Overrepresentation of Males in Traumatic Brain Injury of Infancy and in Infants With Macrocephaly

Further Evidence That Questions the Existence of Shaken Baby Syndrome

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Abstract: Shaken baby syndrome (SBS) has been thought to be caused by violent shaking of an infant and is characterized by the triad of findings: subdural hematoma (SDH), retinal hemorrhages, and neurologic abnormalities. The triad is not specific for SBS and can be seen in accidental trauma and in certain medical conditions. Recent observations, however, question whether SBS exists. Herein, we review the gender differences in 3 groups of infants with traumatic brain injury: (1) neonates with SDH from birth trauma, (2) infants with SDH from accidental trauma, and (3) infants with SDH from SBS. Gender differences are also presented in a fourth group of infants with macrocephaly related to increased extra-axial fluid spaces (IEAFS). Compared with the expected male frequency of 51.4% in newborns, there was a statistically significant overrepresentation of males in each of the 4 groups–65.3%, 62.2%, 62.6%, and 68.8%, respectively. We believe that the most likely explanation for these findings relates to the larger head size of the male compared with the female which has several relevant consequences. First, the larger head circumference of a male newborn compared with a female newborn may increase the likelihood that a male newborn will incur a small SDH from the minor trauma of the birthing process that can later rebleed and present with a symptomatic SDH that could be misdiagnosed as SBS and child abuse. Second, a short fall would have a greater likelihood of causing a SDH in a male infant than a female infant who could subsequently become symptomatic from hours to weeks later and could thus present as an unexplained SDH. Third, infants with macrocephaly related to IEAFS may be at increased risk for developing a SDH from the larger head size and greater tautness of the bridging vessels in the extra-axial fluid spaces. We believe that many infants who have been diagnosed with SBS have been given incorrect diagnoses of child abuse. Rather, their SDH may occur as a result of a small SDH from the birthing process that enlarges during early infancy, a short fall, or from macrocephaly with IEAFS.

Key Words: forensic science, shaken baby syndrome, traumatic brain injury of infancy, increased extra-axial fluid spaces, macrocephaly, subdural hematoma, retinal hemorrhages, male gender

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Traumatic brain injury of infancy (TBII) can be accidental or nonaccidental and is characterized by 3 primary findings: (1) intracranial bleeding, most commonly subdural hematoma (SDH), (2) retinal hemorrhages (RH), and (3) neurologic dysfunction (ND) which can include vomiting, lethargy, decreased level of consciousness, seizures and focal neurologic findings. (These 3 findings–SDH, RH, and ND–will be referred to as the "triad" in this article). The triad can also have a nontraumatic etiology and can be seen in infants with meningitis/encephalitis, cortical vein thrombosis/sagittal sinus thrombosis, bleeding disorders, ruptured vascular malformations, and glutaric aciduria- type 1.[1–4]

The triad associated with trauma can be seen in several different clinical settings. Severe birth trauma can cause the triad and present immediately after birth, and severe accidental trauma such as in a motor vehicle accident or high fall can cause the triad in an infant.[5] Controversy had existed as to whether short falls could cause the triad in an infant, but recent observations clearly indicate that short falls can also cause the triad.[6]

When a young infant presents with the triad and there is no history of trauma to explain the triad and none of the aforementioned conditions is present that might explain the triad, it is likely that the diagnosis of nonaccidental trauma from shaken baby syndrome (SBS) will be made. The triad has been the sine qua non for the SBS.[7,8] However, there is recent evidence that SBS may not even exist and that the triad in these alleged cases of SBS may have a different pathogenesis.[9–13]

Several factors have shown an association with TBII including chronic hypoxia, macrocephaly associated with increased extra-axial fluid spaces (IEAFS), and possibly male gender.[9,14,15] Herein is a formal evaluation of (1) the association of TBII with gender and (2) the association of macrocephaly associated with IEAFS with gender. Our findings show an overrepresentation of males in both groups. A hypothesis to explain these findings is set forth which has implications that raises further doubt about the existence of SBS.

METHODS

Relevant scientific articles of series of infants with TBII published between 1966 and 2005 were found using the keywords "Shaken Baby Syndrome," "Retinal Hemorrhage," and "Subdural Hematoma" using MEDLINE. Articles referenced in these articles and published before 1966 that described relevant series of infants with TBII were also included. Articles were included if they indicated the frequency of males and females with TBII and were classified into 3 TBII groups:

Group 1–neonatal SDH from birth trauma[5,16–21]
Group 2–SDH in young infants from accidental falls[21–32]
Group 3–SDH from nonaccidental TBII (SBS).[21,22,27–30,33–51]

The same type of literature search was also done to determine if there are any gender differences in macrocephaly related to IEAFS (also called external hydrocephalus, benign subdural collections of infancy, benign subdural hygromas, and other terms) which is group 4.[52–72]

If there was no association of gender with TBII or with macrocephaly, then the expected percentage of males would be about 51.4% which is the percentage of births that are male in North America and Europe over the second half of the 20th century.[73] We used a one sample Z test of a single proportion to test whether the percentage of males in each group was significantly different than the expected percentage of 51.4%.[74]

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TABLE 1. Neonatal SDH From Birth Trauma (Group 1)

	No. Cases	M (%)	F (%)	Time	Location	First Author	Reference
1	42	27 (64)	15 (36)	1958–1962	Columbus, OH	Natelson	5
2	41	32 (78)	9 (22)	1971–1986	Kiev, Soviet Union	Romodanov	16
3	25	13 (52)	12 (48)	1963–1982	Nagoya, Japan	Takagi	17
4	17	11 (65)	6 (35)	1980–1983	Paris, France	Pierre-Kahn	18
5	15	9 (60)	6 (40)	1986–1995	Toronto, Canada	Perrin	19
6	7	6 (86)	1 (14)	1950s	Hartford, CT	Schipke	20
7	26	15 (58)	11 (42)	1998–1999	British Isles	Hobbs	21
Total	173	113 (65.3)	60 (34.7)				

TABLE 2. SDH in Infants From Accidental Trauma (Group 2)

	No. Cases	M (%)	F (%)	Time	Location	Mean Age (mo)	Range (mo)	First Author	Reference
1	7	3 (57)	4 (43)	1998–1999	Great Britain	9	NA	Hobbs	21
2	8	4 (50)	4 (50)	1990–2000	Taiwan	6.8	3.0–11	Loh	22
3	26	23 (88)	3 (12)	1972–1983	Tokyo, Japan	8.1	3.0–11	Aoki	23
4	16	10 (63)	6 (37)	1989–1997	Korea	5.4	1.0–9	Hwang	24
5	14	10 (71)	4 (29)	1970–1990	London	7.4	1.0–18	Howard	25
6	15	12 (80)	3 (20)	1975–1985	Tokai, Japan	A	NA	Ikeda	26
7	5	4 (80)	1 (20)	1995–1998	Hong Kong	10	1.0–24.0	Fung	27
8	15	11 (73)	4 (27)	1987–1996	Australia	12.4	2.0–23	Tzoiumi	28
9	109	60 (55)	49 (45)	1991–2001	Milwaukee, WI	11.7	NA	Wells	29
10	23	13 (57)	10 (43)	1988–1998	New Zealand	14	NA	Kelly	30
11	6	4 (67)	2 (33)	1970s	New York	8.5	3.0–12	Sparacio	31
12	18	9 (50)	9 (50)	1985–2001	France	5.8	1.1–21.4	Vinchon	32
Total	262	163 (62.2)	99 (37.8)						

A indicates 12/15 infants less than 12 mo of age; NA, not available.

RESULTS

The results of the literature search for the 3 different groups of TBII are shown in Tables 1 to 3 and for macrocephaly in Table 4. For all 4 groups there was a greater frequency of males than females beyond the expected 51.4% that reached statistical significance at $P < 0.001$. Table 5 summarizes the gender data in each of the 4 groups.

DISCUSSION

The results of this study show that for the 3 specific types of TBII and for macrocephaly associated with IEAFS there is an overrepresentation of males compared with females in each group. There are several possible explanations for this gender difference in the 3 groups of TBII.

The first explanation is related to possible gender differences in behavior such as in curiosity or danger seeking behaviors. However, this would not explain the increased frequency of males over females in neonatal SDH from birth trauma. Moreover, most of the infants in the series of SDH from accidental falls are under 1 year of age, a time when danger seeking behaviors are unlikely to be present. The possibility of differences in how parents might react to a male versus female, such as in infant crying, could lead to gender differences in SBS, but again the available evidence suggests otherwise.[75] Thus, the explanation of behavior differences to explain the gender difference in TBII seems unlikely.

A second possible explanation is that X-linked recessive disorders that predispose to SDH could explain this gender difference. Hemophilia and Menke disease are 2 such disorders. The frequency of hemophilia (hemophilia A and hemophilia B) in the general population is about 1/5000, and only about 4% of individuals with hemophilia have an intracranial bleed.[76,77] Since there is a positive family history of hemophilia in about two-third of cases of hemophilia and since most infants with TBII have an evaluation for a coagulopathy, it is highly likely that the specific diagnosis of hemophilia would be made as the cause of the TBII. Menke disease is likewise quite rare (1/250,000), and there are other clinical features that are usually seen in Menke disease that would suggest this specific diagnosis.[78] Thus, the explanation of unappreciated cases of hemophilia and Menke disease as the basis of the male excess in TBII is unlikely.

A third possible explanation relates to the larger head size of males than females that is present at birth and persists thereafter. Table 6 shows the mean head circumference or Occipital-Frontal Circumference (OFC) in males versus females during the first year of life. At birth the mean OFC for males is 1.1 cm greater than that of females.[79] At 12 months of age the mean OFC for males is 1.3 cm greater than that of females. Over the first year of life the mean OFC for males is about 3% greater than that of females. The larger size of the male head at birth increases the potential for birth-related injuries that may be clinically apparent immediately after birth and for birth-related injuries that may only become apparent and clinically important later in life.

The implication of this gender difference in head size is 3-fold. First, the greater mean OFC for a male than a female at birth indicates that for a given pregnant mother who gives birth, there would be greater forces on the head of a male than a female.

TABLE 3. SDH From Inflicted Head Trauma (SBS) (Group 3)

	No. Cases	M (%)	F (%)	Time	Location	Mean Age (mo)	Range (mo)	First Author	Reference
1	54	31 (57)	23 (43)	1986–1991	Cleveland, OH	8.4	1.0–48	Reece	33
2	173	95 (55)	78 (45)	1990–1995	Denver, CO	8	1.0–35	Jenny	34
3	39	26 (67)	13 (33)	2001–2003	Bali, Indonesia	NA	0.25–5	Golden	35
4	20	14 (70)	6 (30)	1977–1982	Philadelphia, PA	5.8	1.0–15	Ludwig	36
5	38	25 (66)	13 (34)	1995–1997	Cleveland, OH	5.5*	1.0–120 (A)	Dashti	37
6	16	11 (68)	5 (31)	1970s	Philadelphia, PA	9	3.0–15 (B)	Zimmerman	38
7	10	6 (60)	4 (40)	1994–1998	Hong Kong	6.5	2.0–13	Lee	39
8	364	204 (56)	160 (44)	1988–1998	Canada	4.6*	NA	King	40
9	48	31 (65)	17 (35)	1978–1985	Philadelphia, PA	7.9	1.0–24	Duhaime	41
10	186	133 (72)	53 (28)	1996–2001	Paris, France	4.6	1.0–29	Pierre-Kahn	42
11	14	8 (57)	6 (43)	1991–1994	Los Angeles, CA	12	2.0–54	Gilles	43
12	21	17 (81)	4 (19)	1987–1996	Australia	5.3	1.0–20	Tzoiumi	28
13	148	90 (61)	58 (39)	1991–2001	Milwaukee, WI	8	NA	Wells	29
14	4	4 (100)	0 (0)	1995–1998	Hong Kong	6	3.0–9	Fung	27
15	39	25 (64)	14 (36)	1995–1998	Seattle, WA	8.7	NA	Feldman	44
16	41	24 (59)	17 (41)	1988–1998	New Zealand	7.7	NA	Kelly	30
17	13	10 (77)	3 (23)	1995–2001	Hong Kong	5.8	1.0–14.5 (B)	Sun	45
18	81	53 (65)	28 (35)	1981–2001	USA (3 centers)	3.5	0.5–20	Starling	46
19	11	7 (64)	4 (36)	1990–2000	Taiwan	5.2	2.0–12	Loh	22
20	106	74 (70)	32 (30)	1998–1999	Great Britain	4	NA	Hobbs	21
21	10	7 (70)	3 (30)	1997–2002	France	3.3	0.6–5.4 (C)	Vinchon	47
22	47	30 (64)	17 (36)	1997–2002	Rhode Island	5.2	0–34	Tung	48
23	33	24 (73)	9 (27)	1992–1997	Buffalo, NY	6.7	1.0–26	Dias	49
24	75	46 (61)	29 (39)	1993–1999	Toronto	10.6	2.0–48	Morad	50
25	18	12 (67)	6 (33)	1989–2000	Toronto	13.3	1.0–36	Wygnanski-Jaffe	51
Total	1609	1007 (62.6)	602 (37.4)						

*Median age.
A indicates 32 of 38 <24 months; B, only infants under 15 months considered; C, only infants under 12 months considered; NA, not available.

Tentorial tears leading to a SDH are related to excessive pressure on the skull during the delivery process.[80] Thus, there would be a greater chance that a male would incur a severe SDH immediately after birth that was clinically apparent, and our findings in neonatal SDH (Table 1) confirm this.

Second, the birth process could result in a small, subclinical SDH that could either be resorbed or could rebleed later in the first year of life. In the former, the small, subclinical SDH would never have been known to have existed, whereas in the latter situation the enlarging chronic SDH could lead to the triad and be called SBS. Small, acute SDHs can spontaneously rebleed over time or rebleed with minimal impact forces to the head and become chronic SDHs.[81–87] As they enlarge and become chronic SDHs, they can reach a critical size and then become clinically apparent with the classic signs of the triad. Whitby et al reported that a prospective head magnetic resonance imaging study within 48 hours of birth of 111 normal term babies showed that 9 (8.1%) had a SDH.[88] Looney et al reported that in normal infants delivered vaginally and studied between 1 and 5 weeks of age with head magnetic resonance imaging, there was a 26% incidence of intracranial hemorrhage.[89] Thus, the scenario of a small, asymptomatic SDH enlarging and then becoming clinically significant without any apparent explanation is plausible–with males more likely than females because of their larger head size.

Third, if one assumes that the brain has a sphere-like shape, then a comparison can be made of the mean brain weight of males versus females during the first year of life. A 3% greater mean OFC during the first year of life for males compared with females translates into an approximately 9% greater mean brain weight for males compared with females during the first year of life (volume of a sphere = $4/3\ \pi r^3$). Brain weights of males and females at autopsy from infants who died under 1 year of age confirm this gender difference.[90–92] Table 7 shows the autopsy weights of male infants versus female infants from the Schultz study.[91] Thus, if a male and female of the same age were to have an identical, accidental fall, then the male would have a greater chance of incurring a brain injury because of his greater brain mass (force = mass × gravity). This mechanism would be especially relevant for short falls. The pioneering work of Ommaya et al related to brain size and brain injury showed that there is a brain injury threshold–the greater the brain mass, the lower the threshold for developing brain injury. Thus, according to the Ommaya model of scaling, the same force applied to a male brain and to a female brain of the same age (male brain would be slightly greater in mass than the female brain) would cause brain injury in the male before the female.[93] Our findings in accidental TBII (Table 2) showed a greater frequency of males than females.

This hypothesis of gender differences in brain size explaining, in part, the gender difference in TBII is graphically shown in Figure 1. We believe that this explanation is plausible and likely accounts for most of the excess of male infants with TBII.

A fourth possible contributing factor to the greater frequency of males than females in TBII relates to the entity macrocephaly associated with IEAFS. In this condition there is increased OFC as

TABLE 4. Macrocephaly From IEFS (Group 4)

	No. Cases	M (%)	F (%)	Time	Location	Mean Age (mo)	Range (mo)	First Author	Reference
1	74	50 (68)	24 (32)	NA	Switzerland	NA	NA	Laubscher	52
2	9	6 (67)	3 (33)	1981–1983	Portland OR	6.1	3.0–10.0	Nickel	53
3	47	35 (74)	12 (26)	1990–1999	Birmingham, England	6*	1.0–24.0	Tolias	54
4	31	22 (71)	9 (29)	1993–1998	Leuven Belgium	6.7	NA	Van Calenbergh	55
5	15	11 (73)	4 (27)	NA	Scotland	NA	NA	Day	56
6	15	11 (73)	4 (27)	NA	Cardiff Wales	NA	NA	Cole	57
7	33	26 (79)	7 (21)	1993–1995	Izmir Turkey	6.8	1.5–21.0	Ersahin	58
8	19	10 (53)	9 (47)	NA	Belgium	7.7	NA	Wilms	59
9	36	19 (53)	17 (47)	1975–1983	Bronx, New York	NA	NA	Alvarez	60
10	6	4 (67)	2 (33)	1975–1977	Madison WI	5	3.0–8.0	Robertson	61
11	16	9 (56)	7 (44)	1991–1992	Tokyo Japan	5.4	2.0–10.0	Aoki	62
12	15	12 (75)	3 (25)	1985–1989	Qatar	11	3.5–30.0	Odita	63
13	13	9 (69)	4 (31)	1983–1985	Oklahoma City, OK	6.7	2.0–11.0	Hamza	64
14	88	59 (67)	29 (33)	1994–1997	Cincinnati, OH	8*	NA	Medina	65
15	20	15 (75)	5 (25)	1970s	Kyoto Japan	4.9	2.0–8.0	Mori	66
16	15	11 (73)	4 (27)	1981–1985	Cincinnati, OH	4.2	2.0–6.5	Carolan	67
17	16	13 (81)	3 (19)	1993–1995	Oman	7.9	NA	Roshan	68
18	5	4 (80)	1 (20)	1998–2001	Alberta, Canada	4.4	2.0–8.0	Zouros	69
19	7	5 (71)	2 (29)	1984–1986	Taiwan	7.3	6.0–9.0	Shen	70
20	25	16 (64)	9 (36)	1998–2002	Korea	6*	2.0–120.0	Cho	71
21	7	5 (71)	2 (29)	1998–2004	Montreal	7.4	3.6–17.8	McNeely	72
Total	512	352 (68.8)	160 (31.2)						

*Median.
NA indicates not available.

TABLE 5. Summary of Gender Differences in the Four Groups

Group*	Total	Males†	Females	*P*
1	173	113 (65.3%)	60	<0.001
2	262	163 (62.2%)	99	<0.001
3	1609	1007 (62.6%)	602	<0.001
4	512	352 (68.8%)	160	<0.001

*Group 1–Neonatal SDH from birth trauma.
Group 2–SDH from accidental falls.
Group 3–SDH from inflicted TBBI (shaken baby syndrome).
Group 4–Increased extra-axial fluid spaces (IEAFS).
†Compared to expected male percentage of 51.4%.[73]

TABLE 6. Mean OFC in Males Versus Females From Birth to 12 Months

	Mean OFC (cm)	
Age (mo)	Female	Male
Birth	34.71	35.81
3	39.92	41.21
6	42.40	43.72
9	43.94	45.27
12	45.04	46.35

2000 CDC growth charts–www.cdc.gov/growthcharts.

TABLE 7. Mean Brain Weight in Males Versus Females From Birth to 12 Months

	Males		Females	
Age (mo)	No. Cases	Brain Weight (gm ± 1 SD)	No. Cases	Brain Weight (gm ± 1 SD)
1	56	469 ± 47	28	433 ± 59
2	53	506 ± 67	39	490 ± 51
3	43	567 ± 81	36	525 ± 89
4	42	620 ± 71	29	595 ± 80
5	40	746 ± 91	24	725 ± 62
6	47	762 ± 73	23	730 ± 85
7	27	767 ± 32	21	750 ± 92
8	27	774 ± 95	24	770 ± 96
9	25	820 ± 49	15	810 ± 82
10	20	850 ± 96	14	830 ± 117
11	16	875 ± 89	18	875 ± 64
12	19	954 ± 35	15	886 ± 64

Schulz DM, Giordano DA, Schulz DH. Weights of organs of fetuses and infants. *Arch Pathol.* 1962;74:244–250.[91]
SD indicates standard deviation.

a result of increased cerebrospinal fluid (CSF) volume that resides in the extra-axial fluid spaces. The etiology of this situation is thought to be a transient imbalance in the production and resorption of CSF with a net relative increase of CSF relative to the normal situation. The initial reports of this entity used terms that indicated the increased CSF resided in the subdural space. Recent analysis of this entity with more precise imaging studies and analysis indicates that in most of these cases the increased CSF is in the subarachnoid space, but enlarged subdural spaces can also exist in which the space can be filled with CSF, blood, or proteinaceous material.[59,62] In both

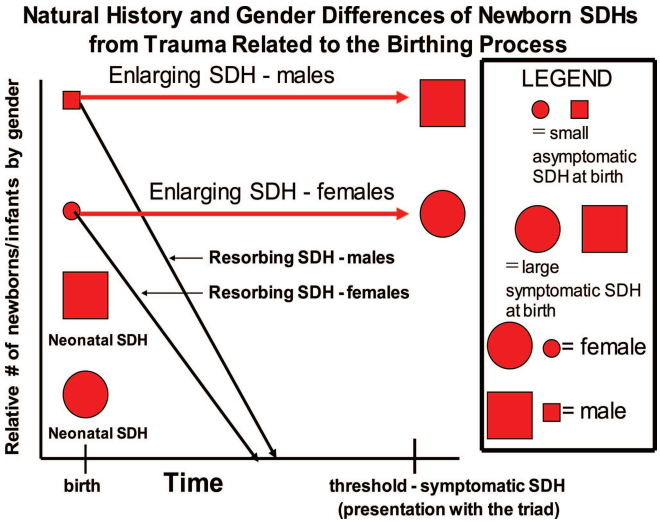

FIGURE 1. Shows the relative increase of SDH in males compared with females in both the newborn period and in early infancy. It is hypothesized that this gender difference is a consequence of the larger OFC of males compared with females. This OFC difference would predict a greater incidence of symptomatic, neonatal SDH in males compared with females as a result of trauma from the birthing process. In addition, there would be a greater number of males compared with females who have a small, asymptomatic SDH at birth as a result of trauma from the birthing process. While most of these small, asymptomatic SDHs from birth trauma will eventually resorb, a very small proportion will rebleed and enlarge during early infancy and eventually become symptomatic. These enlarging SDHs from rebleeding can arise spontaneously or from minimal force impacts of the head and may be incorrectly diagnosed as SBS. full color online

situations there is macrocephaly and thus increased head weight. In some families autosomal dominant inheritance has been described with a male preponderance in these families.[57,94]

The same mechanical analysis of what happens when a larger than normal head hits a surface (ie, an infant falls or hits their head against a hard object) that was previously discussed likewise applies to the infant with macrocephaly from IEAFS. Moreover, there is another relevant consequence of having excessive fluid in the extra-axial fluid spaces that predisposes to SDH. In infants with IEAFS the bridging veins in the subdural and subarachnoid spaces are more taut and thus probably more likely to tear with forces that might not otherwise cause a tear. This finding has been previously reported and emphasized in the literature.[72,95–97]

Thus, there are 2 important differences in the infant with macrocephaly associated with IEAFS that likely increase the risk for a SDH, and thus the triad–increased head weight and more taut bridging vessels. Our review of the literature of macrocephaly related to IEAFS shows a significantly increased frequency of males versus females as indicated in Table 4. Thus, in this group of infants with IEAFS who are at increased risk to incur the triad compared with infants with normal head size and normal extra-axial fluid spaces, there is significantly more males than females. We believe this entity also contributes to the increased frequency of males with TBII.

Implications of This Hypothesis to the Existence of SBS

Several recent observations have converged to raise serious questions about the existence of SBS and whether shaking alone can cause the triad. These following 5 observations have been noted:

1. Biomechanical Analysis

When a biomechanical evaluation is done of the forces that cause the triad in experimental animals and extrapolated to the human, it appears that the forces generated by shaking are insufficient alone to cause the triad. It has been suggested that impact is needed.[6,11–13,93,98,99] Moreover, one would expect severe neck trauma with cervical spine dislocations in infants who are violently shaken, and they are conspicuously absent in SBS.

2. Lack of Witnessed Reports

Despite this diagnosis being in the literature for over 30 years and professed to the general public as a real entity, there have been few credible, witnessed reports of violent shaking causing SBS. When Leestma evaluated the same SBS literature for an accounting of "candidate" cases of shaking causing the "triad," he only found 11 cases in over 30 years of case reports. Eight of these survived and only 3 were autopsied. These were reported not to show impact injury to the head. Thus, the number of possibly valid cases of injuries caused by shaking is very few.[100]

3. Nonspecificity of RHs for Child Abuse

For the past 30 years the finding of RHs has been a finding that almost always led to a diagnosis of SBS, and thus child abuse. The recent, independent observations of Lantz and of Lueder now show that RHs can be seen in accidental injuries of young infants and seriously undermines this long held dogma.[10,101,102]

It has been thought that shaking causes the RHs in SBS. However, there is evidence that RHs are unlikely to be caused by shaking and have a different cause. The individual components of the triad are probably not independent events. Rather, following a traumatic brain injury, the SDH is the primary event and the ND and RHs are secondary to the brain swelling and injury from the SDH. The RHs arise from increased intracranial pressure that exceeds the pressure in the retinal veins.[103–105] SDHs have to be of sufficient size to cause ND and RHs.

4. Problematic Confessions

Confessions in SBS cases are fraught with difficulties, and distinguishing between a true confession that represents real wrongdoing and one that is either coerced or represents innocent shaking related to resuscitating the infant is often difficult.[106–110] Confessions can also be encouraged and tinted, misrepresented, or may occur from alleged perpetrators who may mistakenly believe what they think they did (shaking) caused the injuries in the child, and out of guilt may confess to an act they actually did not do. These coerced confessions have been part of the foundation of the SBS literature that have misled the scientific community to believe that shaking alone can cause the triad.[111]

5. A Flawed SBS Literature

When a careful meta-analysis of the SBS literature was performed by Donohoe, he found the scientific foundation of SBS to be lacking.[112] A child abuse literature related to inflicted TBII has been generated over the past 30 years based on (1) questionable confessions, (2) criteria such as RHs which are not diagnostic of SBS, (3) a false premise that shaking alone can cause the triad, (4) rejection of the idea that short falls can cause the triad, and (5) rejection of the idea that infants with IEAFS are at risk for the triad with minimal head trauma. Thus, it is increasingly apparent that inflicted TBII from shaking is controversial with emerging evidence that it may not even exist.

How could such a diagnosis based on such flimsy evidence and with such far-reaching implications become so entrenched in pediatric and legal medicine? The original reports of shaking as the

mechanism for the triad arose from the anecdotal reports of Caffey and Guthkelch in the early 1970s.[113,114] This work has never been substantiated by any sound scientific investigations. SBS became a legitimate diagnosis in the 1970s because it was not possible to identify a unique and specific cause of the triad based on the available information on the pathogenesis of the triad at that time. In such infants there was usually no evidence of external trauma (no skull fractures and no bruising), so the idea that shaking might cause the triad, based on the anecdotal reports of Caffey and Guthkelch, was appealing and embraced by the medical community. Infants who presented with the triad were diagnosed with SBS, and very little additional evaluation was performed to look for alternative explanations. The diagnosis of SBS thus became a default diagnosis, but defended as an irrefutable dogma. It was based solely on the finding of the triad–not for lack of other evidence, but for lack of looking for other evidence. Interestingly, law enforcement also readily accepted the triad as being pathognomonic of child abuse and their interrogations of alleged perpetrators have been conducted with the idea that someone clearly shook the infant. Such interrogations are biased to finding shaking and coerced and false confessions have resulted. Moreover, a literature began to develop on SBS, usually diagnosed only on the finding of the triad, and this massive literature, albeit much of it unsound, convinced many in the medical community that SBS was a real entity.

However, the fallacy of the triad being pathognomonic of SBS is now apparent. A number of relatively uncommon pediatric disorders have now been described that can be associated with the triad including glutaric aciduria-type 1, vitamin K deficiency, Menke disease, hemophagocytic lymphohistiocytosis, and others.[3,4,78,115,116] These conditions are mimics of SBS, and unfortunately wrongful diagnoses of child abuse in these cases have been made under the SBS rubric. Clearly, pediatricians faced with an infant with the triad or pathologists faced with an autopsy of an infant with the triad may not have been aware that these rare disorders can mimic SBS because the association had not yet been recognized. Or they were just not aware of these mimics of SBS and may not have done the appropriate tests to evaluate for these mimics of SBS. Thus, an insidious selection process has been operating over the last 3 decades that has been biased to making the diagnosis of SBS whenever an infant presented with the triad, as other possible explanations were not known at the time or were not considered in the differential diagnosis of the triad.

If shaking was not the cause of the triad in these cases that have been previously called child abuse, then what was the underlying basis of the triad in the majority of these cases? The findings of the present study are relevant to this issue and cast further doubt about the existence of shaken baby syndrome. There is a biologic explanation for the excess of males with TBII compared with females that gives us an insight into how many of these cases of TBII occur, some of which have previously been called child abuse or SBS. Small, asymptomatic SDHs from the normal trauma of the birth process can spontaneously rebleed or rebleed with minimal forces, enlarge, and then present with clinical symptoms and the triad in the first year of life. Infants with macrocephaly associated with IEAFS can incur SDHs and present with the triad with minimal physical forces. Both of these situations mimic child abuse, and we believe many such infants in the past have been mistakenly diagnosed as victims of child abuse, when they were likely not. In these 2 situations there is no history of severe trauma immediately prior to the presentation with the triad, so it is understandable why a superficial analysis of the situation could lead to a diagnosis of child abuse. It is the subtle nature of the rebleeding SDH from birth trauma and the infant with IEAFS that have fooled us into believing this is necessarily child abuse.

When plausible, alternative explanations of causes of the classic triad have been presented in the past, the child abuse protection community (CAPC) has offered an unyielding defense of SBS without due consideration of these other explanations. In the 1980s the Japanese reported that short falls could cause the triad.[23] The CAPC responded by saying the Japanese were actually describing missed cases of child abuse. Other investigators have presented recent compelling evidence that reconfirms the Japanese observations and discounts the CAPC position that short falls cannot cause the triad.[6,25,117] Short falls can cause the triad, and a biomechanical analysis of short falls shows that they produce far greater forces on the brain than shaking.[12,13,93,98,99]

A second important cause of the triad that has been dismissed by the CAPC as not relevant is birth trauma. It has been known for decades that birth trauma from a difficult delivery can cause a neonatal SDH with RHs and ND. As noted previously, a SDH from birth trauma can sometimes be small and without clinical features at birth.[88,89] Most of these eventually resorb without problems; however, it is likely that some of these small SDHs can spontaneously rebleed, get larger, and then some months later cause clinical symptoms. Such an occurrence will likely be called SBS by the CAPC, because the triad will be present, and there is no immediate history of severe trauma. The birth history is typically not considered in the analysis.

In conclusion, the present study noted an increase of males compared with females in series of infants who were diagnosed with SBS. If SBS does not exist, then some other explanation for the cause of the triad must be posited. The findings of the present study suggest such explanations. We believe that some infants diagnosed with SBS are infants who incurred small, asymptomatic SDHs that later enlarged during the first year of life and some are infants with macrocephaly with IEFS who are at increased risk to incur a SDH with minimal trauma. Some of these infants with the triad may have been abused, but shaking alone is unlikely the cause. Short falls can cause the triad in young infants, and this also contributes to the increased frequency of males as a result of their larger heads. We believe that these explanations are, in part, the basis for the greater frequency of males compared with females with TBII.

ADDENDUM

Recent observations since the acceptance of this article in 2007 suggest that the SDH associated with the triad in young infants may not always be caused by torn bridging veins, but can also be caused by damage to the intradural venous plexuses (a, b, c, d, e). Intradural hemorrhage can disrupt the dural border cell layer, leading to a "thin film" SDH. Bleeding from the thin-walled intradural venous plexus is an anatomically more plausible cause of the small volume SDH typically seen in the triad than a ruptured bridging vein, and there are likely multiple causes of such dural bleeding. The dura appears to be sensitive to hypoxia, and the contribution of hypoxia to the development of the triad needs further exploration. The mechanism of hypoxia-mediated intradural hemorrhage causing the triad does not alter the conclusions of this article, as hypoxic events are also more likely to occur in males than in females based on differences in brain size, as discussed in the present article.

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