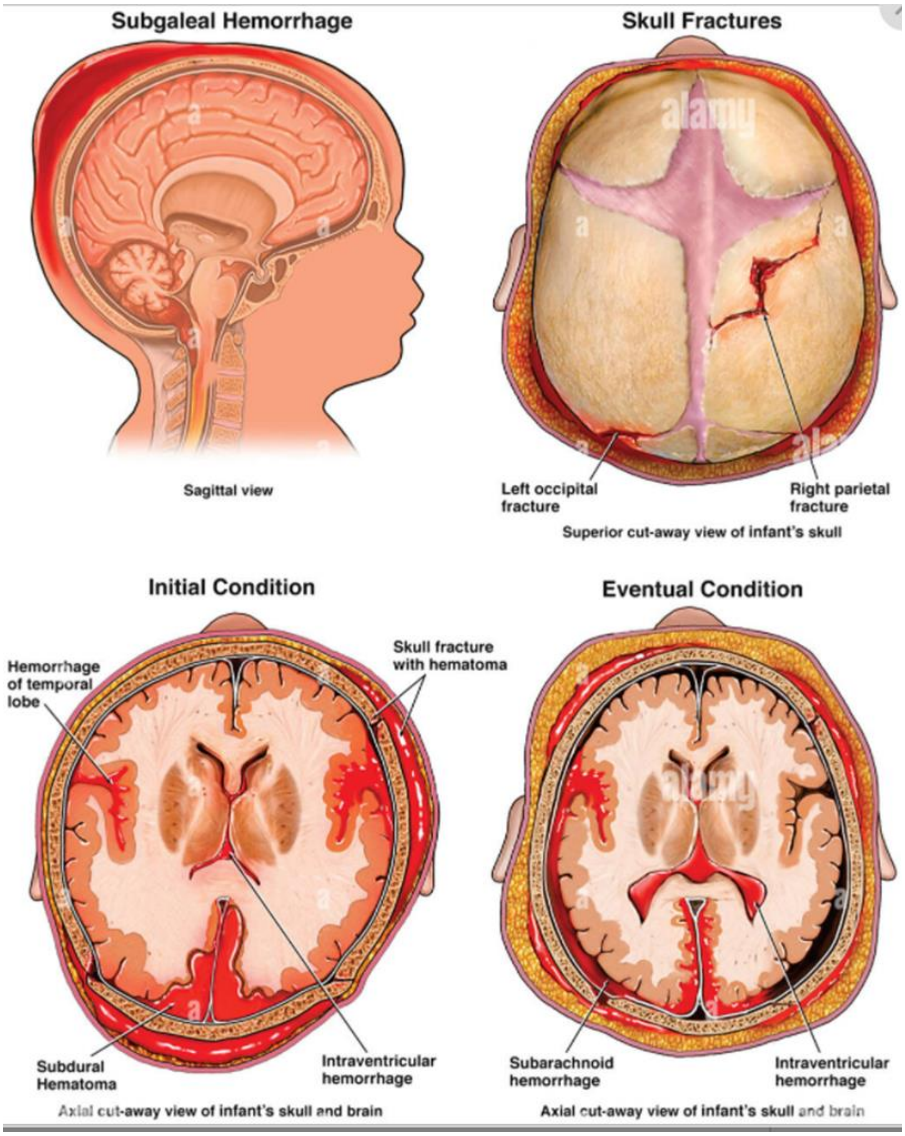


Figure 4. Complications of vacuum extraction and instrumented (forceps) delivery

2) **Fussiness** results in positioning problems when breastfeeding, latching and **feeding problems** can be identified and may be all that is noted in the birth period. Since these findings are normally transient, they are almost universally disregarded or dismissed as normal postnatal behavior. These symptoms, in babies with PSDH, can be the harbinger of a spectrum of future complications related to CSDH. That occurs in a small fraction of the 40% (Note 33), of that subgroup with PSDH and complications.

3) **Undetectable nondisplaced inner surface rib cracks** from deformation of the rib cage during labor contractions can occur and are a common finding. These cracks, or more accurately, "distraction fractures" (extreme stretching of the inner outer covering of bone, the cortex), can only be identified weeks to months later on plain chest x-rays by the presence of fracture healing callus (the healing reaction). Ultrasound has recently been shown to be reliable to identify, in the newborn period, the common distraction fractures that occur (Note 34), but is not in current use.

How the cracks occur is that during contractions, curved ribs are straightened via compressive deformation of the rib cage from oval to round. Distraction (stretching/straightening) on the inner surface of the cortex of the ribs is the companion to impaction (compression) of the cortex on the opposite side. Impaction does no create a defect or an unstable fracture which is considerably more painful over weeks. Below is a graphic representation of the rib cage's deformation (Figure 5) during a labor contraction that can lead to cracks (more common; not visible) and fractures (less common; visible on US and imaging) in the newborn ribs.

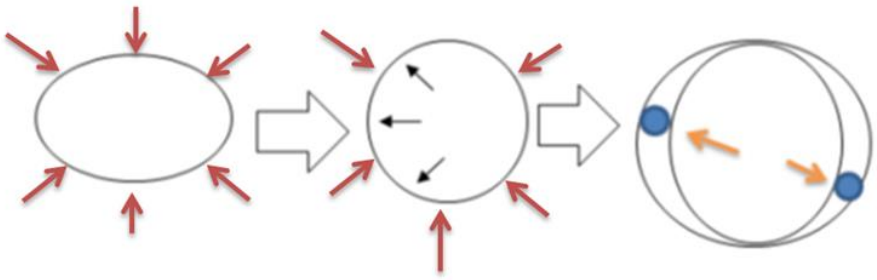


Figure 5. The substantial forces during uterine contractions are applied equally all around (red arrows in the far left side of the graphic) to the rib cage which is normally oval in shape. This transforms the oval to a more circular shape and stresses the boney ribs and if deformed enough, to a breaking point where cracks or overt fractures can occur. (Black smaller arrows). With VDD/IR the ribs are much more prone to extreme deformation cracks and fractures to occur at less than the normal force required to break ribs in healthy bones. Because the bones are diseased they are called "pathologic fractures" (fractures in diseased bones). In the far right configuration, the small blue circles are the baby's arms. The arm's location is variable (anterior, lateral, and posterior) in the uterus and during labor they move around. Where they are during a contraction is where deformation and stress forces are highest. The movement of the arms accounts for unpredictable locations of cracks, fractures, and callus seen weeks later on skeletal survey. Thus, rib callus positioning/location, miused to allege abuse, has no probative value (in spite of statements from CAPs that they do), in diagnosing abuse vs VDD/IR causation based osteoporosis.

In the end, the interior crack is splinted by the intact outer surface of the rib. Cracks cannot be seen on perinatal chest x-rays (CXRs) taken for other reasons. With the splinting, the situation is less painful then any postnatal conventional through and through fracture. The full fractures are unstable and painful to touch and obvious. The cortical cracks on the inside surface, are less painful. The callus or calluses, the remnant of healing from this birth trauma, are the only way these distraction (straightening) crack/fractures) can/will be seen. Once the calluses are seen, however, they are then speculatively alleged to be postnatal physical acts of violent abuse, when no abuse has occurred and no expected

postnatal chest tenderness was noted by caregivers, medical providers, family, or anyone seeing the baby with any regularity after birth.

4) **Intranatal traumatic fractures in the axial skeleton** that result from dysmorphic weak bones being subjected to normal nonabusive forces (e.g. intranatal collarbone fractures, deformation based rib cracks, occasional skull fractures, etc.) can and do occur too. These injuries are not detected at birth. Later, when seen in the context of a child abuse investigation, they are almost always alleged to be intentionally inflicted postnatally. Once this occurs, the likely status of parents as decent people is disregarded or dismissed in a large percentage of cases where they are then portrayed as repeat abusive liars. The designation as liars has tremendous negative inflammatory impacts personally and in the hospital and in a number of legal ways, generating negative impressions that are quite incriminating and lead to litigation.

5) **Visible abnormal structural bone morphology** seen on skeletal surveys in weeks old newborns, other postnatal imaging, and at autopsy, will be mischaracterized as abusive injury; ignoring known imaging findings of IR and the lack of any identifiable clinical trauma.

All these findings when they are seen in the weeks after birth, are then almost universally and unthinkingly, misdiagnosed as SBS/NAT/AHT and/or physical abuse.

1.12 Common CSDH Findings Used to Misdiagnosis Abuse after the Birth Period

Below is a list and explanations of common complications and events related to false accusations that occur beyond the 30 day long neonatal period.

1.13 Rebleeds

1) **Recurrent bleeds from the sites of PSDH**, now chronic SDH This related damage will be alleged to be new, unrelated, acute postnatal abusive head trauma, even in the context of no acute external trauma or neck damage. Evidence of abnormal fluid filled spaces around the brain (called hygroma) and mixed density fluid (spinal fluid with particular matter from old blood and inflammatory debris mixed in) support the conversion of PSDH to CSDH with complications.

2) **Ongoing, or intermittent, rebleeds** from the damaged PSDH areas **further damage the dural reabsorption system**. The new hemorrhage exacerbates the decreased capacity to handle the steady production of cerebrospinal fluid from the choroid plexus, where CSF is produced in the ventricles deep in the brain. The obstructive process is progressive and pressure increases incrementally. This has the potential to cause fatal increases in symptomatic cerebral hypertension (increased intracranial pressure) and perpetuates the accelerated enlargement of head circumference and hygroma to pathologic levels.

3) **False reassurance** is also in play during this period. Parents observing mild to moderate neurologic symptoms related to CSDH, including excess head enlargement, brain infections, and rebleeds, often are misdiagnosed as reflux or other gastrointestinal maladies (colic, gas, formula related problems reflux or regurgitation) or normal findings. This will lead to encounters in which the gravity of the situation is missed completely by the provider, and false reassurance is offered to the parents. False

reassurance that "things are normal" is a key mistake, causing delayed interventions in the next iteration of the underlying problem when similar or slightly worse symptoms persist or recur. With a progressive trajectory, these false assurances can lead to later more serious presentations, fallacious accusations, and even, out of hospital deaths.

4) Small recurrent rebleeds over months can create **unexplained anemia** as well (without evidence of hemorrhage or blood dyscrasia). These dramatic decreases in hematocrit are frequently mis-ascribed to the small volumes of blood taken related to the testing of the current incident (which is insufficient to cause anemia) or other blood draws (which occur after anemia is identified and are also generally insufficient to cause significant anemia). When anemia is extreme, transfusions must occur.

5) **Layering of old free blood in dependent areas of the brain** can be noted on advanced head imaging, from multiple rebleeds and as a result, the layers are speculated to be from unspecified multiple acts of abuse from the past. Recurrent bleeds from CSDH, the more likely cause, are ignored or dismissed. When this is seen, authorities cast parents as serial abusers and portrayed as monstrous. Serious charges of continuous child abuse, resulting in conviction with life sentences, frequently follow. Dynamics with social services and the doctors are altered further and the parents are treated as criminals while grieving about their child's medical events that had occurred or death.

As the falsely incriminated parents' sense of hopelessness, fear, and anxiety related to these accusations increases, they are watched closely by accusers who unjustifiably incorporate into their false accusation narrative, the instability of the parents**, interpreting the deep grief, anxiety and trauma they are living through, as incriminating behaviors.** These emotions and the escalation of a variety of stress related behaviors, in all nonabuse cases, in the current environment, are justified. Compounding the instant tragedy for the caregiver, the behaviors, whatever they are, are then unjustifiably incorporated into the false abuse narrative as incriminating behaviors by guilty parents, to energize the false accusations.

6) On skeletal survey, **dysmorphic bone growth** with transient configurations are likely to become more obvious and to be misinterpreted as postnatal abusive fractures when they are expected rickets findings (i.e. spurring and breaking and other rickets findings) and that are directly linked to VDD too. In some cases, these rickets presentations alone are enough to allege physical abuse, even when the first prong of misdiagnosis, intracranial blood, is missing.

7) **Accidental pathologic fractures can occur** too with normal handling in undiagnosed rachitic infants. With the reflexive dismissal of VDD/IR as causative, these findings will lead to false accusations of intentional bone breaking behaviors, in even ultra-low risk caregivers, who are overwhelmingly likely to be innocent.

8) **Discovery of the healed ribs cracks** from birth or healing collarbone fractures, both related to VDD/IR, are then alleged to be postnatal abusive fractures.

It remains important to keep in mind that these findings in the first year of life are, in a majority of cases, all directly linked to VDD/IR and increased ICP

1.13.1 The Formation of CSDH and Its Complications

With VDD/IR responsible for PSDH, VDD/IR is, likewise, the root cause of CSDH and its complications. When the conversion of PSDH to CSDH at about 3 weeks of age of the hemorrhage is identifiable, a new set of findings, related specifically to CSDH, and a broad range of vulnerabilities arise, very different from the acute SDH event.

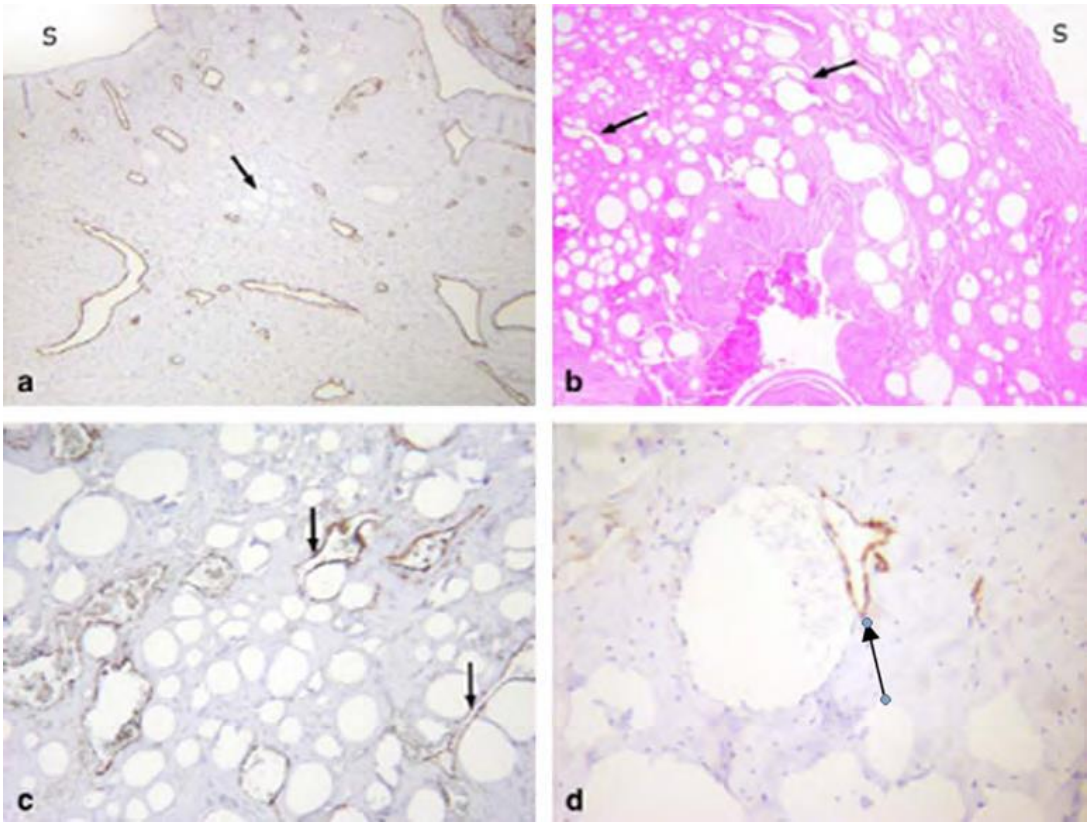
The most identifiable external finding of CSDH in infants in the first months of life, and a marker for complicated birth related trauma is accelerated head circumference (HC) growth from persistent low and medium grade increased intracranial pressure (ICP) (Note 35), an element of the CSDH picture, usually dating back to birth. Understanding how the pressure forms and its impacts on the infant brain, is necessary to get a complete picture of key events.

1.14 Accelerated Head Circumference (HC) Growth

The accelerated HC growth is caused by the obstruction of the reabsorption of cerebrospinal fluid (CSF) in the birth damaged dural capillary bed. (See Figures 6 and 7). This part of the CSF circulation is the normal reabsorption location and the circulation a necessary component to maintain the central nervous system. CSF is produced at a constant rate. Birth related trauma does not impact the volume or rate of creation of CSF, which is steady and generates pressure that after a bleed can cause pathologic increased pressure. The steady production generates low-grade pressure as it is being produced. That pressure drives the circulation. Reabsorption completes the CSF circulation. Reabsorption occurs after the CSF leaves the ventricles and the brain, fills the space around the brain and spinal cord, and then migrates to the dura through the pia and arachnoid layers of the 3 layered meninges, where reabsorption occurs. This is essential to maintain equilibrium in a normal functioning central nervous system (CNS).

The circulation normally operates in a low pressure environment, but if obstructed anywhere or reabsorption is blocked or obstructed in the dural capillary bed (see Figures 6 and 7, then higher pressures are generated. When this occurs, with the capillary bed not functioning normally at low pressure, marginally understood absorption points in the dura and the brain, perhaps via the lymph system (Note 36), are activated.

CSF, as it moves toward the reabsorption point, after it passes through the arachnoid and then innermost layer of the dura, the dural border cell layer, enters “channels”, (see Figure 7) identified by Squier (Note 37). These channels conduct the CSF to the middle layer of the dura, the intradural capillary bed, where reabsorption normally occurs (see Figures 7 and 8). The interface of CSF conducting channels connecting with dural capillaries is the place where the CSF is reabsorbed and recirculates to the blood (see Figure 7c). The dural capillary bed with its delicate structures and the functions, when damaged at birth, and disrupted, is the obstruction point (see Figure 8).



Figures 6 and 7. Section of the dura in various stages of maturation in infants, showing intradural channels bringing CSF from the dural border cell layer into the capillary bed. In ‘‘a’’, the relationship of the CSF channels (arrow) to the capillaries, which are stained brown and to the sagittal sinus (‘‘s’’), is noted. The channels are unlined and do not stain and the capillaries and vessels have epithelial cells that can pick up the brown stain. In ‘‘b’’ the channels are dense near the sagittal sinus (“s”) and also adjacent to capillaries (the capillary lining is stained darker pink on “b”) at arrows. In “c” and “d” the channels’ relationship to brown stained capillaries is clear. Used with permission by Elsevier under license 3055561168939 from Squier W, Lindberg E, Mack J, Darby S. Demonstration of fluid channels in human dura and their relationship to age and intradural bleeding. Childs Nerv Syst. 2009 Aug; 25(8): 925-31. Figure 1c.

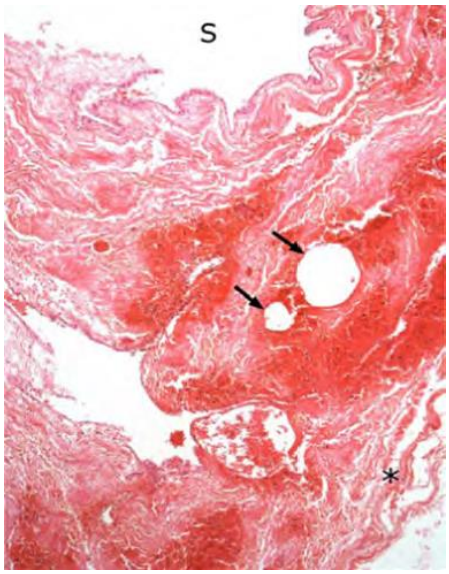


Figure 8. Dural channels (arrows) and intradural bleeding (dense redness) in an infant dural capillary layer, at 17 weeks of age. There is patchy intradural bleeding as well throughout. The compressed structure of the intradural compartment at the dural border cell layer, are separated by fluid filled clefts (*). The sagittal sinus is seen (S). This represents a typical picture of intradural bleeding. Capillaries are no longer clearly visible and the vast majority of the dural channels conspicuously absent. From Squier and Mack's 2009 study of dural channels [19]. Used with permission by Elsevier under license 3055561168939) from Squier W, Lindberg E, Mack J, Darby S. Demonstration of fluid channels in human dura and their relationship to age and intradural bleeding. Childs Nerv Syst. 2009 Aug; 25(8): 925-31. Figure. 3.

Once damaged, the reabsorption, which must occur to maintain the balance between production and reabsorption, must operate at higher pressures. In any case, the malabsorption creates back pressure due to the constant production of CSF and raises intracranial pressure above normal. The elevated constant pressure causes bone movement, akin to braces moving teeth with low pressure applied over time. That low grade pressure pushes the upper and frontal skull bone out, generating what can be seen as "frontal bossing" (see Figure 9 below) and visible or measurable macrocephaly (large head) occurs. Both are evolving over the early weeks and months of life and can be tracked with serial HC measurements done during well baby checks. The accelerated movement of the bones, along with slower steady brain growth, creates the space. The enlarged space is a fluid filled, extra-axial space (outside the brain) that forms all around the brain, and is called a hygroma (Note 38). On imaging with the baby on its back the space will appear larger in the front.

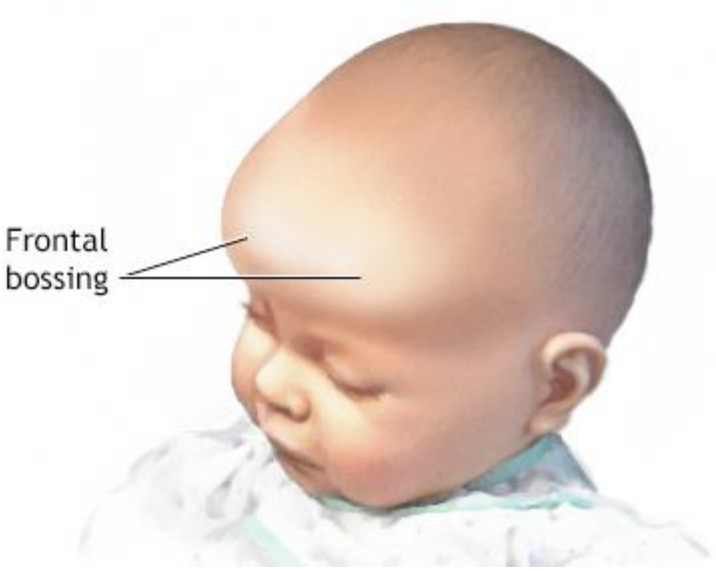


Figure 9. Macrocephaly and resultant frontal bossing

1.15 Hygroma Imaging

The rate of brain growth is the normal driver of skull growth (Note 39), but with pressure elevated around the brain, the size of the skull increases in advance of brain growth. This asynchrony creates what is called "cerebrocranial disproportion" (CCD). The accelerated outward movement increases HC pathologically that is usually measured at outpatient pediatric visits. With even modest vigilance by family pediatricians, often lacking, enlarging HC can be compared to prior percentile rankings of HC for the subject patient, and against the general population, using growth charts or calibrated software. There, percentage rankings by size, can be tracked, and jumps (enlargement) greater than 2 standard deviations from baseline values, and/or absolute values above 95th percentile, signal pathology (Note 40).

The hygroma created in some cases can be quite large. The space, just inside the dural border cell layer, appears to be expansions of the subdural and subarachnoid spaces which are only separated by the porous arachnoid layer which allows CSF to pass through freely. These enlarged spaces fill with CSF fluid and the subdural component often contains easily seen particulate debris collectively identified as "mixed density fluid". (See Figure 10) The particulate matter in the fluid are the remnants of old metabolized blood and inflammatory debris from the reaction to the initial bleed or from common, rebleeding event(s) in these fragile, partially healed, birth-related, damaged PSDH area(s) (Note 41).

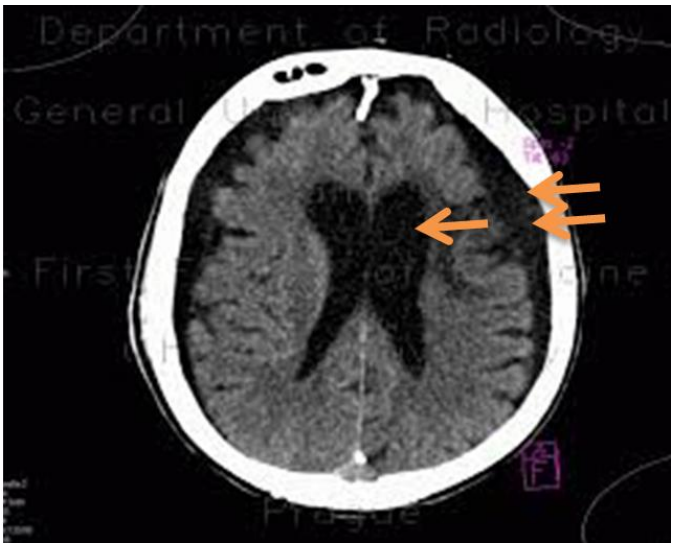


Figure 10. Extra Space around the brain (black space-double arrows) from enlarged skull, creating cerebrocranial disproportion (CCP). Enlarged ventricles at single arrow

If small, but frequent, rebleeds are occurring, over weeks, unexplained anemia can occur (Note 42). Marked anemia without prior hemorrhagic event is a red flag to recurrent small bleeds with minimal or no symptoms. 1-2 ml leaks per day over 90 days can decrease red cell volume (the measure of anemia) by 30-40% (Note 43).

Furthermore, each rebleed can create more obstruction and exacerbate the baseline malabsorption in the capillary layer and increase ICP incrementally. As pressure increases, ICP related findings and symptoms can arise. They can be intermittent, progressive, and/or transitory. History is frequently obtained from parents about persistent or episodic fussiness, feeding problems, decreased appetite, and spitting up, which are symptoms of symptomatic increased ICP and supports the presence of increased ICP (intracranial hypertension). In a tiny fraction of cases, higher intracranial pressure thresholds can be reached that can potentially lead to vomiting, inconsolability; change in cry to high pitched, intracranial vascular compression, ischemia, seizures, apnea spells, cerebro-venous thrombosis (CVT), other ALTEs, and deaths. These higher pressures, if unrecognized, or dismissed as benign, are potentially fatal.

1.16 "Benign" Extra-axial Spaces and Asymptomatic CSDH

The commonality of PSDH and the high conversion rate from acute SDH to forms of CSDH manifests most often into asymptomatic cases of hygroma formation. The hygromas in the asymptomatic group can be diminutive or prominent. In either case, they are now so common, and frequently found on CT's in asymptomatic babies, that over decades, without understanding the role of CSF malabsorption secondary to CSDH, that they have been mischaracterized, ironically again by consensus only (not valid research), as "benign" extra-axial spaces. It is so established in the mind-set of radiologists and providers that there are at least 15 "benign" appellations for this finding in use (Note 44).

Table 1. These different name have been used in the literature for the same or similar conditions

TABLE 1 These different names have been used in the literature for the same or similar conditions

Benign/idiopathic external hydrocephalus[1,78]
Benign familial macrocephaly[79]
Benign infantile hydrocephalus[80]
Benign subdural collections[62]
Benign extra-axial fluid/collections[40,81]
Benign extracerebral fluid collections[82]
Benign communicating hydrocephalus[83]
Benign enlargement of the subarachnoid spaces[84]
Subarachnoid fluid collections[85]
Chronic subdural hygromas[86]
Pericerebral fluid collection[87]
Idiopathic macrocephaly[7]
Chronic subdural haematomas[88]
Subdural effusion[89]

The notion of calling this a benign space, improbable as it might seem, has been effective, perhaps unknowingly, perhaps knowingly, in disconnecting birth trauma to later findings that fulfill the diagnostic criteria for CSDH that would have evolved from PSDH. In reality, in seemingly all cases, the space is not "benign", but a key finding in asymptomatic CSDH (Note 45), most common, by far, after PSDH and the key to the "acute on chronic" abuse allegations, discussed below.

1.17 Acute on Chronic SDH

The link between PSDH, CSDH, and rebleeds are most clear when "benign" hygromas rebleed and cause symptoms that engender a medical encounter, which is common. At that point, a CT head will be done in almost all cases. With old and new blood seen on CT, almost all radiologists, neurosurgeons, and CAPs are ready to diagnose the condition known as "acute on chronic" SDH. (See Figure 11). The "benign" space now is recharacterized as a chronic SDH. Acute on chronic is an abuse diagnosis that begins abuse prosecutions.

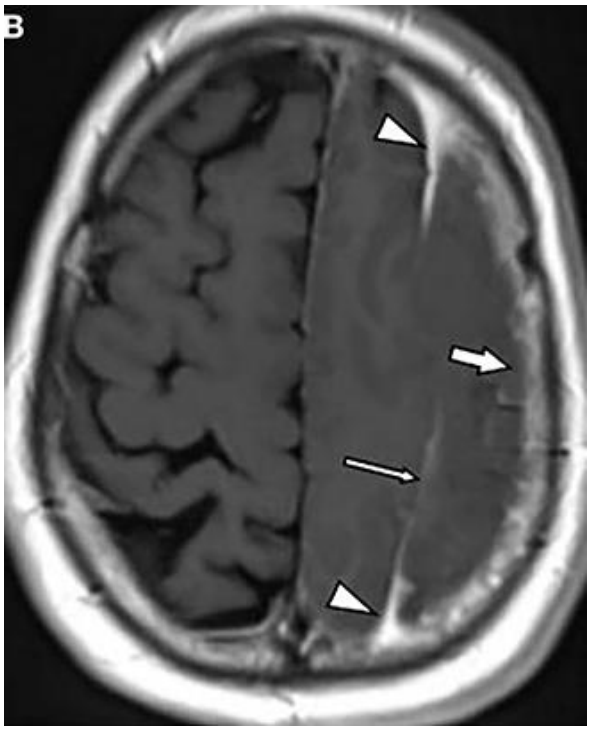


Figure 11. Chronic SDH seen on the left side and white areas on right (and lighter area on the left) are acute blood justifying the "acute on chronic" diagnosis

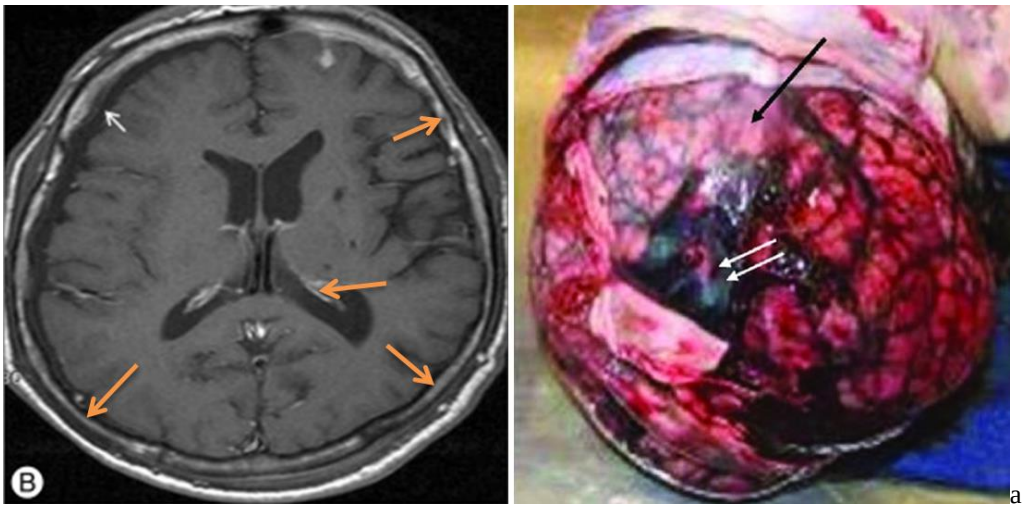
Worse, the new situation/designation soon morphs into a new unsupported narrative that two separate speculative acts of postnatal abusive head trauma (AHT) have occurred, both resulting in acute SDH with blood now in two stages of resolution. This occurs even when there is no credible evidence by history or clinical exams, that either abuse event, or any physical trauma, inflicted postnatally, has occurred. That misdiagnosis too, is yet another consequence of VDD/IR.

It should be noted that the 1000 or so cases of AHT/SBS based on the "acute on chronic" state, prosecuted in the US each year, is a tiny fraction of the total CSDH population. From this tiny percentage, but high absolute number, this misperception of what happens, and the misnomer of "benign", has evolved. In the current environment, in spite of literature, pathology, and logic, that all support the linkage between CSDH and hygroma (Note 46), there has been continued resistance to reclassifying "benign hygroma" as "asymptomatic CSDH", a key factor in the perpetuation of false accusations of abuse in infants.

1.18 Neomembranes

Another impact of "acute on chronic" is that bleeding that results in free, or extravasated blood, that generates an inflammatory scarring reaction is likewise disregarded. The scope of that inflammatory reaction is defined by genetic idiosyncratic patient responses to inflammation, different amounts of scar tissue formation and the frequency of rebleeds. When a threshold level of inflammatory scar tissue deposition from common small rebleeding from the damaged area occurs, pathologic thin

membrane-like structures form. These anatomically visible structures, frequently seen on advanced imaging and at autopsy, are made up of fibrin scar tissue and a matrix of loose fragile capillaries and form, what are called, "neomembranes". These neomembranes are more fragile and more prone to bleeding than the previously damaged PSDH areas and add to potential complications. (See Figure 12).



a b

Figure 12. a: Neomembranes on CT imaging (at orange arrows and all around) with acute blood (white and lighter color) highlighting their structure within the underlying chronic subdural hematoma. b: neomembrane at autopsy as thin film at large black arrow and acute blood for recent rebleed at double white arrows.

When the neomembranes bleed, leaking blood external to the membrane structure, or frequently just internally, the new blood can be seen on advanced imaging. This can occur after a variety of occult, subtle or obvious acute events. The level of force to create a rebleed can be as minimal as normal handling, and often undetectable as a significant event. Yet, when they occur there can be transient symptoms as noted above, (with transient local cerebral edema swelling) and impulse increases in ICP. The underlying condition engenders that type of bleeding that occurs and is vastly more important than the amount of force involved in the rebleeding event. The force needed to create a rebleed of a CSDH or neomembranes, can be nominal.

The lack of identifiable and sufficient force in the history given by parents, trying to help the doctors with understanding what happened to their beloved child, creates, according to CAPs, gaps in the history that are almost always used against apparently decent caregivers. Instead of considering non abuse explanations, CAPs say over and over, that if parents cannot provide an "acceptable" (to the CAPs) significant trauma history to explain the "injuries" (which should be called more accurately "findings"). This, by agreement with themselves is prejudicial against the caregivers. At this point the accusers invoke the default diagnosis of abuse. The impropriety of this, is clear from a medical

perspective, and is particularly egregious thinking, especially when no significant trauma findings have been seen. In a legal framework, no findings and an inference that abuse and only abuse can cause the findings should not be regarded as evidence of abuse. In that scenario, the blood, old and new, and neomembranes, is folded into a fabricated abuse narrative that will be the basis of impending false abuse accusations and prosecution.

1.18.1 Retinal Hemorrhage

As the SBS hypothesis was being crafted, the first add on to the SDH criteria used to diagnose SBS was the presence of retinal hemorrhage (RH). RH logically almost was always present after SDH. RH are caused by increased pressure in the head of any etiology. SDH is a space occupying lesion that causes impulse increases of intracranial pressure. Impulse increases of intracranial blood pressure were known at that time and for the previous 75 years, to cause RH. It was so common and established it was named Terson's Syndrome after the French ophthalmologist who in 1900 established the relationship between increased ICP and RH (Note 47). This construct was unequivocally ignored in 1975 when the SBS hypothesis was fabricated. It had been validated in numerous studies from 1950 to 1975 and on to 2011, discussed in detail below. Given the link to SDH, RH also tracks back, in the majority of cases, explained below, to VDD/IR.

It later became clear that the set up at birthing, in the context of VDD/IR, was a perfect one to create RH in newborns and cause PSDH with its downstream, increased ICP related problems. RH at birth clearly has nothing to do with shaking. Instead, impulse increases in ICP with contractions, and decreased oxygen levels in the blood which also occurs during each labor contractions had a combined impact. Capillary wall structural integrity depends on adequate oxygen (Note 48), especially in high pressure situations and in other situations like PSDH forming, with bleeding, can cause RH too.

When birth related RH was finally looked for in 2003 by Reece (Note 49), a stalwart and vocal American Academy of Pediatrics (AAP) supporter of SBS, it was confirmed that birth resulted in RH in 45% of normal newborns. Later it was found by Rooks that had PSDH occurred in virtually the exact same percentage of cases of RH at birth (46%). Based on logic, Reece's newborns with RH are highly likely to be babies with PSDH. If there is any evidence needed of RH and SDH together in a nonabuse framework (birthing) from the same cause (impulse increases in ICP), this is it. This nexus also invalidates any diagnostic specificity of RH for SBS and its later rhetorical mutations: acceleration/deceleration, rotational acceleration, and AHT.

After birth, it has now become clear that any impulse increase in ICP, after that period, would also cause RH. RH was soon identified as an almost constant concurrent presence with SDH, which on its own, causes and is associated with increased ICP. Furthermore, impulse increases in ICP can occur from a variety of other causes, as Guthkelch and Caffey reported decades before, and are discussed below.

Unfortunately, RH, instead of being recognized as ICP related as it was in 1900, when noted in conjunction with SDH was immediately redefined and mischaracterized as a separate abuse finding.

The group fabricating and crafting SBS had decided to speculatively link RH directly to shaking; the same shaking that they said caused intracranial bleeding used to misdiagnose SBS. The mechanism got a name: "vitreous traction". Ostensibly, RH's value to current and future accusers was to augment abuse allegations by unlinking SDH and RH, into separate abuse findings and then by speculating about multiple acts of abuse.

The reality is that RH is essentially always a consequence of the intracranial forces that created SDH/PSDH. Additionally, any rebleeding adds to baseline increased ICP, via new mass effects, related to the volume of the hemorrhage or edema related to rebleeds, and could cause new RH. Any free blood that occurs for any reason also stimulates additional focal cerebral edema (swelling of the brain) via an inflammatory reaction. The progression of edema causing increasing pressure can lead to additional RH, often delayed by 1-2 days until the inflammation becomes sufficient for intracranial pressure increases to occur.

This delay in development of RH and postponed ocular examinations (which is common), highlights a lesser problem of delayed eye exams. Immediate exams would identify any RH allegedly related to shaking, since the shaking was being promoted as the cause of RH via mechanical rupture. Of course with immediate exams, too soon for swelling and increased pressure, RH would not be found, as animal experiments and selected cases have shown. Delayed eye exams allows time for increasing ICP, related to progressive underlying medical problems (like rebleeds, stroke and brain infections and cerebral edema/swelling), to generate pressure-related RH. The delay, intentional or not, then allows the RH seen, to be mis-ascribed to the fabricated shaking incident and the bogus hypothesis of RH formation via vitreous traction (VT), discussed below.

Over time, how RH formed was later understood via electron microscopy (Note 50), ultrasound (Note 51), and advances in neuropathology. Terson had shown in 1900 that impulse increases of ICP associated with subarachnoid bleeding caused RH. How it occurred was later determined. The ICP in the head is transmitted down the optic nerve sheath (ONS), to the eye, hydraulically, using the CSF as the hydraulic fluid. The ONS is a true extension of the dura and part of the brain sheath. The CSF in the sheath is in the same space and under the same pressure as the CSF around the brain in the skull. As hydraulics would mandate, increased pressure in the head is increased pressure in the optic nerve sheath. This increased ONS pressure when it rises quickly, well above intravenous capillary pressures of 20 mmHg, it will compress the venous capillaries in the eye easily. When this compression occurs it obstructs the outflow of blood from the retina capillaries, and in the central retinal vein located in the center of the ONS. When this occurs, decreased blood flow occurs by blocking egress and then ingress of blood to and from the eye in the vascular structures. See Figure 13.

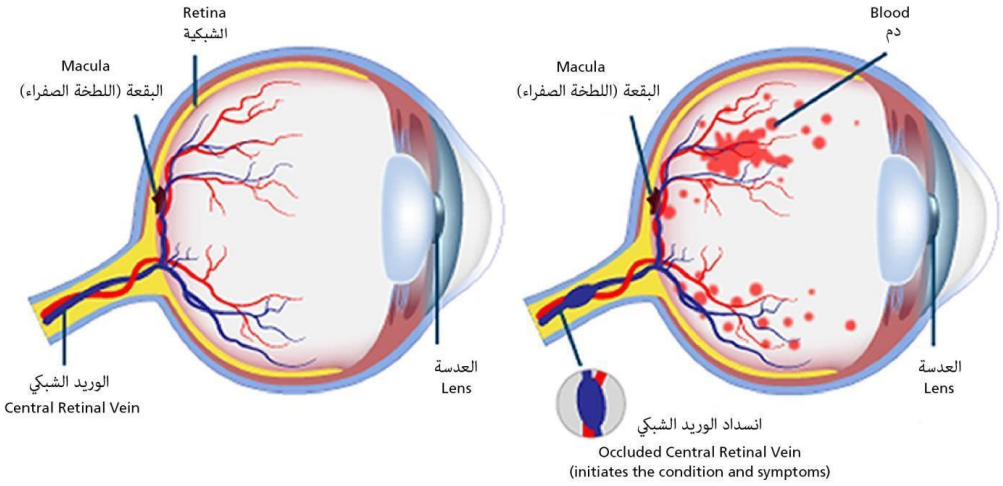
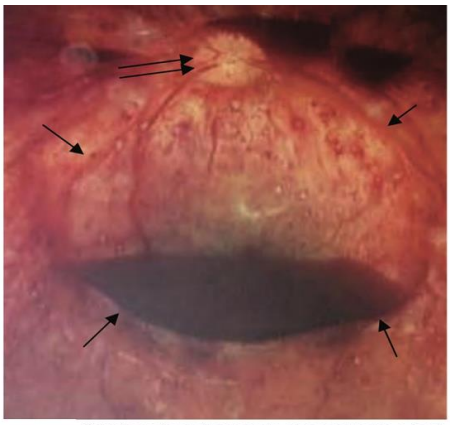


Figure 13. Graphic representation of central retinal vein compression and retinal hemorrhage

With decreased flow, decreased oxygen to the retina occurs too. There, hypoxia directly impacts capillary wall (Note 52) cells and the structural integrity of the capillary wall. Hypoxia in the cell wall causes breakdown of the bonds between the single layer capillary wall cells. With separation of the cell wall cells, gaps occur about the size of red cells, and leakage of small, micro amounts of blood from the compromised capillaries occurs that form the RH that are seen. We now know that if enough RH occurs, the small amounts of free blood will coalesce under the internal limiting membrane of the retina (the most frontal structure of the retina) to form collections of free blood called schisis cavities.

The fluid filled, blister-like structure has raised margins that have been misnamed retinal folds when they are not folds at all, but a swollen edge of the schisis cavity. (See Figure 14 below)



Schisis cavity on the macula, also referred to a boat shaped retinal hemorrhage or macular schisis, of a child lying right side down with free blood in the cavity creating a red blood cell/serum interface. The edges of the schisis cavities (single arrows) form ridges that are called retinal folds. Optic disc at doub arrows.

Figure 14. Schisis Cavity

The latter findings, schisis and folds, are at the extreme end of the entire spectrum of heme-related ocular findings. Schisis cavities soon were speculatively advanced as diagnostic of “severe” abuse,

shaking, and vitreous traction, when they are not. From the start, the eye findings are promoted as another additional, "separate" abuse specific finding. In reality, RH are seen in a variety of nonabuse situations as well, all with the common denominator of increased ICP (Note 53, Note 54). 17% of all patients at autopsy have RH. RH is an unreliable indicator of abuse with no evidentiary value to use in court as proof of abuse.

With progression of ICP and further hypoxic injury, schisis cavity can break down and then blood leaks internally in the eye, into the vitreous (jelly of the eye) and the worst of the ocular findings also improperly alleged to be indicative of severe abuse, occurs: vitreous hemorrhage. Vision is lost and the retina can longer be seen through the bloody vitreous.

1.19 Vitreous Traction

In the early years of modern child abuse pediatrics (after 1975), the common coexistence of SDH and RH, in the same patient outside the birth period needed an explanation to deal with RH. The well accepted Terson's syndrome, identified 75 years before, was eschewed in favor of shaking, for no valid or reasonable reason, even when it was likely. The concocted hypothesis that shaking caused RH by mechanical damage of the vitreous jelly was invoked to link with the shaking alleged to cause SDH and embellish the false SBS hypothesis. This became the "vitreous traction" (VT) hypothesis, and is still being promoted, since it supposedly relies on shaking, and is used to allegedly strengthen the prosecution's primary deceptions regarding shaking forces causing SBS. VT was hypothesized to cause RH by pulling and tearing capillaries in the retina apart during human shaking, by movement of the vitreous jelly tugging on its attachments to the retina. The hypothesis, in later animal experiments (Note 55), and in video of infants being shaken hard, with no resulting RH or SDH (Note 56), confirmed that this notion of VT was contrived and false.

Since RH was now part of SBS, it soon began to be used on its own at times, or even when only present in one eye (Note 57), as a sign of abuse, contorting the CAPs own already concocted SBS hypothesis to fit any abuse narrative the accusers desired. The practice became common.

RH, not unexpectedly, was so predictable that ophthalmologic exams became routine in suspected child abuse cases. Concurrently, pressure was exerted by CAPs working with prominent pro-SBS ophthalmologists, especially pediatric ophthalmologists, like Alex Levin (Note 58), to routinely promote the role of ophthalmologists in SBS. AAP/COCAN encouraged consultants to include in their ophthalmology consult that RH was "highly associated with", "consistent with" or "diagnostic of" shaking and abuse. These efforts lent undeserved credibility to RH especially in court, where false certainty statements as testimony, declaring RH diagnostic of SBS/AHT/NAT, were accepted as valid evidence, when it is not. It was another form of circular logic. So many misdiagnosed cases of child abuse had RH that RH was mis-elevated to a diagnostic sign of abuse and included in the now disavowed diagnostic SBS triad.

The identification of RH became routine and the false linkage to shaking surprisingly, was effectuated with the formal cooperation of the American Academy of Ophthalmology (AAO). The prominent

pro-SBS ophthalmologists who had joined this movement and were influential in AAO, were able to generate untrue official policy statements (Note 59), elevating RH to be one of three criteria to diagnose SBS and child abuse. This linkage, of course, is false, but was a devastating turn that further complicated defending false accusations.

The triad linkage emerged as the core teaching of CAP for decades, which was used to promulgate the hysteria around SBS. Ironically, the triad was actually a monad, SDH alone and its secondary impacts (both RH and encephalopathy, the third leg of the triad) were mere consequences of SDH, and none of the findings were ever uniquely diagnostic of abuse.

The lessons of Terson's syndrome first identified in 1900 and verified in subsequent studies, proving that impulse increases of ICP, of any etiology, caused RH, were ignored. Ultimately shaking was declared the etiology of RH to sustain as valid the illusion of SBS and its false linkage to RH with remarkable success even after the true mechanism was clearly exposed after 1985.

1.20 Previous Hypotheses Re: Early PSDH

It is worth noting how long the spectrum of actual causes of PSDH and its complications have been known. Prior studies that cataloged the spectrum of possible causes for retino-subdural hemorrhage of infancy and defined a differential diagnosis for these findings have been disregarded since 1975 when the SBS hypothesis was crafted.

The most important of these early studies, was written by Dr. Norman Guthkelch, a British neurosurgeon, later ironically referred to as a founding father of SBS, a designation he later renounced in 2012 (Note 60). In 1953, years before modern CAP emerged, he studied cases that produced retino-subdural hemorrhage of infancy (SDH and RH). The latter term is an objective term that Guthkelch coined to describe the findings as they should be, without any reference to mechanism or intent. He included events related to VDD/IR, which was still quite pervasive in the early 50's.

In that 1953 article, nonabuse causes for the findings related to vitamin D deficiency, birth trauma and complications were elucidated (Note 61). In addition to craniotabes related findings, other nonabuse VDD/IR medical findings were noted. Also accidents, brain infections, birth related problems, among other known etiologies were also noted. The latency period, cerebrovenous thrombosis (CVT), prematurity, twin births, chronic SDH, rebleeds and neomembranes were all identified as important and relevant to making the correct diagnosis and part of CSDH. Guthkekch's 1953 article defined a differential diagnosis that is extremely relevant and still applicable to almost every current case. Furthermore, it highlights the most common nonabuse explanations offered by defense experts today, that are fiercely attacked by disregarding evidence-based science and clinical findings.

In the 2012 article, Guthkelch wrote this about his earlier work.

[Square bracket text is explanatory by author]

"In my 1953 article, I [Guthkelch] reviewed 24 cases of infantile subdural effusions [fluid collections around the brain-hygroma], primarily subdural hematoma. In the article, I emphasized that infantile subdural effusions are not rare and that "the frequency with which [they] are found is proportional to

the intensity with which they are sought." Most of these cases occurred in the first few months of life (11 before age 3 months, 5 between 3 and 6 months, and 2 thereafter). "Seventy-five percent were associated with abnormal or difficult labor, and two cases were associated with a head injury two weeks prior. [A latency period]. 12 Subdural effusions [hygromas] were also found in association with meningitis (5 cases) [brain infection] and venous sinus thrombosis [CVT], which may complicate any infective focus (1 case) [encephalitis, brain infection]. Of those with known histories, there were two sets of twins and two premature babies. In 75% of the cases, the hematoma was surrounded by a membrane that was adherent to the dura [neomembranes], and in almost all instances the fluid obtained from subdural tap was xanthochromic [clear yellow, colored by remnants of old metabolized blood], with a variable quantity of fresh blood [mixed density and acute on chronic], confirming that this process had been ongoing for some time, in many cases possibly from birth.

In spite of his celebrity, and being regarded as a founder father of SBS, this important study (in 1953) was ignored for no valid reason in 1975 when the small group of self-proclaimed "child abuse" pediatricians got organized and fabricated the first iterations of SBS. When this reprise of his findings emerged in the 2012 period, sadly, Dr. Guthkelch was then no longer celebrated but instead, vilified by the child abuse establishment for his efforts to clarify his position and contribute to the elimination of false accusations, which he had become acutely aware of leading up to 2012.

Any of the entities described by Guthkelch in 1951, can generate symptoms and/or findings sufficient to lead to false accusations of abuse. This seminal 2012 article retrospectively forms a documented 60 years history of knowledge of the nonabuse etiologies of findings used to misdiagnose child abuse. The etiologies and findings he documented in 1953 remain intentionally disregarded today in spite of an AAP published obligation to rule out each nonabuse etiology, in every case of suspected abuse using definitive testing before alleging abuse. This obligation is ignored in deed. Only after that obligation is met can a diagnosis of abuse be made; something that is not done except with nonevidenced based belief statements of false certainty. Beliefs, lack of objective testing, and false certainty statement are what dominate CAP thinking, paving the path to false accusations.

1.21 The Addition of Detail to False Allegations of SBS/AHT

To increase impact, details related to the primary false hypothesis were concocted as well. After the additional hypotheses were crafted, nonmedical legal authorities were told that shaking tore the "bridging veins" (BVs) in the skull. BVs in the head are large stretchable, high volume veins, approximately 20 per infant that drained large volumes of blood (10-15 ml/min each) from the brain tissue to the primary drainage channel for blood in the brain, the superior sagittal sinus (SSS). The torn BVs (see Figure 15) were promoted as the source of SDH, although too small volumes SDH and the lack of pathological and imaging corroboration of torn BV's were overlooked. Below the stretchability of BVs to tolerate the limited movement that occurs with shaking, is clear.

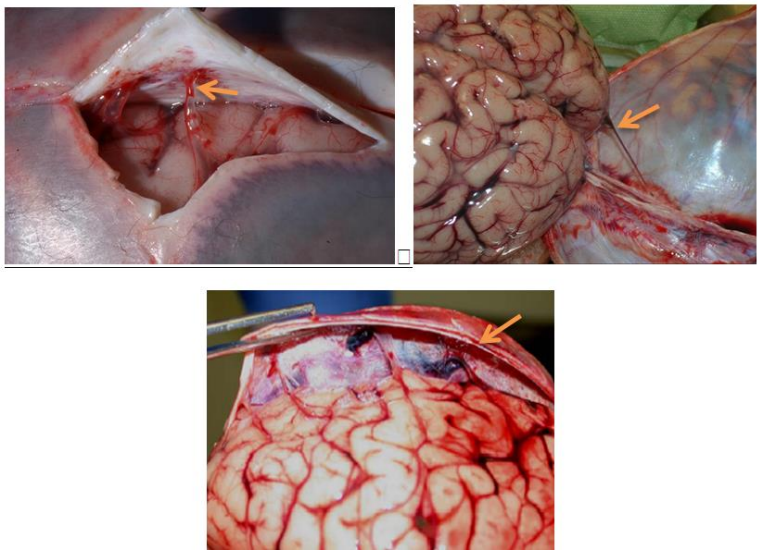


Figure 15. Stretched BVs (arrows) showing their structural integrity, decreasing the probability of tearing.

In the eye, the RH were explained with VT (vitreous traction) which was consistent with the 'shaken' baby syndrome; and the VT hypothesis that was concurrently being promoted intensely in that early time. The patina of science, really a pseudoscience, could not be, and was not, questioned by lay personnel, lacking independent medical knowledge, which only defense experts could later bring forth. The add on details, the authorities were taught, were never proven to be valid ideas at that time, and have since been disproven via imaging and autopsy, video of abusive shaking and animal studies. Video of repeated acts of abusive shaking that did not cause any findings (Note 62), are quite compelling and readily accessible on YouTube, and numerous animal studies, including studies unable to produce RH at the 600g level of force, 100 times the force an average adult can generate in shaking force experiments (5g) (Note 63). In another study, small animals shaken to death, with broken necks as the cause of death, at autopsy, had no RH. Lastly, as explained above, the nexus of birth related SDH and birth related RH, seen in newborns, showed that any notion that these two findings were uniquely diagnostic of abusive shaking (vs increased ICP), were unsupported and false.

2. Part II- VDD Based Infantile Rickets Being Misdiagnosed As Physical Abuse

2.1 Introduction to the Use of IR/VDD as Alleged Physical Abuse

The evolution of the second prong of false accusations, physical abuse-PA, was related specifically to VDD/IR too. Added to findings of SBS/SDH with intracranial blood and increased ICP, in the late 80's, then consistently convicting innocent caregivers with machine-like precision, as the epidemic of rickets matured, this prong was well honed to work in tandem and its devastating impacts, had plateaued by the late 80's. The process had evolved to such a degree; the characterizations of a "prosecution machine" were not farfetched (Note 64) as conviction rates approached 95%. However, active

resistance to the dogma and unfounded basis of SBS/AHT was now present by the mid 80's as defenses experts emerged. This second prong was energized however and resulted in new opportunities to promote VDD/IR dogma (Note 65) that led to further growth, apparent solidification and pseudo-validation of the child abuse establishment's reliability and the child abuse pediatricians' credibility. These were well cultivated efforts and were welcomed by law enforcement with open arms and enthusiasm as it led to more charges and convictions; arguably the primary goal of prosecutors and to some extent CAPs.

2.2 The Evolution of PA to Buttress SBS

It was during the first 10 year period (1975-1985), following the cues from the battered child era that an increased numbers of skeletal surveys were being done, on an increased number of suspected SBS child abuse cases. In the following 10 years, the frequency, severity and obviousness of the VDD/IR findings were increasing as well.

After 1985, rickets findings caused by VDD, and complications related to IR in the skull (craniotabes), ribs, and long bones were starting to be misidentified regularly in skeletal surveys of infants who were alleged to have SBS. Child abuse pediatricians and their radiology confederates began to take notice and call out possible abuse as the cause in reports, even when the findings were known to have no probative value. The failure to recognize these findings as the manifestations of a resurgence of VDD/IR, created the opportunity for COCAN (with cooperation from pediatric radiologists and other confederates), to introduce a physical abuse angle that would augment the problems that had already arose with the defense and the prosecution of false accusations of SBS child abuse. This opportunity was seized. This created a period of ascension for the shaken baby syndrome and child abuse misdiagnosis and convictions. Ironically, that momentum soon crossed paths with the first wave of credentialed defense experts who pointed out 1) the emergence of IR and 2) the newly crafted notion of using rickets related findings as supposed proof of bony physical abuse (PA). PA and SBS worked well together for accusers and the age of rickets being diagnosed as PA, began in earnest.

In the mid 80's Paul Kleinman, prominent pediatric radiologist from Seattle Children's Hospital, and an SBS proponent, was reviewing skeletal surveys and failed to recognize, or chose to not recognize, what before, were common IR findings. Instead, he began to promote the incorrect notion that the rickets findings he did not identify, were, in fact, a new contrived category of abuse related fractures divorced from well-established thinking and clinical injury patterns. He advanced the improbable (and essentially false) idea that previously ~~that~~ was essentially unheard of. Kleinman began to promote the notion of painless, asymptomatic "fractures" in apparently genetically normal children. He made these assertions ipse dixit (Note 66) style, without pathologic or biomechanical corroboration, and was essentially novel and improbable thinking. Ignoring all common knowledge about fractures, he said, the IR findings were specific for abuse. Odd mechanisms of injury (twisting, pulling, squeezing, and intentional deformation) were promoted as common forms of abusive behavior without any actual evidence they were occurring or any clear evidence they had occurred in apparently decent families.

These improbable and foreign notions were strangely accepted without question. Furthermore, there was no meaningful response from involved orthopedic and emergency physicians who knew that was fabricated thinking. These physicians were constantly seeing traumatic fractures and suspected fractures in infants and children, with all the conventional collateral findings, and the notion of painless fractures was essentially fantasy. Kleinman promoted painless fractures in virtually all rickets babies. This was all done with no witness evidence of such actions, credible denials and plausible explanations by apparently loving parents. There was also profound absence of collateral soft tissue findings and pain related symptoms; essentially universal findings with fractures. Kleinman, because of who he was, succeeded beyond his wildest dreams of instantiating the dogma of painless asymptomatic fractures into modern child abuse pediatrics prosecutions. At the same time authorities and medical providers in the hospitals grew increasingly disinclined to disagree with anything emerging from COCAN for a wide variety of specious reasons; including professional and social threats. Beyond the obvious lack of fracture symptoms and findings, fracture staging/dating and healing sequences, well established for both VDD (Note 67) and non VDD children for decades (and centuries) were cast aside, to allow for fabricated timelines designed to implicate targeted abuser suspects, all at the expense of known science. This second front of physical abuse was immediately widely accepted and allegations of physical abuse due to the commonality of IR, in addition to blood in the head, were the new norm. The ability to convict, based, in reality, on two prongs of false accusations of abuse AHT and PA, both specifically related to VDD, was perfected in the legal system by the CAPs. For innocent defendants this created more, near insurmountable, problems discussed above and below.

2.3 The First Recognitions of Metabolic Bone Disease (MBD) in the Current Timeframe

Almost beyond belief, and testament to the power the child abuse establishment was concentrating in their own hands, no formal opposition in the medical literature to 'rickets as abuse' occurred for 20 years. A second "epidemic": an epidemic of false accusations of physical abuse had begun. Sadly, the damage done in those 2 decades is incalculable.

The first person to speak up was Colin Paterson, M.D., in 2003 in England, where he, like everyone else, had no awareness of the concurrent evolving VDD/IR medical epidemic. At that time, he was seeing families being prosecuted for abuse, primarily SBS, for unrecognized IR findings being misdiagnosed as abusive fractures. He coined the term "temporary brittle bone disease" (TBBD) to describe this apparently transient condition. This construct was almost entirely the VDD/IR issue but was 3 years ahead of awareness of the VDD/IR. Fortunately in 2006, Bodnar's seminal screening study in Pittsburgh identified the high percentage of VDD mothers and IR impacted babies in the general population (80% and 90% respectively), and subsequently VDD/IR was recognized worldwide.

With years (1985-2003) of defense experts exposing the flaws in other aspects of child abuse pediatrics, the controversies around many issues in CAP were now fully raging. Dr. Paterson's prescient notion, instead of stimulating new thinking, was overwhelmingly crushed by the British child abuse establishment, long committed to American child abuse pediatric dogma. Efforts to destroy Dr.

Patterson (Note 68) were exemplary of cruel and unethical ad hominem attacks that would be leveled against countless challengers to the existing child abuse myths since. The attacks, that had been ongoing for near two decades and included Dr. Guthkelch, myself, and many other colleagues.

British CAPs mounted a brutal systemic effort, modeled after American pediatric tactics in use since 1985. The American pediatricians, and to a lesser extent their foreign counterparts, had schemes, strategies, court transcripts, attack strategies, and widely circulated tactical plans distributed through secret databases among prosecutors, to unprofessionally try to quash any and all medical challenges and shut down the careers of effective defense experts.

The challengers were exposing the false constructs the CAPs had used, and were using, to accuse and convict innocent caregivers. The decision to defend the status quo versus re-examine their beliefs, had to be made. Reexamination would involve CAPs and legal professionals too, admitting perpetrating child and family abuse by promoting false accusations, was a bitter pill left unswallowed.

In England, the suppression efforts went farther. The medical board of England (the English nation-wide General Medical Council) took Colin Peterson's license to practice medicine for suggesting TBBD. They later (March 2016) did the same to one the world's premier pediatric neuropathologists, Dr. Waney Squier for consistently successfully challenging other existing dogma about SBS (Note 69).

With Dr. Paterson, the loss of his medical license was his punishment for defying establishment dogma by suggesting that there was an unidentified disease causing broken ("brittle") bones that were not related to abuse. Clearly, allowing any nonabuse explanation to gain traction would expose thousands of misdiagnoses and assign responsibility for countless iatrogenically disrupted, harmed and/or destroyed families; something that had to be avoided at any cost, no matter how craven.

2.4 The Discovery of the Epidemic of VDD in 2006

The epidemic of vitamin D deficiency (VDD) was discovered in 2006 when the first significant study to identify the problem was published by Bodnar (Note 70) in Pittsburgh. Within a short time the epidemic of VDD was unequivocally validated in numerous studies (Note 71).

Even after these studies showing worldwide, widespread VDD, the acknowledgment of the epidemic and initiating the simple interventions to correct it, which were known, were not implemented by AAP or the American Academy of Obstetrics and Gynecology (AAOG), seemingly knowingly.

As the issue was brought to the fore by defense experts, any pretext of not being unaware, evaporated. VDD/IR was treated as irrelevant. Soon, the very existence of mild to moderate rickets with bone abnormalities was overtly denied. When VD levels were done, which was rare, pathologic VDD levels began being ignored, misinterpreted, and/or dismissed. In the last decade, new low lab "normals" were introduced in children's hospitals that conflict with Center for Disease Control (CDC) and Mayo normals. The "new" normals (30% lower) impacted the ability to use VD levels. the most reliable indicator of IR caused by VDD, to support the diagnosis of nonabuse.

Acting in concert with the thinking about TBBD and new, important information about VDD that would prevent the vast majority of false accusations based on VDD/IR, it was decided by AAP to disregard and suppress the emerging revelations occurring in radiology, pathology, and endocrinology related to VDD/IR. They did so in the face of dozens of studies proving the VDD/IR connection being constantly presented to them by the challengers, in case after case. They did so under the guidance COCAN, and with the consent of virtually all CAPs, the vast majority of practicing pediatricians, and the AAOG.

The latter specialty (OB/GYN) could easily have acted on its own by recommending VD testing in newly pregnant women and fixing any deficiencies during pregnancy. Once informed that the VDD epidemic was present, either specialty could have tested for and treated VDD, but neither did. AAP made a gesture to increase its daily recommended dose of VD for infants to deflect criticism, from 200 units to 400 units when corrective doses in use in Europe, were in the 2000 to 5000 units/day range (Note 72, 73). No meaningful efforts to prevent VDD in pregnant mothers or VDD/IR in newborns were ever made.

As the nature and of the epidemic began to be appreciated, a small number of family pediatricians (Note 74) responded by empirically (without testing) prescribing VD to newborns. Child bearing aged mothers were ignored. The consequence of that disregard is that VDD/IR is still causing fetal tickets, birth related dural damage (PSDH), and bony rickets findings in newborns. Compounding these misdiagnoses, the findings were then used to amplify the majority of false allegations of child abuse.

2.5 The Use of VDD/IR as Physical Abuse (PA) in Court

With apparently two sets of abuse findings (1) SBS/AHT and (2) PA, now being used to accuse, the intensity of the prosecutorial zeal applied against the target caregiver increased. Furthermore, any plausible explanations for the AHT component of the allegation (blood in the head) was more likely, under any circumstances, to be dismissed in favor of an overall impression of abuse. With a “new” and “separate” set of findings of physical abuse (PA) augmenting the false AHT related abuse allegations, the ability to preserve the status quo and suppress the fraudulent nature of what was occurring (dismissing nonabuse etiologies), was significantly augmented. Getting convictions was proportionally increased.

2.6 Other IR Findings in the Skeleton

Other dramatic findings of IR, that fell into use with respect to alleging PA in the appendicular and axial skeleton linked to IR/PA, beyond craniotabes tied to AHT, included pathologic fractures, rib cracks with healing callus and a variety of developmental bone abnormalities seen on imaging. All these findings were well studied (1900-1950), and since, are known IR findings and have been used many times over to accuse caregivers of physical abuse. The addition of allegations of physical abuse and bone fractures, often multiple, to allegations of SBS/AHT, complicates defending false accusations of abuse. Cases with 20 alleged fractures in asymptomatic pain free infants were common in spite of being implausible and ridiculous to the knowing. Knowing that real fractures are quite painful and

being tricked into the notion that previously normal infants don't feel fractures, creates a nonsensical delusion. When considering the frequency of "normal" medical encounters and family contacts in the first few months of life for infants, during which broken bone abuse was often alleged to have occurred, the lack of any pain related symptoms with handling the child, renders PA as the cause of rickets findings as implausible at best, and essentially impossible at the other end. Yet, this false construct of the painless fractures, has been used countless times to testify that the IR bone findings on imaging, are abusive fractures, and it continues to work. The legal authorities bought it then, have relied on it frequently, as needed, and seemingly defend it now.

2.7 Add-on Findings Used to Misdiagnose Abuse

Other add-on findings like minor or moderate skin markings which are routinely mis-elevated to signs of intentional physical abuse (PA) and mischaracterized as abusive bruises are used to augment false accusations of abuse. This is done even when the minor bruises are remote by location to the more serious findings, or within the range of normal play-related rough housing, minor accidents, or common household falls, most often reported by parents. These reports are then reflexively dismissed as inconsequential or parents explanations dismissed as lies. The bruises then are mischaracterized as abuse.

2.8 Sentinel Injuries

These markings, once mischaracterized, are declared part of an unsupported minor abuse prong. In fact, the strategy has recently been codified as the fabricated concept of "sentinel injuries". The concept of a sentinel injury (minor markings), no matter how minor or improbable for abuse, is now an AAP supported, speculative, supposed precursor to abuse, in use in courts today as evidence. Supposed sentinel injuries are used to support abuse occurring over a longer arc of time and on multiple occasions. All children have markings and small bruises; some many. Yet, the concept will be invoked, even when findings are explained, consistent with history, and abuse is implausible based on a social history of verified past positive parental behaviors.

Isolated small bruises have no probative value in abuse work-ups but are commonly included to support and falsely buttress other specious non abuse findings that together generate the devastating constellation of findings to allege false accusations; that are far too common.

2.9 Diagnostic Findings in IR

2.9.1 Fuzzy Growth Plate Areas

On imaging, the inflamed and frayed appearances of growth plates of rickets have been mischaracterized as abusive Salter style fractures. (Note 75) (See Figure 16)

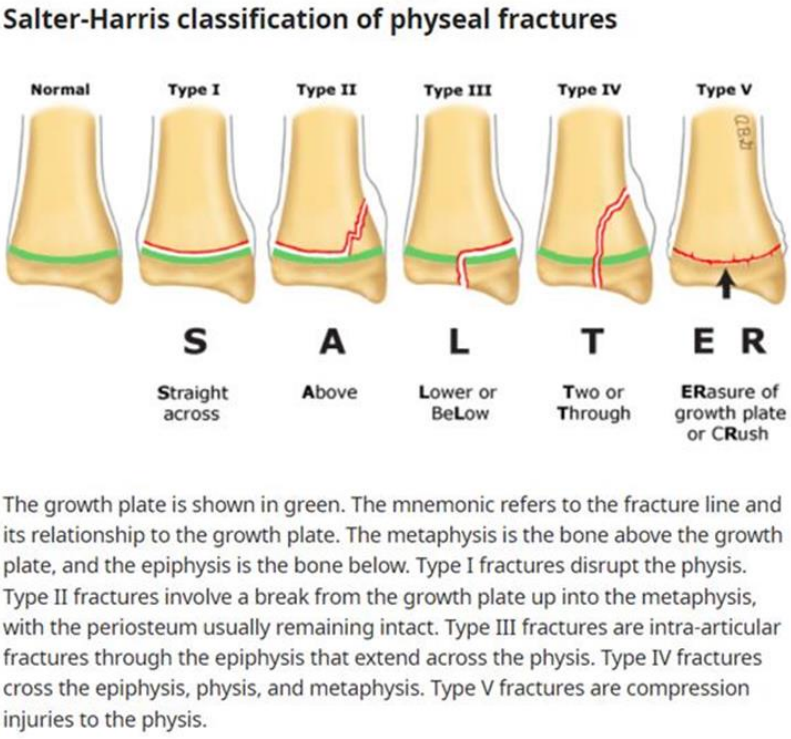
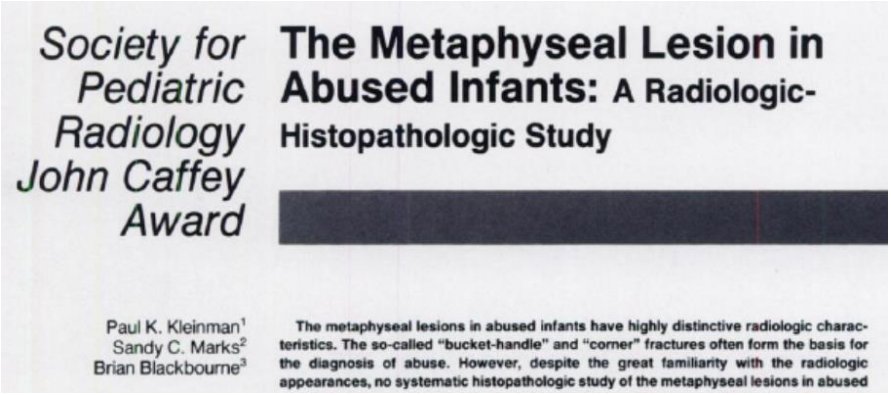


Figure 16.

The findings, whatever they are, are then arbitrarily called acute, subacute, or chronic abusive fractures. Even at times, 'late stage' healing fractures, will be used to comport with timelines fabricated in abuse narratives. This is done in complete disregard for the entire medical fund of knowledge regarding the timeline of fracture healing. This imprecise dating is necessary, and widely used, to set up specific target caregivers. To do this, there is a clear divorce from the expectations of conventional fracture findings, healing, symptoms, and progressions.

2.10 CML

One of these specific rickets findings. (beaking and spurring at the growth plate areas), known to be part of rickets via autopsy and imaging, was audaciously linked to abuse, and misnamed by Kleinman himself (Note 76), the "classic metaphyseal lesion" (CML). (See Figure 17)



Society for Pediatric Radiology John Caffey Award

The Metaphyseal Lesion in Abused Infants: A Radiologic-Histopathologic Study

Paul K. Kleinman[1]
Sandy C. Marks[2]
Brian Blackbourne[3]

The metaphyseal lesions in abused infants have highly distinctive radiologic characteristics. The so-called "bucket-handle" and "corner" fractures often form the basis for the diagnosis of abuse. However, despite the great familiarity with the radiologic appearances, no systematic histopathologic study of the metaphyseal lesions in abused

Figure 17. Title page of Kleinman article

This finding, which is not "classical", has been known for nearly a century to be transient growth abnormality related to irregular and pathologic pattern of deposition of calcium into excess new cartilage at the diseased growth plates of long bones, was and yet, still is, promoted successfully and fraudulently as virtually diagnostic of PA. Instead, it is a diagnostic sign of IR.

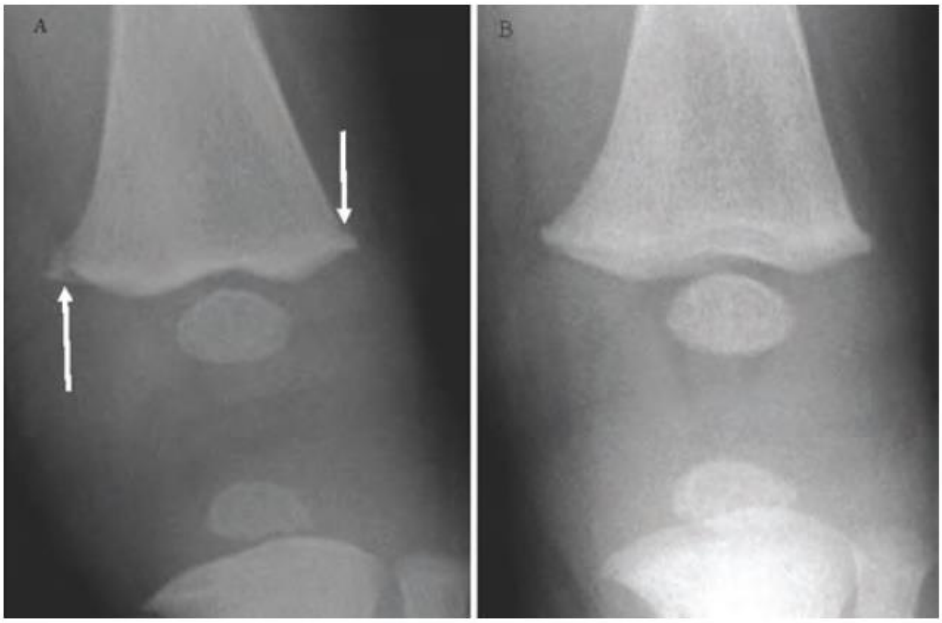


Fig e "corner fractures" or classic metaphyseal lesion of L femur seen in vitamin D deficient babies that represents dysplastic growth. In this case the leg was nontender with normal painless range of motion. B. Sixteen days later the findings are resolved without callus formation after vitamin D supplementation.

Figure 18

2.11 Infant Ribs

Once a child abuse investigation starts and a skeletal series is done, seemingly any abnormal bone contour, especially on the ribs, is called a "healed fracture". (See Figure 19)

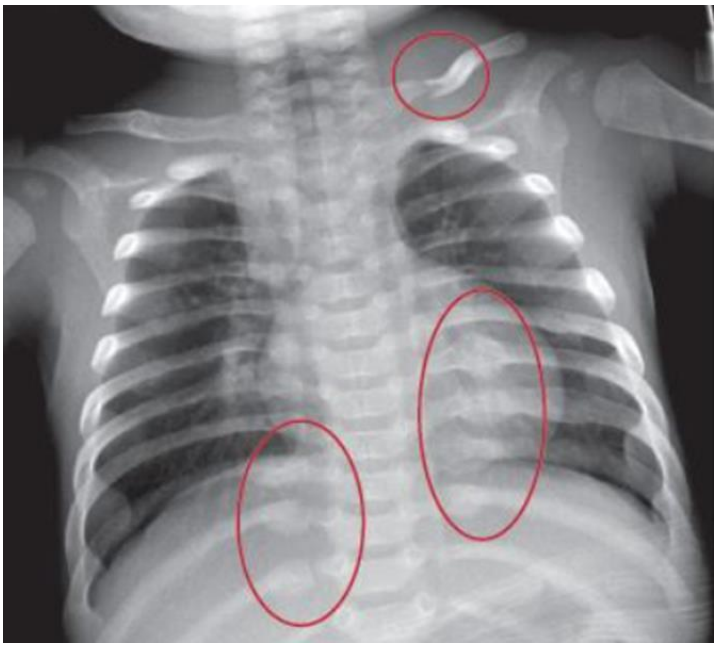


Figure 19. The bulbous calcified shapes in the circles are the healing callus seen in healing and healed rib fractures of any etiology; in this case birth related

Without an acceptable accidental trauma explanation, that is acceptable specifically to the CAP, which almost never occurs, the inference and accusations that they were abusive, follows. With MBD/VDD/IR at birth and no postnatal clinical findings to suggest postnatal traumatic bone fracture, "acceptable" explanations essentially don't seem to exist. The accuser in that scenario ignores common birth related trauma, minor accidents, and falls, pathologic fractures in weakened boned caused by IR and MBD, and inappropriately defaults to abuse as the cause.

Some, if not essentially all, of these healing callus based deformations are occult healing rib cracks. They occur during labor, as discussed above. With VDD/IR so common, birth rib cage deformation and rib straightening that occurs during repeated contractions, in the context of osteoporotic rachitic fetal ribs, result in cracks. Because of the stretch exerted on the inside surface with straightening due to deformation, the cracks occur on the interior surface of the ribs. These cracks are not visible on plain chest X-rays until the healing reaction is visible. As healing progresses callus can be seen after 2 weeks, up to a year of life, and occasionally for years longer. As the broad contour of callus on plain x-ray forms it is assumed to be, not a birth related crack, but an abusive healing conventional postnatal through and through traumatic rib fracture. With no history or clinical sign of postnatal chest pain that

would occur with normal handling, it is virtually certain to be a false assumption that it is the result of postnatal trauma.

The 3D chest CT below illustrates the location of the end result. There are two healed cracks with minimal remodeling (arrows), visible on the inner surface in the right image. This is paired with a normal posterior cortex (rib surface) of the previously cracked ribs seen in the left image. These are specific findings for a healed deformation related crack. It is almost never looked for even though a mid-level percentage of suspected abuse cases have this finding.

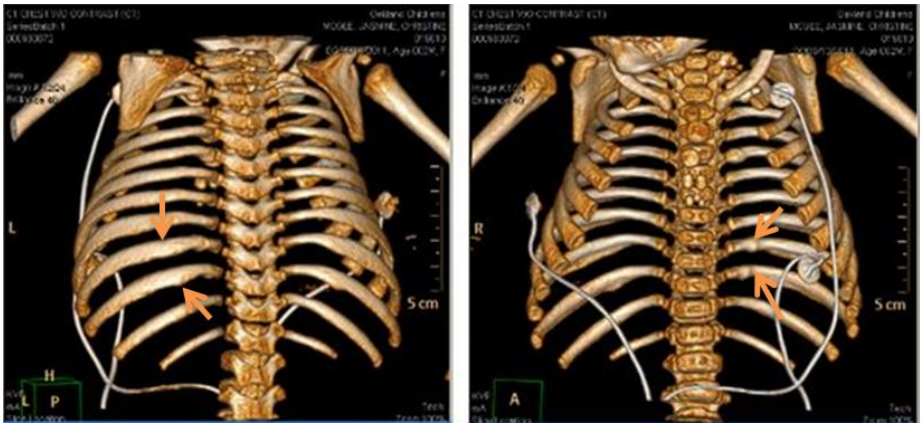


Figure 20. In the right image the healing bulbous callus is seen on the inside surface of the rib cortex (at arrows) where distraction cracks occur. In the left image the posterior ribs appear normal in the areas highlighted by arrows where compaction of bone occurs and no healing reaction is engendered

When callus is seen weeks or more after birth, the above mechanism is ignored and postnatal abusive rib breaking is alleged. This is done unthinkingly, even without any history of chest wall tenderness that occurs with postnatal traumatic fractures. When babies held by the chest daily with traumatic fractures there is pain, and pain with breathing as well. Any positive social history further supports that such a brutal and painful form of abuse did not occur.

2.12 Neoperiostosis

With VDD, delayed calcification of cartilage creates excess cartilage accumulating along diaphysis (shafts) of long bones. After birth, and out of the womb, in almost all cases VDD/IR, at least modestly, due to modest intake of dietary VD and sun exposure begins to improve. As VD and calcium become available, this excess cartilage starts receiving calcium and forms visible calcified layers adjacent and parallel to the existing calcified bone. (See Figure 21). Later with growth, the layers meld together. When this finding is seen without other characteristic findings seen in painful, slow healing, high force, and rare longitudinal fractures, abuse is highly unlikely if not implausible. These layers of new bone in most cases will then be misdiagnosed as healing abusive longitudinal fractures.

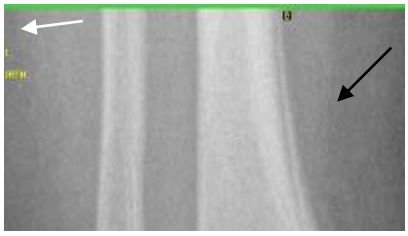


Figure 21. Periosteal neostosis in VDD child at black arrow

Longitudinal fractures in real life are painful and obvious. With neoperiostosis, infants are pain free and moving freely as they would with no trauma and no abuse. The newly calcified cartilage just inside the fibrous periosteum is painless. This finding is normally found with infantile rickets during resolution. Regardless, the finding has been assigned as abuse to be misused to support previously misdiagnosed abuse cases. The unscientific and fabricated link of neoperiostosis to abuse misdiagnosis are testified to with false certainty, as healing longitudinal or spiral fractures with zero clinical correlation. Yet another form of nefarious speculation misused causing damage, so common in child abuse pediatrics (CAP) related to VDD/IR.

2.13 Pathologic Fractures

Another rickets complication, in the context of extreme osteoporosis and structural abnormalities in bones, is actual pathologic fractures. (See Figure 22). These can occur with normal handling, and, are likewise diagnosed as intentional fractures. This particularly speculative brutal act will be charged as abuse regardless of appearance or age. Credible benign explanations, other signs of VDD, plausible explanations, and positive social history are ignored here, as well.

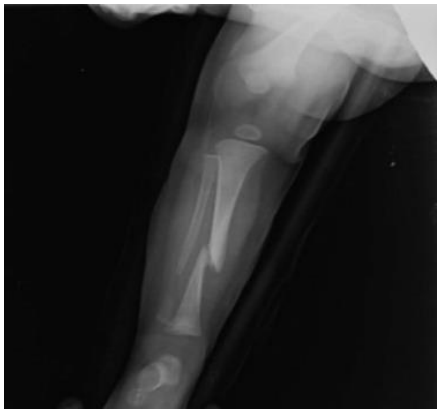
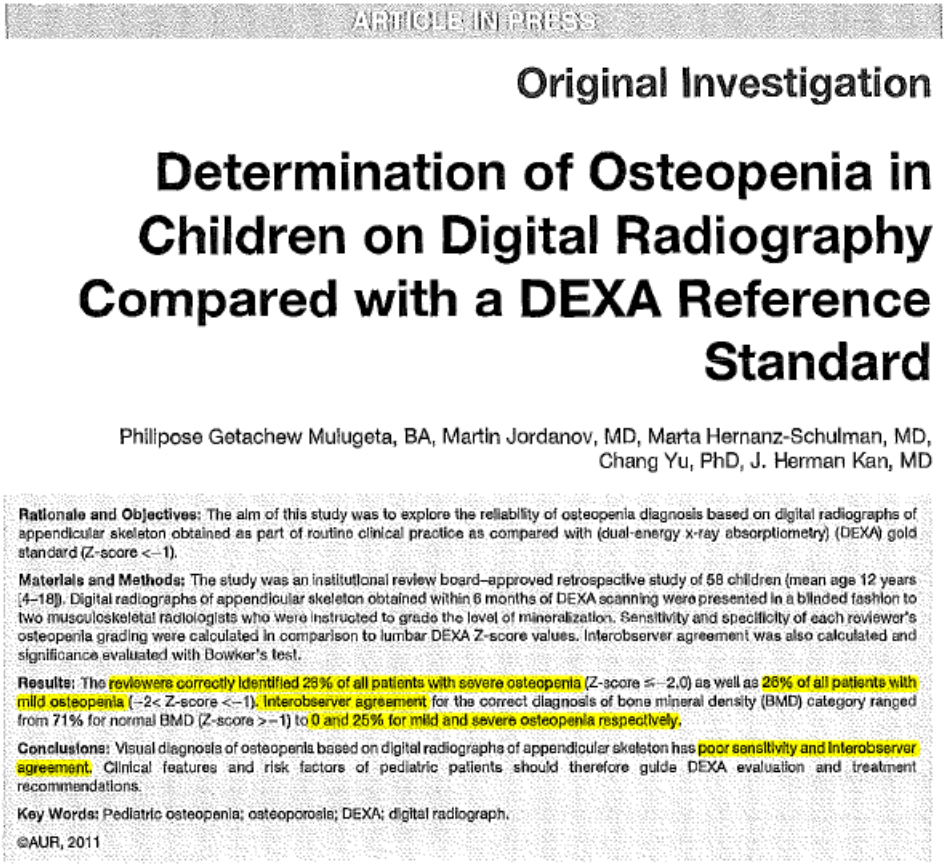


Figure 22. Pathologic fracture of R humerus typical when a baby is pulled up by arms with normal handling. Note the diminutive or absent cortex of the affected bone. The baby's own weight is sufficient to cause this fracture with VDD/IR.

2.14 Osteoporosis

Osteoporosis, never tested for and avoided in pediatric child abuse, will be subjectively declared absent by radiologist in their reports and relied on by CAPs in court testimony, even after studies showed 0% accuracy in determining osteoporosis in known cases of IR (Note 77) by looking at plain X-rays.



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Original Investigation

Determination of Osteopenia in Children on Digital Radiography Compared with a DEXA Reference Standard

Philipose Getachew Mulugeta, BA, Martin Jordanov, MD, Marta Hernanz-Schulman, MD, Chang Yu, PhD, J. Herman Kan, MD

Rationale and Objectives: The aim of this study was to explore the reliability of osteopenia diagnosis based on digital radiographs of appendicular skeleton obtained as part of routine clinical practice as compared with (dual-energy x-ray absorptiometry) (DEXA) gold standard (Z-score <−1).

Materials and Methods: The study was an institutional review board–approved retrospective study of 58 children (mean age 12 years [4–18]). Digital radiographs of appendicular skeleton obtained within 6 months of DEXA scanning were presented in a blinded fashion to two musculoskeletal radiologists who were instructed to grade the level of mineralization. Sensitivity and specificity of each reviewer's osteopenia grading were calculated in comparison to lumbar DEXA Z-score values. Interobserver agreement was also calculated and significance evaluated with Bowker's test.

Results: The reviewers correctly identified 29% of all patients with severe osteopenia (Z-score ≤−2.0) as well as 26% of all patients with mild osteopenia (−2< Z-score <−1). Interobserver agreement for the correct diagnosis of bone mineral density (BMD) category ranged from 71% for normal BMD (Z-score >−1) to 0 and 25% for mild and severe osteopenia respectively.

Conclusions: Visual diagnosis of osteopenia based on digital radiographs of appendicular skeleton has poor sensitivity and interobserver agreement. Clinical features and risk factors of pediatric patients should therefore guide DEXA evaluation and treatment recommendations.

Key Words: Pediatric osteopenia; osteoporosis; DEXA; digital radiograph.

Figure 23. Abstract of Mulugeta article

Efforts to develop scanning techniques (DEXA) to identify osteoporosis, a diagnostic sign of rickets, appear to have been abandoned.

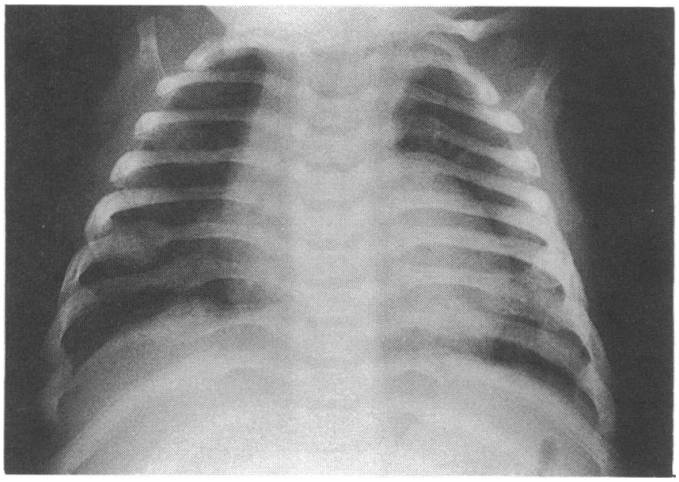
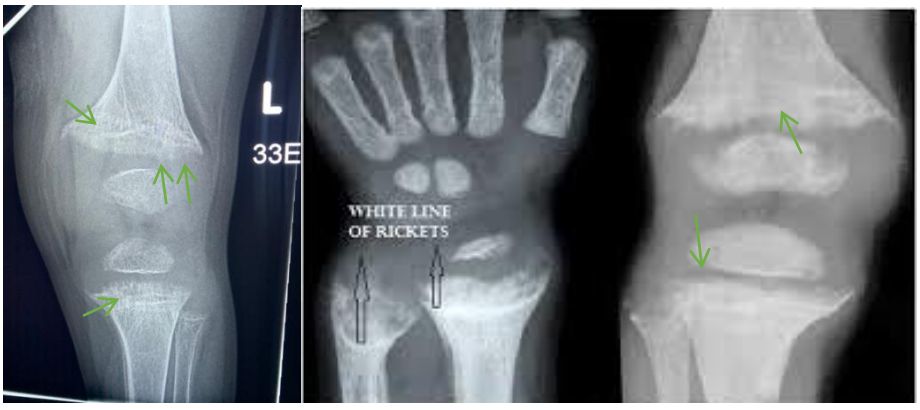


Figure 24. Multiple rib fractures and absence of cortex (thickened outer surface of ribs) in osteoporotic ribs

2.15 Osteoporosis

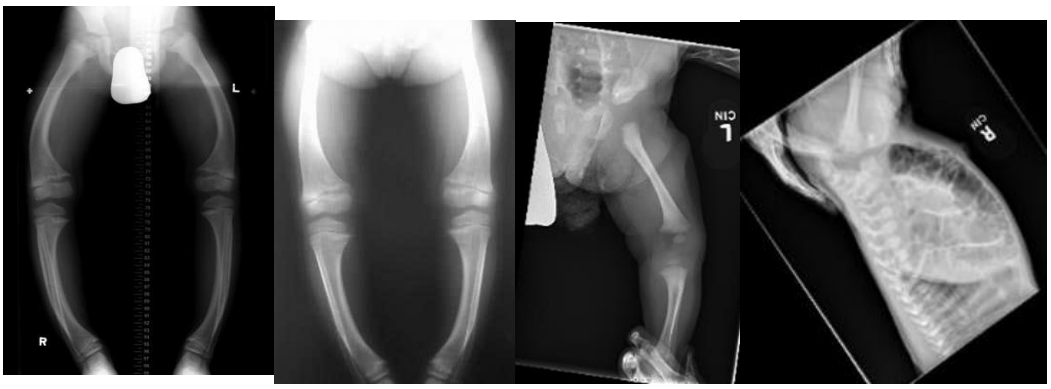
2.15.1 Some Other Common Rickets Findings

Other rickets findings: beaking, spurring, irregular calcium deposition into cartilage, bowing, lack of bony cortex in long bones and abnormal anatomy in bone growth area are routinely ignored or declared normal. (see Figure 25)



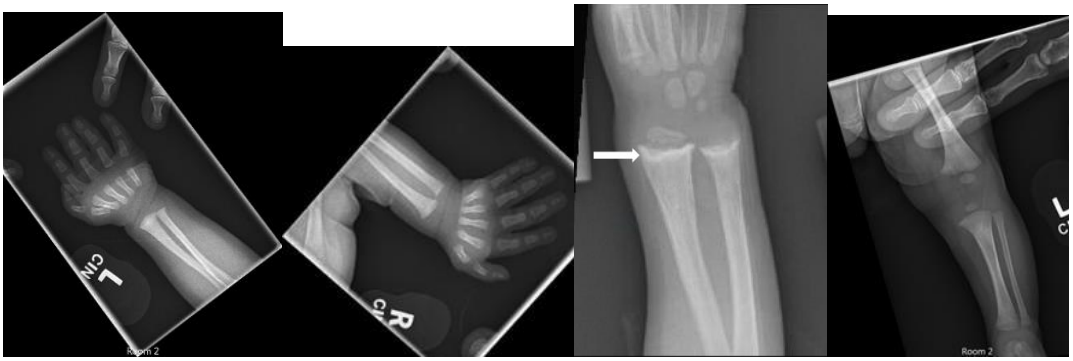
a b c

Figure 25 a-c. Figure a: shows frayeds appearing growth plate at double arrow and bealing and spurring at single arows. Upper spur is what is called a classic metafeseal leasion (CML). Figure b: more intesnse fraying (blurring) of the growth plate and white hyperdensity (metafeseal hyhperdensity marked as “white lines:. Figure c: another example of fraying of the metaphysis (growth plate),



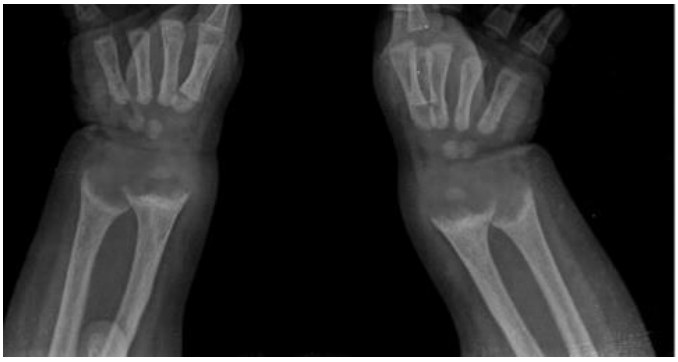
e f g h

Figure e, f and g: varying degrees of bowing of the legs. Figure h lateral view of spine showing irregular deposition of calcium in vertebral bodies creating the illusion of "bone within bone"



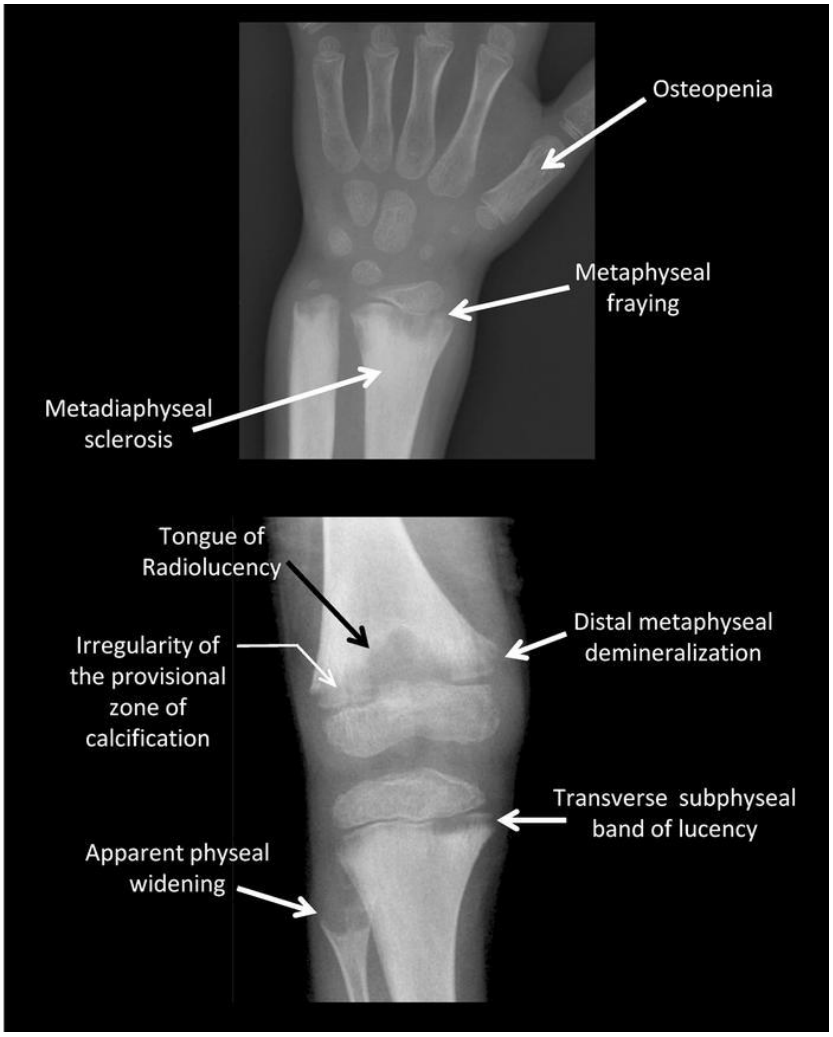
h i j k

Figures h through j: metaphyseal hypermineralization and cupping at the distal dend of the radius and ulna (wrist bones-white arrow). Figure k: beaking and spurring at the left elbow



l

Figure l: extreme cupping of both wrists and metaphyseal hypermineralization



m

Figure m: additional rickets findings

2.16 Delayed Healing with VDD

Compounding the issues with VDD present, healing of any bone injury from birth is retarded (extended) and the timeline stretched (Note 78), creating more confusion and latitude to accuse parents of PA by taking advantage of elongated and unpredictable timelines of healing with VDD.

2.17 VDD, the Common Thread

All these bone findings are swept into abuse narratives whenever possible and were posited and diagnosed as postnatal abuse fractures, and worse, that the seemingly decent parents are openly accused of lying about causing them.

The complete absence of pain, no decreased range of motion or tenderness and limited, or absent, conventional healing findings seems to do nothing to stop the proposition that all these rickets findings are physical abuse and real abusive “fractures”.

2.18 The Impact of the Second Set of Abuse Findings

The inflammatory nature of a second set of abuse findings cannot be underestimated. With SBS under attack from 1985 on, misdiagnosing rickets as abuse was a strong accelerant to the misuse of power and manipulation of the legal system to generate accusatory energy in critical early moments in a child abuse investigation. The failure to follow or acknowledge normal healing patterns through the months that follow, is further ignored as accusations of PA will be permanently be used over years of CPS involvement, which is inevitable.

With deficient VD, blood in the head without external injury (AHT) and rickets related false accusations of physical abuse (PA), links VDD as the common thread in the vast number of false accusations of child abuse that have occurred in the 50 years of modern child abuse pediatrics. It should be clear that VDD sits at the core of both of these prongs (AHT and PA) of these false accusations.

3. SBS/AHT in Court

3.1 First Uses of IR Finding to Alleged Abusive Fractures

In the late 80's, with SBS/SDH now consistently convicting innocent caregivers with machine-like precision, the resurgence of infantile rickets in the 1980's (caused by the same VDD that resulted in false accusation of SBS), opened a new and expansive front for false accusations that proved irresistible to committed accusers.

3.2 Recapping the Modern Child Abuse Pediatrics; the Organization, Promulgation, and the Application of a Pseudoscience in the Courts

Historically, with the emerging VDD/IR findings (with or without SDH and/or RH) slowly becoming more common in the 80's, the conundrum of what was perceived as "unexplained" retino-subdural hemorrhage of infancy, became of particular interest to a previously nonexistent group of academic pediatricians (the future self-proclaimed child abuse pediatricians). Almost all the cases being seen had no sufficient external injuries to account for the intracranial findings or the bony findings. Without reference to existing science, this group of pediatricians, only well known to each other, met under the auspices of the American Academy of Pediatrics (AAP), and formed the Committee on Child Abuse and Neglect (COCAN) within AAP. This committee became, and remains, the controlling entity within AAP on all matters related to child abuse. Included in its functions are the creation and rapid distribution, specialty wide, and to legal authorities, of new tactics, strategies, policies, acceptable rhetoric, promotional strategies, training, legislative initiatives, and the distribution of criticism and strategies to denigrate any critics/challengers to their dogma, among other things. All these initiatives are done via official AAP publications including the house organ, "Pediatrics" and another in-house publication called the "Quarterly". The "Quarterly" is focused on the quick distribution of information to the child abuse pediatricians that is openly promoted as useful in defeating medical and legal challenges and prevailing in court proceedings. (Note 79)

At AAP, in 1975, this group, started by eschewing experimental biomechanical analysis and using a flawed extrapolation from ~~a~~ high force whiplash research on monkeys in simulated 40 mph motor vehicle collisions studying head rests in rear end impacts (Note 80). The study was used effectively to conjure up false and inaccurate images and comparisons to shaking abuse which generated 1/125th of the force in the simulated MVAs. From this point, they began to concoct an explanation focused on shaking. Misusing this Ommaya study that had been published just a year before, their primary challenge was to craft a hypothesis that could deal with the unexplained SDH/RH they were seeing, particularly in the context of no external trauma, and promote it.

What they decided was that shaking (presumably a form of "whiplash" taken from Ommaya's studies related to whiplash head injuries and headrests in cars), not impact, was going to ~~be~~ promoted as the cause of the findings. The SBS triad hypothesis (SDH, RH and the redundant notion of forms of brain damage) were formally codified and promoted without a valid scientific basis, via official AAP position papers, as a diagnostic "triad"; pathognomonic of shaking abuse. These policy papers/statements were immediately hugely impactful in the US and later across the globe. After, without any biomechanical testing or anatomical corroboration, the "hypothesis" was promoted to a "theory"; falsely representing SBS as a proven fact. The explanation for the findings both in medical training and through published papers was disseminated via an organized campaign to produce papers, using specious or invalid peer review, to promote these unsupported ideas (Note 81) and instantiate them in the legal system. As these ideas were created and promulgated, with a decidedly PR feel, there was a concurrent determined effort to use the ill-conceived SBS hypothesis in child abuse medicine practice, to suspect, accuse, and then control the prosecutions of caregivers in cases that child abuse pediatricians "believed" were abuse and were willing to testify, beyond a reasonable doubt, that the caregivers were guilty of child abuse.

At the behest of AAP, SBS was soon incorporated as a formal medical diagnosis in the well-established and universally used ICD-9 diagnosis coding system. This inclusion lent credibility to SBS and has been used very effectively, because the name itself, "shaken baby syndrome", insinuated intent, mechanism, and criminality, key elements in establishing a crime. These elements must be incorporated into indictments and in establishing intentionally; key in a medical diagnosis' for successful convictions. SBS is not a medical diagnosis, it was a verboten legal conclusion. What more accurately should have been called unexplained "retino-subdural hemorrhage of infancy" soon became both a bogus diagnosis and at the same time, a legal conclusion (Note 82) conveying that the crime was intentional abusive shaking. Later, forced to eschew "syndrome's" rhetorical constraints and unique linkage to findings, the word "constellation" was promoted. A constellation of findings allowed any grouping, however unrelated, like stars in the sky, to convey a cumulative inculpatory impact even when unjustified. It remains in use, as well as the SBS hypothesis, to convey guilt by amalgamating allegedly inculpatory findings into constellations in yet another rhetorical deception to convict innocent caregivers.

3.3 The Misuse of Inapplicable Evidentiary Standards in Prosecuting Child Abuse

After organizing, and crafting the hypothesis, the newly minted child abuse pediatricians almost immediately, approached legal authorities offering their hypotheses as "theories". Hypotheses need to tested and theories are supposedly proven. Their hypotheses were not proven but we're falsely represented as such. Their testimony was however accepted and regarded as valid evidence under a "general acceptance" or "consensus" standard (the Kelly-Frye evidentiary standard) which was the dominant evidentiary standard at that time in state courts for expert testimony to be admissible. Since, a more stringent scientific standard (the Daubert standard (Note 83)) has had increased acceptance in a significant number of states. Yet, CAPs aware their thinking could not satisfy the Daubert standard continue to actively promote as validated "consensus" beliefs (Note 84) to prosecute child abuse.

Using false assertions, cavalier accusations, suspect interrogation techniques, training programs for authorities, new "medical" diagnosis, and "authoritative" AAP policy statements they wrote themselves, they conveyed, and continue to convey, that they could "prove" abuse and deliver convictions to prosecutors. With that, their impact and influence grew. They were quickly, predictably, and reliably able to allege and "prove" abuse. They were so successful in using these unreliable hypotheses and supposedly "diagnostic" findings, in the first 10 years of modern child abuse pediatrics that 94% of accused caregivers were convicted and of the remaining 6%, 82% pled out to some form of abuse (Note 85). Via direct contact with local authorities and evolving partnerships with prosecutors interested in high profile cases, they were immediately welcomed by local law enforcement and elevated in the local legal community and became the sine que non of successful prosecution of child abuse; which, indeed, they are!

The reception was so overwhelming, that it justified mass training sessions for thousands, conducted through the late 80's and 90's in San Diego, promoted by David Chadwick, who could be regarded as the founding father of modern child abuse pediatrics and a hugely effective promoter of modern child abuse pediatrics. Thousands of lawyers, judges, doctors, DAs, social workers, and other MD's, were indoctrinated in week long training sessions, year after year, with these unproven beliefs and new ideas that were already demonstrably successful for prosecutors and were already in use. Prosecutors themselves promoted strategies to other prosecutors to win abuse cases relying on these bogus constructs and widely distributed legal attack strategies against successful challenging experts. As more challenges and medical criticism emerged in the mid 80's, prosecutors and CAPs began to craft and disseminate new attack strategies and methodologically flawed pseudoscience via flawed published articles to be used against any critics or challengers, the future defense experts.

In the courts, the CAPs and the prosecuting DAs decided to intentionally continue to take advantage of the inapplicable evidentiary standard used at that time: the Kelly Frye standard, ("general acceptance").

In the 1975-1985 time frame, the founding members of COCAN and their colleagues, touting consensus among themselves and the body of medicine, were allowed to present so-called "expert testimony" that "proved" abuse, simply because only this small group all agreed with each other about

their own fabricated, self-crafted hypothesis. They falsely claimed "general acceptance". However, these like-minded pediatricians, numbering in the low hundreds at best, were only agreeing with themselves, which is neither general acceptance nor legitimate consensus (Note 86).

3.4 The Amalgamation of Findings and False Accusations

In a final contortion of logic, and increasing challenges to the constraints of the term "syndrome", any and all findings, substantial or not that were identified in any case, it was proposed, would be amalgamated into a "constellation" or a relatively new term, "cluster" of findings and packaged and promoted to support the abuse narrative. These concocted, nonmedical, nonscientific constructs (c.f. syndromes) have no place in these cases. All the elements above create an illusion of linkage of separate findings to abuse, however, the link is illusionary and intentionally designed to illogically take a number of unreliable findings used to misdiagnose abuse and suggest against laws of logic, that together there is an increased probability of abuse. Logic says that individual, improbable, nondiagnostic, and nonspecific findings, when collated, collectively, result in significantly lower probability of the fabricated conclusion of abuse. Improbability times improbability equals more improbability. Yet, these flawed amalgamations are increasingly invoked with success and then improperly and thoughtlessly accepted as valid testimony and evidence by a seemingly mesmerized legal system. Medical and legal authorities in the past, have accepted, without question, child abuse pediatrician's' biased, false certainty statements that align with their training by CAPs, past practices and career ambitions, to accuse, prosecute and convict. Ignoring their individual oaths to do no harm and to seek justice, they collectively, knowingly or unknowingly, seek convictions by ignoring established evidence based science, evidentiary rules, principles of logic, probability, and common sense.

Soon, they adopted a GroupThink approach (Note 87) to maintain control in the hospital and court by falsely representing that the body of medicine agreed with them, when no one outside of their group knew what they were doing, nor could any other authority (other specialists) seemingly have any impact on this small group. In reality, the declaration of general acceptance offered to the courts was a deception. Those within the hospital who had doubts were suppressed and at times threatened by GroupThink strategies.

For the first 10 years after COCAN built the SBS myth, without outside experts, willing to stand up to GroupThink, there was no awareness in the body of medicine about what COCAN was hypothesizing and to what exactly they were testifying about. The fact that many cases were in juvenile court, which is closed and secretive, helped perpetuate the lack of scrutiny. The inappropriateness of such a standard (consensus) of determining the evidentiary validity of such science-dependent propositions was just bowled over by the instant success of convicting virtually any target caregiver. Touting convictions as validation, along with plea deals called "confessions" they established a toehold of undeserved credulity in the legal system. In large part, based on these convictions and pseudo confessions, the modern age of child abuse pediatrics began on a new scale, not previously seen.

3.5 Misleading Published Statistics and Circular Logic

As the cases accumulated, previously misdiagnosed cases of SBS/AHT and PA in the hospital databases recorded as "abuse" could be amalgamated into what is proposed to be "research." The flaw was the circularity to use findings (like SDH and RH) to diagnose abuse, and then to declare these findings, when present in all cases misdiagnosed as abuse, common to previous misdiagnoses, were now represented as diagnostic findings of abuse. The claim was that the findings used to misdiagnose "abuse" in the first instance of misdiagnosis (blood in the head and skeletal abnormalities), could now be characterized as proof of abuse. This circular logic is flawed and was later revealed as an (Note 88) illegitimate methodology, unexpectedly exposed by the same Swedish scientific panel that picks the Nobel Prize winners. The abuse diagnoses built on such "research", in reality is bogus, and is derived from the amalgamation of other misdiagnoses.

3.6 The Deal That Can't Be Refused

With almost certain convictions hanging over every innocent caregiver, many hopeless parents, and most interested in getting their kids back or avoiding long sentences that had been engineered by CAPs and DAs via legislation, will give up the fight and take a "deal". Most favorable deals are likely an acknowledgment by prosecutors that target caregivers are innocent. These accused are effectively offered an opportunity to admit to lesser things they have not done, get their kids back and avoid incarceration, and to take the settlement deals that are much worse than a frank dismissal of charges. Any guilty verdict or deal serves the prosecution and the CAPs. The somewhat coercive settlements are designed to be "deals that cannot not be refused" and for the most part work. The reason that such deals are offered, vs dismissal, is that for CAPs and prosecutors, the deals and confessions are regarded as true guilty verdicts. As such they can be used to augment their professional aspirations and "successful" stats, when many, if not most, were nothing but caring parents seeking medical care for sick children.

3.7 The Role of Convictions and Confessions to Establish Validity

For the DAs and CAPs, convictions "confessions" and settlement deals are considered convictions. These stats, shocking as it may seem, were then used for the initial scientific validation of SBS accusations and we're offered as "proof" to authorities that SBS was real. Strangely, based on blind faith, these non-medical pronouncements were embraced by law enforcement as validation of SBS, pushing new prosecutions forward.

Other MD's, emergency physicians, neurosurgeons, neurologists, orthopedists, and biomechanics, with experience with these findings, the future defense experts who would question the validity of SBS, were not in the picture for that first 10 years. The future defense experts' arguments would highlight that there were many other causes of SDH and RH, already known, all more possible, plausible, and more probable than abuse. They would testify accurately that the SBS hypothesis was fatally flawed (Note 89, Note 90, and Note 91). They would identify an increasing number of symptomatic cases of increased ICP in the postnatal period, how they occurred, and evidence of rebleeds from PSDH damaged areas, all became more common, and would emerge as the likely non abuse etiology of

rebleed findings being used to misdiagnose SBS as "acute on chronic". Social science would identify low risk parents accused of heinous acts that were highly likely to be innocent (greater than 95% likely), and that legal evidentiary standards, like Daubert, based on defined scientific standards, when calculated (see below analysis of probability with Bayes Theorem), would disallow and invalidate false certainty statements declaring guilt. Analysis invoked the need for the Daubert-like standard to be applied to reach the threshold of valid evidence and testimony in all SBS cases.

3.8 The Abuse of Authority

The notion of authority figures saying anything and expecting to be believed when operational is a powerful tool. Authoritative statements from well credentialed full professors or "certified" professionals, whether consistent with facts, conventional thinking, or contradicted by accepted scientific studies, obviates the need for a valid scientific foundation, or even the application of common sense in court. The early CAPs were ironically, despite their pronouncements of general acceptance, initially operating outside the mainstream of known, existing medical knowledge, published research, and scientific methodology. Realizing the latitude they created through this abuse of power, and using it, became integral to the rise and persistence of modern CAP and legislative immunity from malpractice, both of which accelerated the lack of caution in misdiagnosing abuse. Their reports were encoded with probabilistic language (language suggesting imprecise mathematical probabilities (like "suspicious of", "concerning for", "consistent with", etc.) yet inferring via a code-like agreement, "diagnostic of". Authorities' probabilistic statements were blindly assumed to be reliable and definitive. In these cases, without a valid scientific basis, this persuasive capability is based entirely on abuse of power.

Regarding general acceptance, literally, almost every other doctor, anywhere, had no idea what so-called child abuse pediatricians were doing from 1975-1985. When exposed to these self-proclaimed academic child abuse pediatricians, the trainees that followed them, predictably and naively accepted the single compact, and catchy notion that "SDH and RH was uniquely diagnostic of SBS". It is not. (Note 92). Yet, locally, operating out of regional children's hospitals, the expanding corps of child abuse pediatricians were more than willing to spread these ideas and educate local prosecutors, police, social workers, trainees, and other doctors, about their pseudoscientific hypothesis creating local prosecution systems that were well oiled. Based on the presence of SDH and RH, the CAPs would say they were "certain" (actually falsely certain)**,** "beyond a reasonable doubt" that the child abuse accusations, they had created themselves, using the flawed SBS hypothesis, were valid and true. Authorities taught by the same CAPs agreed and prosecutions followed.

The CAPs' willingness to abdicate scientific methodology and to testify for prosecutors in court, misusing false certainty statements, was the essential ingredient and the evolving foundation of a false accusation of child abuse. As convictions piled up in the mid 80's and early 90's, a prosecution machine of sorts, evolved, this became virtually unstoppable. Thousands went to jail unable to rebut the made up hypotheses. Many innocent caregivers, who did not abuse, took plea deals and made false "confessions" to save themselves from threats of long sentences, the threat of permanent loss of their

children, the incarceration of their spouses, and to try to reconstitute their families. For CAPs and prosecutors both end points (confessions and plea deals) were regarded as true guilty verdicts, when many, if not most, were not.

Absent any scrutiny, the falsely high conviction rate pushed the movement forward. With no experts for the defense being used until after 1985, and other outside specialists unaware of what was happening, there was no resistance. Later, after challenges from defense experts, conviction rates decreased and rates of convictions were replaced in child abuse statistics by the rate of accusations. Guilty or not, accusations, which could be leveled without comporting to legal principles and standards, probability analysis, evidentiary standards, or scrutiny in court, could be generated by a single person and were always assumed to be correct, regardless of legal endpoint, as valid guilty verdicts. Accusations were regarded by the accusers and their CAPs colleagues as true positives. Guilty or not, accusations became the metric in defining the scope of child abuse used in generating an energized hysteria regarding child abuse in the public consciousness, winning cases, elevating careers, and importantly increasing grant funding. With accusations, proving actual guilt was not necessary to advance the cause that abuse was common and the false notion that it could be perpetrated by anyone, regardless of character. At that point, everyone accused, regardless of judicial end point, is regarded as a child abuser in reporting and documentation by the child abuse establishment. Not guilty in that paradigm, according to CAPs, was an abuser being given a pass by hoodwinked, misguided triers of fact, being tricked by defense experts. That said, based on the experience of current contested cases, it is likely that >90% of defendants contesting their charges in that time frame (and now), with plausible nonabuse explanations, and favorable social histories, both routinely dismissed by their accusers, were innocent.

Abuse of power created a combination of accusations, false confessions, and convictions that continue to push the field forward. Together, it has been used very effectively to promote and generate a purported pseudo-validation among law enforcement, jurists, prosecutors, and the public, and demonstrated that “say anything and expect to be believed” works.

3.9 The Co-Opting of Legal Authorities and Other Professionals

After the unvalidated ideas of modern child abuse pediatrics, were codified into methodologically (Note 93) flawed published papers in journals controlled by AAP or SBS sympathetic editors, they began and continued to impact key players in law enforcement. These early writings were then systematically and aggressively promoted, referenced, and presented in the official position papers of AAP, COCAN, and the American Academy of Ophthalmology (AAO), advocating the principles of current CAP. Within a short time COCAN and AAP used their fabricated thinking, flawed literature, and power to ask professional organizations worldwide to endorse these AAP supported notions. Without independent testing and research, which did not occur, such consent would be based only on blind faith in AAP; something that most would regard as reflexively justified. The effort on the AAP side was to validate the inapplicable rubric, and make a show of “general acceptance” of their ideas. Of the groups responding, written endorsements were readily given.

Once endorsements was obtained, it allowed for lists of "supporting" organizations to be presented in court to demonstrate a flawed cross validation of consensus or general acceptance, a benefit to both AAP and the endorsees, and it was effective. Done with intent, the consequence of these groups' endorsements is that they were then unknowingly dragged into the emerging controversy about the validity of the supposed fund of "knowledge" they were endorsing regarding child abuse medicine. The understandable and long standing faith in AAP and their CAP's, with the flow of copious, unrecognized but flawed literature, sanctioned intentional false certainty statements to legal authorities. The result created, and continues to create, within the legal community and many physicians, outside the child abuse establishment, a real resistance to any nonabuse explanations. The authorities' long term and unwitting acceptance of such misinformation has created a situation where many authorities, impacted by thoughtless, insufficiently researched acceptance and embrace of these ideas, now have a large psychological stake in this controversy and a massive conflict of interest regarding questioning it.

When controversy arose for the first time in 1985, the decision to continue to support the pseudo-science became one that included factors such as self-preservation, continued tolerance of seemingly improbable ideas, and elevation of their own, now apparent, self-interests. Many prosecutors and ex-prosecutors, now judges were being fed negative commentary about the early challenges and challengers, and the denigration and gross dismissal of the existing and new science (similar to that presented in this article). The choice was to continue to rely on the local CAPs and their self-proclaimed beliefs and to continue to convict many innocent caregivers, or stand up for valid science. Many prosecutors being judges adding a new layer of conflict that is easily identified in court proceedings in some cases. Opinions expressed by CAPs are too often, and commonly, false, or misleading, and the vulnerability of the listeners, to CAP dogma, to be deceived with false certainty, remains intact. Social workers, police and other medical professionals as a result were effectively pressured to believe and defend these false ideas too, as their own identity was at stake in that process as well. This situation, as anyone can imagine, had a huge impact on bringing the truth forward. If they questioned the decisions they had already made in the past and the damage they had done in their professional lives, the potential ramifications were unpalatable. The denial of common facts and findings occurred to such a degree, that it has generated concern about the academic, clinical, objectivity, cognitive competence, and/or allegiance to just decision-making and justice, for all involved. Given all this, the legal alarm and scrutiny necessary to recognize the disregard of scientific methodology, referenced in Daubert, should be at the highest level, but sadly, it wasn't and isn't yet. Accepting all the damage that had been done, and any responsibility for those misjudgments and decisions, proved to be a psychological hurdle that many cannot overcome.

With 60 to 90% of babies being born with VDD/IR, the findings being misused to diagnose abuse, it is clear that VDD/IR findings have no scientific validity as diagnostic of abuse at all, down either prong of misdiagnosis caused by VDD/IR: AHT and/or PA. Yet, the clarity of that remains obfuscated. All along, the establishment's leadership interests have been represented by doubling down and denigrating

the valid challenges. Fear, intimidation, power, position, security, and culpability, define a toxic swarm with the intended goal of the establishment to maintain the status quo.

4. Part IV Bayes Theorem: Calculating Improbability

4.1 Calculating the Improbability of Abuse for Low Risk Caregivers When Using Unreliable Findings

In the final analysis, it is clear that the symptoms, signs, and findings of CSDH and VDD/IR are being misused to diagnose abuse. It is also clear these findings are nonspecific for abuse and unreliable indicators of abuse. The next question is, how unreliable?

The final component of identifying the failure to acknowledge and correct VDD and eliminate false accusations of child abuse is to determine the mathematical unreliability of the thinking and criteria to diagnose abuse in use by CAPs. Both the risk of abuse (prior odds of abuse) and the reliability or unreliability of signs (or indicators) of abuse, can be analyzed using Bayes Theorem of Probability. Bayes Theorem, a 200 year old probability calculation, estimates the reliability of the key factors and findings discussed herein, and returns values that strongly suggest and confirm that the findings of SBS/AHT and IR, being misused to accuse, have no evidentiary value relative to the issue of abuse.

Regarding PSDH to CSDH, what is known is that birth trauma and complications related to VDD/IR, and supported by proven science, are highly probable events. The high probability of birth related trauma however, alone, is not useful. Without direct objective or witness evidence of abuse, Bayes shows us the actual probability of abuse is near zero.

If VDD/IR is regarded as an underlying causation of false accusations of abuse, intentional disregard of existing valid science, and relevant probabilities regarding VDD/IR, by legal authorities, manifests. What is simultaneously increasingly clear is the unreliability of SDH and RH, of any etiology, as indicators of abuse. With nonabuse explanations related to rickets, like VDD/IR/PSDH/CSDH, possible, plausible, and statistically probable, to >95% probability in many analyses, the probability of false accusations is very high as well. Any reliance on the presence of the findings, SDH and RH, as indicators of abuse, is unwarranted and misguided. From a legal and Forensic Epidemiological (Note 94) perspective, claiming that SDH and RH are diagnostic of abuse does not qualify as evidence.

4.2 The Components of Bayes

Bayes theorem is composed of two parts. The first part asks the question what is the frequency of an event or factor being studied (i.e. abuse), in a population that is defined by the user. This probability is called the “prior odds”; the odds of the event (abuse), occurring in the defined population. The second part is an analysis of the reliability of the finding or what is called an “indicator” (like blood in the head, RH or a bruise), to predict that the event in question (abuse) occurred. In other words, how often is 1) the indicator right and will properly determine if the event (abuse) has occurred and 2) how often is it wrong when it is used to allege abuse when no has occurred. These categories do not intersect. The ratio of correct decisions to wrong decisions generates a calculation called the “likelihood ratio” (LR). The LR, old as it is, uses conventional sensitivity and specificity in a novel way to generate the

reliability of the indicator and ultimately, the actual odds of abuse having occurred (the "posterior odds").

Prior odds x Likelihood ratio = Posterior odds

4.3 Prior Odds

The frequency of an event in a defined population is the "prior odds". We can ask questions like, what are the prior odds of abuse among previously caring and loving parents? Or, the odds of child abuse in a population with a history of domestic violence? Without witness evidence of abuse, is it reasonable to assume that any random person in either category is a child abuser? Each of these questions can be explored in combination with other factors, circumstances and findings using Bayes to generate reasonable determinations of the factors' evidentiary value in a legal or medical framework.

The first component in Bayes, the prior odds is a very important statistic that is unfortunately systematically and intentionally disregarded by CAPs. In any defined population, what we will see is a spectrum of probabilities with lower risk caregivers at one end and more likely to abuse caregivers at the other end. Bayes' reliability to show low or high risk in any population or sub population (prior odds) is particularly important, especially when we see how unreliable the designated "indicators" of abuse are when analyzed below.

Addressing prior odds is documented in the medical record, in the social history (SH). SH addresses family life, living situations, friends and relatives, and various personal behaviors in dealing with circumstances related to occupations, finances, child rearing, stress, and the recreational aspects of the caregivers' personal life. These factors have the potential to be, and almost always are, clinically relevant. Some might say they are the most important factor. Data collected in this vein can be used to define various subpopulations. Then we can use available data, to confidently estimate the number of acts (abuse) being studied in that defined population.

The prior odds, based on risk assessment, are likely the most important factor in the identification of potential false accusations of abuse both mathematically and from the perspective of common sense. This thinking allows one to see how inappropriate ignoring key social data is because it defines the actual baseline probability of abuse in a defined subgroup.

Social history in the aggregate, provides criteria to determine what the prior odds are that the caregiver/defendant would abuse at all. Prior positive parental behaviors, or lack thereof, known acts of caring, responsible behaviors with respect to caring for others, prior child rearing, and confirmation of no excessive drinking, drugs, or psychotic behaviors, are strong indicators of nonabuse. Knowledgeable family members' affirmations of seeing love and positive caretaking, witnessing satisfactory management of emotions, are all very relevant and important indicators of nonabuse and should not be ignored. The notion that anyone can abuse and the well-worn, misconstrued rationale to assume any person will abuse, is false. 'Decent people do not abuse', is much, much closer to the truth and a more reliable premise when people appear decent, as most people are.

4.4 Defining Prior Odds

The starting point for defining the actual probability of a low risk parents abusing, occurred in 1977 in a study by Kempe (Note 95). Dr. Kempe is the physician that Denver children's hospital is named for, and the founder of the journal Child Abuse and Neglect. His 1977 paper, trying to identify low risk families likely or unlikely to abuse, looked at 50 mothers at birth who were assessed by Kempe himself, and found to be "low risk" of abuse. These 50 were followed for 2 years; none abused or had any contact with CPS. This establishes a probability of any low risk mother (and their family) of abusing as less than 1 in 50.

A more detailed and usable understanding of probability and prior odds of any group of caregiver committing child abuse, can start with using overestimates of the probability of abuse for the defined group. Essentially, if we assume that all prosecuted cases of abuse in the US (~4000 (Note 96)) are due to one factor, the factor we are analyzing, we will overestimate the frequency of abuse due to that factor by a wide margin. A single factor is involved in only a fraction of all prosecuted cases, and in making an overestimation, in this way, is the most conservative analytic pathway.

Looking at two apparently different situations, the hypothetical maximum probability of abuse can be calculated. The probability of abuse by two different types of groups is illustrated when considering abuse by decent parents with no history of abuse, versus a group with a frequently used "concerning" factor/behavior like domestic violence (DV).

DV is one of a group of circumstances or factors, frequently used to infer abuse by overzealous accusers. Other factors used on a similar way, such as drugs like marijuana, moderate alcohol use, non-child abuse criminal behaviors, military service, being teenage parents, or any other supposed indicator of abuse are frequently used by CAPs to infer abuse to unsuspecting triers of fact.

Once identified, abuse by these groups can be similarly overestimated to the hypothetical maximum (4000) to generate the probability of abuse in any and all of these user defined groups. While the actual number of DV child abusers is difficult to ascertain, this approach to generate overestimated probabilities is quite useful in understanding false certainty and the actual probabilities of abuse related to the subject factor (DV) being analyzed.

Looking at the two groups and doing the math, we can safely assume there are 100,000,000 decent parents in the US and we know that there are about 10 million DV incidents and perpetrators per year in the US (Note 97). If we look at all prosecutions in the US a year (~ 4000) and propose that all are due to one factor (DV), we will overestimate the frequency and prior odds of abuse in the group/subject population. 4000 prosecutions caused by DV alone is an extreme overestimation, but if we use the hypothetical maximum we can then compare that number to the total number of people in the defined subpopulation (all DV perpetrators). With DV, the number of incidents is 10,000,000. This generates maximum prior odds of abuse by a DV perpetrator at 4000/10,000,000 or 1 in 2500. If on the other hand, we ask what are the prior odds of a previously decent parent abusing for any reason, the denominator, with the estimate of decent parents, increases by10 fold. If we estimate 100,000,000

decent parents in the US, this generates odds of any prior decent non-abusive caregiver abusing, without any overestimation needed, at 4000/100,000,000 or 1 in 25,000.

What this means for the first group of DV perpetrators is, if DV is used as evidence of abuse, it will be wrong 2499 times out of 2500 uses. For apparently decent caregivers, without witnessed evidence of abuse (an irrefutable indicator), and any possible, plausible, or probable nonabuse explanation (including VDD/IR), the probability of being right about an accusation of abuse for that decent caregiver starts at 1 in 25,000.

It is normally overwhelmingly clear that an accused caregiver can be identified as apparently loving, caring, responsible, and decent. The opposite is also quite true. The confirmation of this, either way, is soon obvious during complete CPS investigations or any formal risk assessments of accused caregivers and families. Sadly, what is found is systemically dismissed by the child abuse establishment as having no value. Positive caregiver behaviors in various forms may likely be the most reliable indicator of 1) nonabuse, and 2) a high probability of false accusation.

4.5 The Likelihood Ratio (LR)

Once a subpopulation is defined and the overestimated prior odds of abuse is established for that group, a test, behavior, or finding, collectively referred to as an "indicator" must be applied to that population to determine the actual odds of the studied event (abuse), having occurred, which is represented as the "posterior odds". The reliability of the indicator must be determined to reach the target endpoint: the posterior odds.

The indicator must be evaluated to determine its reliability to deliver the correct result. In abuse cases a wide variety of indicators like SDH, RH, bruises, healing fractures, poverty, a lab test, race, age, prior criminal behavior, etc., can come into play. The likelihood ratio (LR) is the calculation that defines the reliability of the indicator to deliver the correct determination. It is expressed as a probability, or an equivalent ratio. This ratio, the likelihood ratio (LR), relies on conventional sensitivity and specificity to be calculated.

When we have statistics from studies about sensitivity and specificity of an indicator, we can calculate the reliability of an indicator expressed as a ratio of two probabilities; in general terms, the probability of being right compared to the probability of being wrong. The indicator's inherent reliability, the likelihood ratio (LR) is expressed as a ratio or probability, calculated separately and independent of the subpopulation's prior odds of event occurring.

In general, the probability of the evidence (an indicator) to lead to correct diagnosis, a true positive (TP), is the sensitivity. The unreliability of a piece of evidence is a variant/mirror image of specificity and is expressed as a false positive (FP); how often is the test wrong (suggesting abuse when it did not occur). The ratio of these numbers is the LR.

LR = percent true positive (right)/percent of false positive (wrong)

Indicators (and their LR) increase or decrease the posterior odds of an event (abuse) occurring, depending on the calculated reliability of the indicator. Unreliable indicators (like random small bruises

which all children seem to have) will decrease the established prior odds (the probability in the defined population of abusing), to even lower levels. The resultant low probability of two low probability components, by any legal or medical standard, have, according to established values in Forensic Epidemiology, no evidentiary value.

Reliable indicators can trump low prior odds to levels where the overall probability (the posterior odds) of an event (i.e. abuse) having occurred, can reach thresholds of probability that can be probative by medical or legal standards (> 95% certainty). For example, a bloody baseball bat with the victim's DNA on it and a depressed skull fracture that matches the shape of the bat, with the perpetrator's fingerprints on the bat in blood, and a DNA match, will trump the low prior odds of abuse even in the general population of all parents.

In summary, with Bayes Theorem, two probabilities, combined by multiplication, generate a probability that integrates a prior probability of the event (abuse) occurring and the reliability of the evidence (indicator) used to diagnose the event (the indicator's LR). This calculation determines the probability (the posterior odds) of that event having actually occurred. Multiplying low prior odds times very weak likelihood ratios (nonspecific indicators) yields near zero posterior odds. Without witnessed evidence of abuse or other irrefutable data, assuming abuse by apparently descent parents, is ill conceived and likely to be wrong more than 24,999 times out of 25,000 cases.

4.6 The Significance of the Calculation of LR in Forensic Epidemiology

With any likelihood, ratios greater than 19 out of 20 (the ratio 19:20 is a probability of 95%), according to Forensic Epidemiology, is an indicator that can be regarded as "highly likely" to lead to the correct diagnosis (95% of the time). This meets the standard "beyond a reasonable doubt" or to "reasonable medical certainty or probability" standard, the evidentiary threshold to find guilt in criminal (Note 98) proceedings. These odds, according to scientific standards, qualify as evidence. On the other hand, any indicator with an LR of less than 19:20 is below criminal evidentiary standards (<95% certainty). Furthermore, as the LR decreases to say 10:20, the LR is 50% likely, (the preponderance standard), the indicator will yield the correct end result only 50% of the time. At 50%, the indicator will be wrong as often as right and is regarded by Forensic Epidemiology (See Figure 20), and hopefully, by thoughtful jurists and juries too, as "not evidence". Below are the Forensic Epidemiologic standards and conclusions.

Table 2. Posterior Odds as Related to Probabilities and Common English Terms

Note. Based on Goodman & Royall, 1988.

Table 3. Posterior Odds as Related to Probabilities and Common English Terms

Posterior Odds of Abuse vs. No Abuse	Probabilities That Abuse Has Occurred	Conclusion (in common English)
1:19 or less	5% or less	Very unlikely
1:19 to 1:4	5% to 20%	Unlikely
1:4 to 2:3	20% to 40%	Somewhat unlikely
2:3 to 3:2	40% to 60%	Undetermined
3:2 to 4:1	60% to 80%	Somewhat more likely than not
4:1 to 19:1	80% to 95%	Likely
19:1 or more	95% or greater	Very likely

5. Final Thoughts

"I know that most men, including those at ease with problems of greatest complexity, can seldom accept even the simplest and most obvious truth if it be such as would oblige them to admit the falsity of conclusions which they have delighted in explaining to colleagues, which they have proudly taught to others, and which they have woven, thread by thread, into the fabric of their lives." …**Leo Tolstoy**

"Personal convictions are more often greater foes of truth than lies." …Friedrich Nietzsche

The notion that accomplished, academically credentialed doctors that challenge the specious, if not unproven, grossly deceptive hypotheses related to SBS, RH, and PA, are "promoters" of child abuse or "denialists", common attack lines, is absurd, shameful, and smacks of school yard name calling. This, among many other epithets used in published materials and in legal proceedings, by CAPs and prosecutors, represents the lowest form of academic and professional discourse.

In reality, false accusations of abuse and actual abuse have nothing to do with each other. They are separate entities that literally have no meaningful crossover or overlap. The willingness to conflate them by child abuse pediatricians is the willingness to knowingly accept and destroy innocent families and the CAPs supposed protectees, the children. The act of falsely accusing, inflicts a uniquely awful form of medical child abuse; abuse caused by medical providers.

The AAP's official and systemic promotion of what, in face of probability analysis, can be called dogmatic (Note 99), cavalier, non-evidenced based, false certainty statements, are easily demonstrated as improper in a Daubert framework. Flawed methodology, circular logic, unsupportable disputed conclusions, no control groups, unknown error rates, non-reproducibility, and specious peer review by confederates, only in proprietary journals, are obvious. False certainty statements are being used intentionally, in the face of disingenuous, defensive, establishment pronouncements of their own supposed objectivity, and false declarations of careful consideration. The value of such unscientific

behavior, knowingly, or unknowingly, is in generating numbers and hysteria about child abuse and increasing the number of reported cases of abuse that accuse and convict innocent families.

All this is done, now overtly, under the rubric of "protecting children". Underlying this notion is the idea that if some number of innocent families are destroyed while rounding up real abusers, that there is a greater good, which benefits society and children. This is nonsense and gross callousness. When one considers the high percentage of innocent caregivers (90%+) falsely accused, and contesting their accusations, the scope of this damage is stunning and shocking.

Caregivers, contesting their cases, absent valid corroborating findings that show abuse, often having and giving plausible medical and nonabuse explanations, are arbitrarily dismissed as liars (Note 100). Reasonable explanations, some established via objective criteria, are called insufficient, based on a preconceived bias to diagnose abuse. Explanations offered by parents and defense experts are statistically possible, plausible, and more probable etiologies than abuse, often by wide margins. The ill-conceived and accusatory policies, destructive acts, and known consequences, that are callus and abusive to families and children, are a form of abuse that could, and should, be regarded as iatrogenic child abuse.

Improbable as that might seem to the unknowing, to whatever extent such policies knowingly promotes and accepts this destruction of innocent families, as collateral damage, is insane, abhorrent and has criminal and sociopathic overtones. The unqualified immunity, inappropriately extended to these child abuse pediatrician abusers, is the foundation of the perpetuation and expansion of false accusers and false accusations; such immunity should be rescinded.

6. Conclusion

Eliminating false accusations of abuse must be the top priority of all of us involved in these matters. There is a valid argument that false accusations of abuse are worse than actual abuse since the victims there are caregivers and children who have done nothing wrong, and whose lives are almost always ruined. It also seems clear that the number of false accusations dwarfs the number of cases of real physical abuse.

VDD/IR sits at the top of the list of causes of false accusations. Literally, thousands of infants and families are vulnerable to the misuse of VDD related findings to generate false accusations and the vast majority of cases accused and prosecuted, involve these issues related to VDD/IR.

Perinatal subdural hemorrhage, the result of maternal VDD induced craniotabes, and its complications, creates the first of two prongs of false accusations, the prong related to SBS/AHT. The second prong is VDD induced infantile rickets, whose findings are misused to allege physical abuse (PA), is equally destructive.

With the end of the epidemic of VDD with its awful consequence, so easily fixed, a real concern arises of why this solution is being ignored by AAP, AAO, and ACOG. The disregard, of the pediatric impacts to infants and family, has been primarily driven by AAP, and it must be addressed. Tolerating false

accusations is unacceptable, loathsome, and despicable. The intent herein, is to stop this large percentage of false accusations caused by VDD/IR and promote bone health and the many other benefits to having therapeutic levels of vitamin D. (Note 101)

Both initiatives can, by addressing mandatory testing and adequate supplements, are easily correctable, yet, still ignored for reasons that seem either thoughtless or fiendish. The positive impacts of an organized corrective strategy on the courts and society would also be substantial, as the negative ramifications of false accusations reverberate through society's consciousness. To test the thesis of this paper, improve bone health worldwide, and decrease false accusations of abuse, let us support the mission of testing and eradicating VDD.

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1). Illusions of unanimity among key decision-makers that cause them to doubt their own misgivings.

2). Unquestioned beliefs that lead group members to ignore potential consequences of the group's actions.

3). Rationalization of potential warning signs that should cause group members to question their beliefs.

4). Stereotyping of contrary viewpoints leading members of the group to reject perspectives that question or challenge the group's ideas.

5). "Mindguards" or members of the group who prevent troubling or contrarian viewpoints from circulating among group members. Rather than sharing important information, they may keep quiet or prevent other members from sharing.

6). Illusions of invulnerability lead group members to engage in unjustified risky behaviors with an overly optimistic hope of success.

7). Direct Pressure may silence group members who tend to pose inconvenient questions or raise objections that may be seen as evidence of disloyalty.

8). Collectively, these behaviors may make members of a group be excessively optimistic about their success, ignoring any possible negative outcomes. Members are convinced their cause is right and just, so they ignore any moral quandaries of the group's decisions. The group body tends to ignore the suggestions of anyone outside the group.

Any dissenters are pressured to come around to the consensus. After the pressure is exerted, members censor themselves to prevent further shunning. Once decisions are made, the group assumes them to be unanimous.

Note 88. See Lynoe above 2016.

Note 89. Elinder G[1], Eriksson A [2,3,] Hallberg B[4,,5], Lynøe N[6], Sundgren PM[7,8], Rosén M[9], Engström I[10], Erlandsson BE[11]. Traumatic shaking: The role of the triad in medical investigations of suspected traumatic shaking. Acta Paediatr. 2018 Sep; 107 Suppl 472: 3-23. doi: 10.1111/apa.14473.

Note 90. Lynoe N, et. al*. The role of the triad in medical investigations of suspected traumatic shaking, a systematic review. Swedish Agency for Health Technology Assessment and Assessment of Social services Traumatic shaking. SBU assessment. Report 255e/2016

Note 91. Donohoe M. Evidence-Based Medicine and Shaken Baby Syndrome Part I: Literature Review, 1966–199.Am J Forensic Med Pathol 2003; 24: 239-242.

Note 92. Frasier, L, Rauth-Farle, K, Alexander, R, Parrish, R. Abusive head Trauma, a Medical, Legal and Forensic Reference. Chapter 14 and 15 authored by Sirotnik called "Medical Disorders that Mimic Abusive Head Trauma" and G. W. Medical Publishing 2006.

Note 93. See Lynoe 2016 above.

Note 94. Goodman S, Richard Royall R. Evidence and Scientific Research. Amer J of Pub Health. December 1988, Vol. 78, No. 12.

Note 95. Gray JD, Christy A. Cutler CA, Janet G. Dean JG, Kempe CH. Prediction and prevention of child abuse and neglect. Child Abuse Negl. 1977: 1(1): 45-58.

Note 96. Tuerkheimer, Deborah, Flawed Convictions: 'Shaken Baby Syndrome' and the Inertia of Injustice: Introduction (March 2, 2014). Oxford University Press, April 2014, Forthcoming , Available at SSRN: https://ssrn.com/abstract=2403499

Note 94. https://www.ncbi.nlm.nih.gov/books/NBK499891/

Note 95. Gray JD, Christy A. Cutler CA, Janet G. Dean JG, Kempe CH. Prediction and prevention of child abuse and neglect. Child Abuse Negl. 1977: 1(1): 45-58.

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Note 97. https://www.ncbi.nlm.nih.gov/books/NBK499891/

Note 98. Goodman S, Richard Royall R. Evidence and Scientific Research. Amer J of Pub Health. December 1988, Vol. 78, No. 12.

Note 99. Matshes, E. Retinal and Optic Nerve Sheath Hemorrhages Are Not Pathognomonic of Abusive Head Injury. Proceeding of the American Academy of Forensic Sciences; 2010 Feb 24; Seattle, WA.

Note 100. Chadwick, DL, et. al. Deaths from Falls in Children: How Far is Fatal? Journal of Trauma-Injury Infection & Critical Care. 1991 31(10): 1353-1355

Note 101. Holick MF. Vitamin D Deficiency. N Engl J Med 2007; 357: 266-81.