Analysis of causes that led to the bleedings in the subdural spaces and other tissues in baby Alan Ream Yurko's case

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Abstract

Careful review of the medical evidence in baby Alan Ream Yurko's case clearly shows that death was the result of adverse reactions to vaccines and medications administered by healthcare professionals instead of Shaken Baby Syndrome on the part of the parent as initially determined. The tissue bleedings were caused by the baby's treatment with heparin following an episode of respiratory/cardiac arrest. The medical examiner and other physicians who evaluated this case failed to consider heparin's ability to cause bleeding in tissues and also overlooked the role of the adverse reactions to vaccines in the baby's health problems. The report presents descriptions of the clinical data and other medical literature explaining the pathogenesis of the baby's illness that support the above conclusions.

Keywords: heparin, adverse vaccine reactions, vaccine-induced death, Shaken Baby Syndrome (SBS), subdural bleeding

Introduction

In November of 1997, Alan R. Yurko was arrested for abusing his son, a two-and-a-half-month-old baby named Alan Ream Yurko, by vigorous shaking of the head. The baby died while Mr. Yurko was in jail; he was convicted of murder by a jury in 1999 and sentenced for life plus ten years in prison. I reviewed the medical evidence in this case and determined that baby Alan died as a result of adverse reactions to vaccines and medications [1, 2]. The medical examiner and other physicians who evaluated this case overlooked the role of heparin in tissue bleeding, even though there were several biomarkers indicating that the bleedings resulted from high doses of heparin.

By way of comparison, in a second case that I evaluated, heparin was also not considered as the cause of the bleeding. A 15-month-old toddler died as a result of adverse reactions to vaccines and medications, and she was treated with heparin prior to harvesting her organs for donation. Heparin caused bleeding under the skin and in other tissues, and her caretaker was falsely accused of killing her by shaking [2, 3]. Thus the Yurko case is not an isolated one.

Heparin has been widely used in hospitals as an anticoagulant and has been given to patients to prevent the formation of blood clots. Also, prior to harvesting organs for donation, heparin has been injected in large doses into the blood of children (and adults) who are considered brain dead and have no chance of survival. In their investigations, treating physicians and medical examiners must consider, prior to accusing caretakers of killing children by shaking, the amount of heparin given in the hospital before and after brain death.

In this report, I present detailed descriptions of the following: (1) heparin doses given to baby Alan in the hospital prior to and during organ harvesting; (2) biomarkers of adverse reactions to heparin observed in baby Alan's case; and (3) predisposing factors that enhanced both heparin toxicity in baby Alan's case and bleeding in tissues.

I hope that the state of Florida will take into consideration the medical facts described in this report and my other reports on this case [1, 2] that clearly show baby Alan died as a result of adverse reactions to vaccines and medications, and the bleedings caused by heparin. Alan Yurko is innocent and should be released from prison as quickly as possible. Furthermore, I hope that the information presented in this report will help physicians and medical examiners in their evaluations of similar cases.

1. Heparin doses given to baby Alan following respiratory/cardiac arrest and prior to harvesting the organs

Heparin is a heterogeneous group of straight-chain anionic mucopolysaccharides called glycosaminoglycans, having anticoagulant properties [4]. It inhibits reactions that lead to the clotting of blood and the formation of fibrin clots, both in vitro and in vivo, and it acts upon multiple sites in the normal coagulation cascade. Clotting time is prolonged by full therapeutic doses of heparin in most cases.

Depending on circumstances, hemorrhage can occur at virtually any site in patients receiving heparin. Patients suffering from anemia, any unexplained symptoms, and/or having low blood pressure are at the greatest risk of having serious hemorrhagic events after receiving a therapeutic dose of heparin. Heparin is contraindicated in disease states where there is increased risk of hemorrhage, or, especially, where it has already occurred.

In addition to serious bleeding, heparin has been found to induce the formation of white clot due to the aggregation of platelets, and to reduce the platelet count due to consumption.

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Reduction in platelet counts are also observed in patients treated with heparin as a result of immune reactions.

Baby Alan was given heparin as continuous infusion into the arterial and venous systems, and as venous flushes, from November 24th through 28th, 1997 (Tables 1 and 2). He was also given a megadose of heparin (22,950 IU) after his brain death, prior to and during organ harvesting, to prevent the formation of blood clots in organs.

The earliest time recorded for the heparin infusion into Alan's blood was 14:45 on November 24, 1997, which was five hours and five minutes prior to the time of the first computerized tomography (CT) brain scan. The baby was given heparin at the rate of 2 ml per hour and the stated dilution ratio was one part heparin and one part normal saline. The biomarkers of heparin toxicity observed in this case indicate that a high dose of heparin was infused on November 24th.

Based upon the biomarkers, the following assumption is made. The minimum concentration for the injectable heparin stock solution reported by the Physician's Desk Reference (PDR) is 1000 units of heparin per ml [4]. Assuming a stock solution of heparin (1,000 units/ml) and a dilution factor of 2, the infusion rate of 2 ml per hour would result in heparin infusion rate of 1000 units per hour. Baby Alan's weight was 4.57 kg. The estimated effective heparin dose in baby Alan's case was 219 IU/kg per hour (or 2 ml/hr x 500 IU/ml/4.57 kg).

The PDR recommends 50 units/kg IV as initial dose for infants and children, and a maintenance dose of 100 unit/kg (IV drip) every four hours, or 25 unit/kg per hour [4, p. 3306]. Based on the assumed dose of heparin infused into the baby (219 IU/kg per hour), the estimated total dose of heparin infused in five hours was 1095 IU/kg, which is about 8.8 times the recommended maintenance dose for infants of 125 IU/kg per five hours [4]. The CT scan of the brain taken at 1950 on 24 November (at five hours and five minutes following the start of heparin infusion) showed a right subdural hematoma and intraparenchymal hemorrhage in the frontal region of the right cerebral hemisphere.

Unfortunately, following his initial heparin treatment and CT scan on November 24th, the baby was again treated with heparin on November 25th through the 28th, despite his bleeding problems and a significant reduction (30.5%) in platelet count (Tables 1, 2, 3, 4).

On November 27, 1997, approximately 75 hours after initial hospital admission, baby Alan was pronounced brain dead. Prior to autopsy on 29 November, Translife (a company specializing in donor organ removal and transport) took the baby's heart, liver, pancreas, and a portion of the intestine for organ transplant. Prior to and during the harvesting of these organs, baby Alan was given 22,950 IU of intravenous heparin to prevent the formation of blood clots in the organs. This amount of heparin is capable of keeping 1000 ml of blood liquid at room temperature. The estimated whole-blood volume in baby Alan's case is about 320-366 ml, which is 7-8% of his body weight of 4.57 kg.

The biomarkers of heparin toxicity observed in baby Alan's case indicate that heparin was the primary cause of the bleedings observed in the subdural spaces of the brain and spinal cord and other tissues, and they support my conclusion that baby Alan was given a high dose. The descriptions of these biomarkers and the supporting data are presented in Section 2 below.

Table 1. Times and rates of heparin infused into baby Alan's blood circulation

Date	Time heparin started[a]	Dilution ratio	Rate (ml/hr)
11/24/97	14:45	1:1	2
11/25/97	08:00	1:1	2
11/26/97	08:00	1:1	2
	20:00	1:1	2
	24:00	1:1	2
11/27/97	08:00	1:1	2
	18:50	1:1	2
11/28/97	08:00	1:1	2
	20:00	1:1	2

[a]Heparin was given via arterial or venous line.

Table 2. Amount of heparin used to flush the venous line in baby Alan's case

Date	Time	Heparin received (Units)[a]
11/26/97	20:30	30
11/27/97	09:30	40
	21:30	40
11/28/97	08:00	40

[a]Total amount of heparin flush given between 11/25/97 at 0800 and 11/28/97 at 0800 was 310 units. Heparin was given via central or peripheral venous line.

2. Biomarkers of the adverse reactions observed in baby Alan's case

There are several biomarkers observed in this case indicating that the bleedings in tissues were caused by the high doses of heparin given to the baby following his hospitalization on November 24, 2004. These include the following: (a) significant reductions in red blood cell count, hemoglobin level, and hematocrit value following heparin treatment; (b) significant reductions in platelet count, and elevation in fibronigen split product following the heparin; (c) the age and the distribution of the bleeding and the blood clots observed in the subdura; (d) the occurrence and age of bleedings in lungs, skin, and spinal cord.

2.1 Treatment with heparin caused significant reductions in the red blood cell count, hemoglobin level, and hematocrit value

The infusion of heparin into the circulatory system of baby Alan was started at 14:45 on November 24th. At 15:15 the red

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blood cell count, hemoglobin, and the hematocrit % were reduced by 18 to 24% as shown in Table 3. These data indicate that a major bleeding event occurred between 12:09 and 15:15 on November 24th and that the likely cause of the bleeding was the treatment with a high dose of heparin.

Table 3. Percentages of reduction in red blood cell count, hemoglobin and hematocrit values observed following the initial treatment of baby Alan with heparin

Measurement Type	Values at 12:09[a]	Values at 15:15	% Reduction observed
Red blood cell count	2.61×10^6/μL	2.14×10^6/μL	18
Hemoglobin	7.8 g/dL	6.3 g/dL	19
Hematocrit	25.3%	19.3%	24

[a]Normal range: RBC: 4.16-5.7 x 10^6/μL; Hemoglobin: 12.1-17.3 g/dL; Hematocrit: 36.5-52%.

2.2 Significant heparin-induced reduction in platelet count

Baby Alan's platelet count at 12:09 on November 24th was 571,000/μL (143% of the upper normal value). The high platelet count resulted from bone marrow hyperplasia in response to baby Alan's severe anemia. At 12:09, Alan's red-blood-cell, hemoglobin, and hematocrit values were at 57%, 48%, and 53% of normal, respectively. Following the beginning of heparin infusion, the platelet counts were reduced by 3.2% and 30.5% at 0.5 hour and 15 hours, respectively (Table 4). Furthermore, blood analysis performed at about 30 minutes post-heparin-infusion showed increased fibrinogen split product level of 160 μg/ml and prothrombin time of 14.6 seconds. These values are 1600% and 115% of normal, respectively. These data clearly indicate that the bleeding observed in the subdural space of the brain at 19:50 on November 24th was caused by the heparin treatment.

Table 4. Percentages of reduction in the platelet count observed following the treatment of baby Alan with heparin

Date	Time	Platelet count (PC) x 1000 μL	Reduction in PC as % of initial value[a]
11/24	15:15	553	3.2
11/25	05:45	397	30.5
	16:45	398	30.3
11/26	05:45	411	28.0
11/27	05:53	335	41.3

[a]Initial platelet count at 12:09 on November 24th (prior to the administration of heparin) was 571,000/μL. Normal range: 150,000 to 400,000/μL.

The significant reduction in the platelet count observed in this case following the administration of heparin could be explained by the aggregation of the platelets and the formation of clot. Heparin has been known to increase the tendency of the platelets to aggregate and form white clot [4]. Reduction in platelet count has also been reported to occur as a result of immune reactions to heparin. Baby Alan was exposed to heparin during the first week following his birth, as described in the next Section 2.2.1 below. It is possible that he developed antibodies against heparin-platelet complex, and his second exposure to heparin following his respiratory/cardiac arrest led to immunologic reaction and the formation of clot.

2.2.1 Doses of heparin given to baby Alan during the first week following his birth

Baby Alan was born five weeks premature on September 16, 1997 at Florida Hospital in East Orlando. His weight was 5 lb, 9 oz (or 2.52 Kg), and his head circumference was 31.3 cm (12.3 inches). He was premature because his mother suffered from gestational diabetes and oligohydramnios. Labor was induced by pitocin. Approximately two hours after his birth, Alan was noted to have a blood sugar of 32 with grunting respirations. He was diagnosed with Respiratory Distress Syndrome (RDS) and sepsis. He was transferred to Florida Hospital Orlando for admission to the Neonatal Intensive Care Unit (NICU)) [1, 5].

In the NICU, the baby was incubated and placed on a ventilator. He was treated with antibiotics (ampicillin and gentamicin) and a surfactant replacement therapy (Survanta) for his premature lungs. He received heparin via his IV fluid (0.5 unit/ml) for five days (October 16-20, 1997). He also received heparin in the parenteral nutrition for three days at a concentration of 1 unit per ml. These doses of heparin did not lead to reduction in Alan's platelet count (Table 5) as was observed following his treatment with heparin on November 24th (Table 4).

Table 5. Baby Alan's hematology values during the first week following his birth

Date & Time	Red blood cell count x 10^6 μL	Hemo-globin (g/dL)	Hematocrit (%)	Platelet count x 1000/μL
09/16/97 15:20	5.16	18.4	55.1	406
09/17/97 06:20	4.90	17.4	51.8	371
09/18/97 05?40	4.62	16.6	48.5	364
09/22/97 06:30	4.59	16.2	47.4	471
Reference Range	4.76-6.95	18.0-26.5	42-60	250-450

2.2.2 Heparin-induced thrombocytopenia

The occurrence of heparin-induced thrombocytopenia (HIT), a serious allergic drug reaction following any exposure to heparin has been widely observed in children and adults. The frequency of HIT is thought to range from 1 to 5% of patients receiving heparin**.** Patients with HIT have an extremely high frequency of developing immune-mediated thrombosis. Mortality associated with HIT approaches 35% [6-23].

HIT is characterized by antibody-induced activation of platelets, leading to thrombin generation. Antibody formation against heparin complexed to platelet factor 4 (PF4) is central to the pathogenesis of HIT. The antibodies (IgG, IgM, and IgA isotypes) can be measured easily by an ELISA that contains a

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complex of heparin:PF4. These antibodies recognize a multimolecular complex of heparin and PF4, resulting in platelet activation via platelet Fc receptors. Heparin:PF4 antibodies promote platelet activation and aggregation as well as excess thrombin generation, which may lead to clinical thrombosis [10, 13, 14, 15, 18, 20, 21, 22].

Many patients with HIT develop thrombosis, even when heparin treatment is stopped. Because of "isolated HIT" detected during routine platelet-count monitoring, 25-50% of patients subsequently develop symptomatic thrombosis [8]. Diagnosis of HIT is based upon clinical findings that can be confirmed with laboratory assay. Thus, when there is clinical suspicion of HIT, all forms of heparin therapy should be immediately discontinued [6, 18, 20-23]. HIT should be suspected in patients who develop thrombocytopenia with or without associated arterial or venous thrombosis while on heparin. The direct thrombin inhibitors lepirudin and argatroban are currently available and approved for use in patients with HIT [10, 14-23].

Baby Alan was treated with heparin during the first week after birth as described in Section 2.2.1 above. His first exposure to heparin did not cause thrombocytopenia (Table 5). However, it probably led to the development of antibodies against heparin-platelet complex, which caused significant reduction in the platelet count (30.5%) upon re-exposure to heparin on November 24th (Table 4). In some cases, it takes about five to eight days from beginning heparin treatment to observe significant reduction in platelet count [23]. However, in this case the platelet count was reduced by 156,000/μL (30.5% of initial value) in just fifteen hours following the beginning of heparin treatment on November 24th (Table 4). These data indicate that baby Alan suffered from a severe immune reaction to heparin and that he was exposed to high doses of heparin.

The studies cited above clearly state that immune reaction to heparin is very common and can cause death. They also state that physicians have the responsibility to monitor closely their patients treated with heparin to check for immune reaction to it. A significant reduction in the platelet count following heparin treatment is considered an important indicator of the occurrence of immune reaction and should be followed by cessation of treatment.

Despite the reduction platelet count by 30.5% in fifteen hours following the initiation of heparin, the treating physicians continued heparin treatment for three more days (Table 1). These physicians also overlooked the subdural bleeding on the CT scan on November 24th. Heparin should not be given to any patient suffering a bleeding episode.

2.3 The distribution and age of the bleedings and the blood clots observed in the subdural space of the brain.

Dr. Shashi B. Gore performed the autopsy on baby Alan at 10:15 on November 29, 1997 [1]. He stated that subdural hemorrhage was seen prominently on the right cerebral hemisphere and that this hemorrhage was in liquid as well as in clotted form. According to Dr. Gore, there was also subdural hemorrhage on the left cerebral hemisphere posteriorly, and this hemorrhage was relatively less prominent as compared to the right. The dura mater of the cortex of the cerebral hemispheres showed thickened and slightly clotted blood adherent to the dura mater. At places, the thickness of this clotted material was between 2-3 mm.

Dr. Gary Steven Pearl, a state expert witness, examined the blood clot microscopically and observed the proliferation of fibroblasts in layers. Based on this observation, he estimated the age of the oldest portion of the subdural hematoma to be two to five days.

I also examined the H & E stained tissue section of the meninges and observed the proliferation of fibroblasts in the blood clot in the subdural space and in the clot attached to the dura matter. I also observed fresh blood in the subdural space [1].

The gross and microscopic description of the nature of the blood clots and bleedings described above indicate that the blood was released from the blood vessels in a continuous fashion during the five days prior to autopsy, or more precisely, in three stages. The thickened clotted blood that adhered to the dura mater represents the first stage of blood release, the clotted blood represents the second stage, and the blood in the liquid form represents the third stage which is the most recent. My conclusions are supported by the findings of the CT of brain scan taken at 19:50 on November 24th. It showed the subdural hematoma present only on the right side of the brain and no bleeding was seen on the left. This means that the bleeding on the left side occurred after 19:50 on November 24th.

These facts contradict Dr. Gore's conclusion that the hemorrhage occurred in minutes, or even in a few seconds, due to vigorous shaking of the head. By his own admission, Dr. Gore did not review baby Alan's medical record and overlooked the facts that he was treated with high doses of heparin on November 24th through the 29th as described in Section 1 above. The significant reduction in the platelet count (41.3%) observed on November 27th (Table 4) supports the fact that the bleedings in the subdura occurred in the hospital.

2.4 Distribution and the age of bleedings observed in the lungs and the subdural space of the spinal cord

On November 29th Dr. Gore examined the lungs grossly and found that both lungs were congested and contained irregular areas of hemorrhagic appearance. Serial cutting sections of both lungs showed irregular areas of hemorrhages. He also examined the H & E stained tissue sections of the lungs microscopically and observed the presence of red blood cells. This indicates that the bleeding was fresh and less than 24 hours old.

Dr. Douglas Shanklin also examined the H & E stained tissue sections of the lungs and observed multifocal areas of fresh hemorrhage.

Dr. Gore observed bleeding in the subdural space of the lower thoracic, lumbar, and sacral regions of the spinal cord. Dr. Shanklin also observed a fresh hemorrhage (6-12 hours old) in the subdural space of the spinal cord. I also examined the H & E stained tissue section of the spinal cord and found a fresh hemorrhage in the subdural space. The age of the bleeding was less than 24 hours.

Dr. Pearl indicated that there was a spinal cord injury, that blood vessels were swollen and nerve cells damaged. Dr. Gore

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and other physicians examined the entire vertebral column of the baby and did not find any injury caused by trauma. These observations indicate that the bleeding occurring in the subdural space resulted from damage in the blood vessels due to hypoxia and from the treatment with excessive doses of heparin.

3. Predisposing factors that enhanced the toxicity of heparin in baby Alan's case

Baby Alan suffered from apnea and cardiac arrest on November 24, 1997 and was treated with heparin as described in Sections 1 and 2. There are several factors observed in baby Alan's case that enhanced the adverse reaction of heparin in causing bleeding. These factors include the following: hypotension, anemia, metabolic problem, systemic injuries, gastric ulcer, bacterial infection, and the treatment with high doses of sodium bicarbonate [1]. Below are descriptions of these factors.

3.1 Anemia

Baby Alan's blood analysis at 12:09 on November 24th showed that he was suffering from severe anemia. In Table 6, hematology values on November 24th following his respiratory/cardiac arrest are compared with those on September 22, 1997 at one week of age. On November 24th his red blood cell count, hemoglobin level, and hematocrit value were reduced by 43%, 52%, and 47% of the values observed on September 22nd, respectively. These significant reductions were caused by severe anemia and were not caused by acute bleeding on November 24th because Alan's platelet count at 12:09 was elevated, 571,000/μL (143% of upper normal). High platelet count usually results from bone marrow hyperplasia in response to anemia, infections, and bone marrow damage. Also, his average initial serum creatinine value on November 24th was 0.5 mg/dL (83% of low normal value) which indicates low muscle mass.

Table 6. Percentages of reduction in red blood cell count, hemoglobin and hematocrit values observed in baby Alan's case on November 24, 1997

Measurement Type[a]	Values on 09/22/97	Values on 11/24/97	% Reduction observed
Red blood cell count (RBC)	4.59×10^6/μL	2.61×10^6/μL	43
Hemoglobin	16.2 g/dL	7.8 g/dL	52
Hematocrit	47.4%	25.3%	47

[a]Normal range: RBC: 4.16-5.7 x 10^6/μL; Hemoglobin: 12.1-17.3 g/dL; Hematocrit: 36.5-52%.

3.2 Metabolic problems, organ damage, and gastric ulcer

Baby Alan's blood analysis following his respiratory/cardiac arrest revealed that he was suffering from diabetes mellitus and complications of diabetes, organ damage, and gastric ulcer [1]. These conditions enhance bleedings in individuals treated with heparin [1, 4]. Baby Alan's serum glucose levels at 12:09 and 15:15 on November 24th were 337 and 397 mg/dL, respectively. Normal serum glucose rage is 70-110 mg/dL. His blood pH was 7.18 at 12:09 PM and dropped to 7.1 at 14:40 and his anion gap was elevated (22 mEq/L).

Dehydration, polyurea, weight loss, and wasting are symptoms and complications of diabetes mellitus. In the first twenty-four hours, baby Alan's total fluid intake was 725.8 ml and his total output was 786 ml. The baby's net output was 60.2 ml. It indicates that he was dehydrated [1]. The baby was treated with antidiuretic hormone (DDAVP) on November 28th to prevent dehydration. DDAVP is a synthetic analog of the natural pituitary hormone 8-arginine vasopressin (ADH), an antidiuretic hormone affecting renal conservation.

On November 24th the baby's weight was 10.05 pounds 4.56 Kg); on November 29th his weight was 9.0 pounds (4.08 Kg). He lost 1.05 lb (or 0.476 Kg, 10% of his weight) in five days during his stay in the hospital despite treatment with relatively high volumes of IV fluid and antidiuretic hormone. Also, his average serum creatinine value on November 24th was 0.45 mg/dL (75% of low normal value) and dropped to 0.2 mg/dL (33% of low normal) on November 27th. Low creatinine is an indicator of low muscle mass and wasting disease [1].

His serum potassium level was 4.9 mEq/L at 12:09 and dropped to 2.3 mEq/L at 0545 on November 25th following his treatment with excessive amount of sodium bicarbonate (blood pH was 7.6-7.7). His hypokalemia was severe. He was treated with potassium solutions by IV infusion several times on 24-25 November 24th to 25th [1].

Furthermore, baby Alan had elevated LDH (1148% of normal), alkaline phosphatase (202% of normal), and SGOT (414% of normal), which indicated damage in the liver and heart. At the time of admission to Princeton Hospital, the baby had a gastric ulcer. The treating physician stated that the child developed bleeding from the gastrostomy tube due to a stress ulcer. The child was treated with cimetidine (histamine H2-receptor antagonist) in the hospital for his ulcer [1]. Heparin should not be given to a baby with a bleeding ulcer.

3.3 Bacterial infections

Baby Alan suffered from a bacterial infection on November 24, 1997 as indicated by his elevated white blood cells, his body temperature and his responses to the treatment with antibiotics. His white blood cell count was 20, 900/μL (174% of upper normal count) and his temperature was 105.8ºF (41.0ºC) at 18:00. Chest x-rays taken on November 24th showed lung infiltrate, which is a sign of lung infection.

The treatment of baby Alan with high therapeutic doses of three types of antibiotics IV on November 24th resulted in significant reductions in white blood cell count, serum glucose, liver enzymes, and anion gap levels. These antibiotics included the following: 20 mg gentamicin (recommended dose 7.5 mg/kg/day); 300 mg rocephin (recommended dose 50-75 mg/kg/day); and 222 mg Claforan (recommended dose 50-180 mg/kg/day). The white blood cell count was reduced by 62% in three hours following the beginning of treatment with antibiotic.

Furthermore, on November 26th serum glucose was reduced by 64% from the level on November 24th. Also low values for

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the following biomarkers were observed on November 26th: LDH, 733 IU/L (reduced by 70%); alkaline phosphatase, 135 IU/L (reduced by 47%); SGOT, 167 IU/L (reduced by 19%); and anion gap 11 mEq/L (50% reduction) [1].

3.4 Treatment with excessive doses of sodium bicarbonate

Baby Alan suffered from metabolic acidosis, as indicated by low blood pH (7.1), low blood bicarbonate level (17.9 mEq/L), and elevated anion gap (22 mEq/L). In diabetic patients, the metabolic acidosis and anion gap are almost totally accounted for by the elevated plasma levels of acetoacetate and beta-hydroxybutyrate, although other acids (e.g., lactate, free fatty acids, phosphates) contribute [23].

Baby Alan was treated with sodium bicarbonate to correct his blood acidosis. However, he was given an excessive amount of bicarbonate (Table 7). His blood pH was 7.1 at 14:40 on November 24, 1997 and rose to 7.67 at 23:00. In addition, he was given bicarbonate by IV infusion at 08:00 on November 25th, when he had a high blood pH. Baby Alan's blood pH stayed elevated (7.55 to 7.70) for at least 13.5 hours. The rate of bicarbonate IV infusion was 5 mEq/hr (Table 7).

Table 7. Baby Alan's blood pH values and bicarbonate dosages

Date	Time	Treatment with HCO_3	pH[a]
11/24/97	12:23		7.18
	14:40		7.10
	14:45	5 mEq given IV bolus + Starting IV infusion (5 mEq/hr) until 08:00	
	15:40		7.19
	17:39		7.25
	20:30		7.39
	22:05		7.36
	23:00		7.67
11/25/97	00:15		7.70
	01:30		7.55
	03:40		7.61
	08:00	5 mEq given IV bolus + Starting IV infusion (5 mEq/hr) until 11:00	
	10:50		7.61
	12:40		7.55
	17:15		7.45

[a]Normal pH range 7:35-7.45

Bicarbonate therapy may be indicated in severely acidotic patients (pH 7.0 or below), especially if hypotension is present (acidosis itself can cause vascular collapse). Bicarbonate is not used routinely in less acutely ill subjects, because rapid alkalinization may have detrimental effects on oxygen uptake in tissues [23, p. 2073]. Alkalinization increases the avidity of hemoglobin to bind oxygen, impairing the release of oxygen in peripheral tissues [1, 23].

The hemoglobin-oxygen dissociation curve is normal in diabetic ketoacidosis because of the opposing effects of acidosis and deficiency of red blood cell 2,3-bisphosphoglycerate (2,3-BPG). If acidosis is rapidly reversed, the deficiency of 2,3-BPG becomes manifest, increasing the avidity with which hemoglobin binds oxygen. If bicarbonate is given, the infusion should be stopped when the pH reaches 7.2 to minimize possible detrimental side effects and to prevent metabolic alkalosis as circulating ketones are metabolized to bicarbonate with reversal of ketoacidosis. The key parameters to follow are the pH and the calculated anion gap [1, 23].

4. Conclusions and recommendations

Baby Alan suffered from serious illnesses such as anemia, bacterial infections, diabetes mellitus and other metabolic problems that led to his respiratory/cardiac arrest on November 24, 1997. In the hospital, the baby was treated with heparin, which caused bleeding in tissues. He was also treated with high therapeutic doses of sodium bicarbonate, which caused hypokalemia, hypoxia, and brain edema. Baby Alan's blood pH stayed elevated (7.55 to 7.70) for at least 13.5 hours.

Baby Alan's treating physicians overlooked the following facts: (a) he suffered from a bleeding gastric ulcer at the time of admission to the hospital on November 24th (treatment with heparin was contraindicated); (b) bleeding in the subdural space of the brain at 19:50 on November 24th following his treatment with heparin (treatment with heparin after this point was dangerous and medically unjustifiable); (c) significant adverse reactions to heparin at 15 hours following the beginning of heparin infusion as shown by the significant reduction (30.5%) in the platelet count (treatment with heparin should have been discontinued immediately).

The bleedings in the subdural spaces of the brain and spinal cord and other tissues of baby Alan were caused by heparin given in the hospital on November 24th through 28th, and prior to, and during, harvesting his organs for donations. The distribution of the bleedings in baby Alan's tissues, the age of the bleedings, and the gross and microscopic compositions of the blood clots in the subdural space of the brain indicate that the bleedings occurred in at least three stages in a span of 2-5 days prior to autopsy. These clinical data contradict the medical examiner's theory that the bleedings in the subdural space of the brain occurred in a few minutes on November 24, 1997.

Furthermore, the medical examiner and the treating physicians did not take the time to review the medical evidence in this case and their conclusions that baby Alan died as a result of vigorous shaking were based on a theory and not on medical facts. They did not take into consideration that the adverse reactions to vaccines and medications given to baby Alan prior to and after his respiratory/cardiac arrest caused the baby's health problems and subdural bleedings.

I have also found that baby Alan's case is not an isolated one. A similar situation exists in three other cases of American children that I have recently evaluated. These children died as a result of adverse reactions to vaccines and medications, and their innocent caretakers were falsely accused of killing them.

I believe that the shaken baby syndrome (SBS) theory has misled physicians for more than thirty years to believe that bleedings in the subdural space of the brain and the retinas of the eyes are pathognomic signs for shaking a child. This error has prevented physicians and medical examiners from conducting complete and valid investigations to learn about the factual causes of bleedings in the subdura and the retinas in

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children, and thus from taking the proper measures to correct the problems.

The SBS theory is not supported by science and medical facts, and it has caused enormous tragedies by putting innocent people in prison. It is also costing society a large sum of money in legal fees and unnecessary trials. I urge our healthcare system, governments, and the society to re-evaluate the SBS theory to save lives and vital resources.

References

[1] Al-Bayati MA, Analysis of causes that led to baby Alan Ream Yurko's cardiac arrest and death in November of 1997. *Medical Veritas*, 2004 Nov/Dec; 1(2):201–31. [www.medicalveritas.com]

[2] Al-Bayati MA. Shaken baby syndrome or medical malpractice? *Medical Veritas,* 2004 Apr/May; 1(1):117–129. [www.medicalveritas.com]

[3] Al-Bayati, MA. Analysis of causes that led to Toddler Alexa Shearer's cardiac arrest and death in November 1999. *Medical Veritas*, 2004 Apr/May; 1(1):86–116. [www.medicalveritas.com]

[4] *Physicians' Desk Reference,* Edition 53, 1999. Medical Economics Company, Inc, Montvale, NJ, USA.

[5] Alan Ream Yurko's medical records from Florida Hospital, September 16–21, 1997.

[6] Spinler SA, Dager W. Overview of heparin-induced thrombocytopenia. *Am J Health Syst Pharm*, 2003; 60(Suppl 5):S5–11.

[7] Greinacher A. Treatment options for heparin-induced thrombocytopenia. *Am J Health Syst Pharm*, 2003; 60(Suppl 5):S12–8.

[8] Warkentin TE. Management of heparin-induced thrombocytopenia: a critical comparison of lepirudin and argatroban. *Thromb Res*, 2003; 110(2-3):73–82.

[9] Jeske WP, Walenga JM. Antithrombotic drugs for the treatment of heparin-induced thrombocytopenia. *Curr Opin Investig Drugs*, 2002; 3(8):1171–80.

[10] Deitcher SR, Carman TL. Heparin-induced thrombocytopenia: natural history, diagnosis, and management. *Vasc Med*, 2001; 6(2):113–9.

[11] Warkentin TE. Current agents for the treatment of patients with heparin-induced thrombocytopenia. *Curr Opin Pulm Med*, 2002; 5:405–12.

[12] Lewis BE, Walenga JM, Wallis DE. Anticoagulation with Novastan (argatroban) in patients with heparin-induced thrombocytopenia and heparin-induced thrombocytopenia and thrombosis syndrome. *Semin Thromb Hemost* , 1997*;* 23(2):197–202.

[13] Warkentin TE. Heparin-induced thrombocytopenia. Pathogenesis, frequency, avoidance and management. *Drug Saf*, 1997; 17(5):325–41.

[14] Lubenow N, Greinacher A. Drugs for the prevention and treatment of thrombosis in patients with heparin-induced thrombocytopenia. *Am J Cardiovasc Drugs*, 2001; 1(6):429–43.

[15] Alving BM, Krishnamurti C. Recognition and management of heparin-induced thrombocytopenia (HIT) and thrombosis. *Semin Thromb Hemost*, 1997; 23(6):569–74.

[16] Warkentin TE. Limitations of conventional treatment options for heparin-induced thrombocytopenia. *Semin Hematol*, 1998; 35(4 Suppl 5):17–25, discussion 35–6.

[17] Aouizerate P, Guizard M. Management of heparin-induced thrombocytopenia. *Therapie*, 2002 Nov-Dec; 57(6):577–88.

[18] Harenberg J, Jorg I, Fenyvesi T. Heparin-induced thrombocytopenia: pathophysiology and new treatment options. *Pathophysiol Haemost Thromb, 2002;*32(5-6):289–94.

[19] Kelton JG. The clinical management of heparin-induced thrombocytopenia. *Semin Hematol*, 1999*;* 36(1 Suppl 1):17–21.

[19] DeBois WJ, Liu J, Lee LY, Girardi LN, Mack C, Tortolani A, Krieger KH, Isom OW. Diagnosis and treatment of heparin-induced thrombocytopenia. *Perfusion*, 2003; 18(1):47–53.

[20] Haas S, Walenga JM, Jeske WP, Fareed J. Heparin-induced thrombocytopenia: clinical considerations of alternative anticoagulation with various glycosaminoglycans and thrombin inhibitors. *Clin Appl Thromb Hemost*, 1999; 5(1):52–9.

[21] Warkentin TE. Heparin-induced thrombocytopenia: a ten-year retrospective. *Annu Rev Med*, 1999; 50:129–47.

[22] Warkentin TE. Clinical presentation of heparin-induced thrombocytopenia. *Semin Hematol*, 1998; 35(4 Suppl 5):9–16, discussion 35–6.

[23] *Harrison's Principles of Internal Medicine,* 14th edition. Editors: Fauci AS, Braunwald E, Isselbacher KJ, Wilson JD, Martin JB, Kasper DL, Hauser SL, Longo DL. McGraw-Hill, New York, 1998.

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