

Robert G. Richards,¹ M.D.; Richard I. Fukumoto,¹ M.D.;
and Darrell O. Clardy,¹ M.S.

Sudden Infant Death Syndrome: A Biochemical Profile of Postmortem Vitreous Humor

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ABSTRACT: Postmortem chemical analysis of vitreous humor from Sudden Infant Death Syndrome (SIDS) infants was done. The results were compared to a pediatric control group. The concentrations of potassium, calcium, phosphorus, glutamic oxaloacetic transaminase, glutamic pyruvate transaminase, creatinine phosphokinase, and lactate dehydrogenase were all significantly different from the pediatric control group. Thus it appears that SIDS infants are a different population from the pediatric control group.

KEYWORDS: pathology and biology, sudden infant death syndrome, chemical analysis

Postmortem chemical analysis of vitreous humor has been reported to be a useful diagnostic aid [1-5]. Similarly, the electrolytes from the vitreous humor of infants has been discussed as it relates to the Sudden Death Syndrome (SIDS) [6-11]. Potassium has been used in the estimation of the postmortem interval [12-19] and recently vitreous drug levels have proven reliable even in embalmed bodies [20-28].

Coe [1], who has written extensively on postmortem chemistry, states that the eye is an isolated well-protected organ which is much less subject to contamination or putrefactive change than either blood or cerebrospinal fluid. In Coe's article [29] on vitreous chemistry, the data are arranged in hourly sequence. The change in vitreous chemistry was used in correlating factors relating to death.

Sturner and Susa [30] have reported that SIDS victims have significantly lower phosphoenolpyruvatecarboxykinase (PEPCK) activity than non-SIDS victims. However, they could not substantiate the impaired gluconeogenesis terminal hypoglycemic hypothesis as being the cause of SIDS. Sturner [10] reported a study of 22 infants with undetermined causes of death in which the constituents assayed included osmolality, sodium, chloride, urea, glucose, and calcium. There were six cases of abnormal electrolytes noted.

In another report by Swift [31], there was good correlation between antemortem serum and postmortem vitreous levels, especially sodium and urea.

Blumenfeld [11] also reported on the chemical constituents of a SIDS postmortem vitreous humor, consisting of sodium, potassium, chloride, magnesium, calcium, urea, and creatinine. He concluded that there was nothing within the eight constituents that set the SIDS

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¹Pathologist, pathologist, and criminalist, respectively, Forensic Science Services, Office of the Sheriff-Coroner, Santa Ana, CA.

vitreous humor apart from those of infants dying of other known causes or that they gave any information pertaining to the etiology of SIDS.

We undertook a study of the vitreous chemistries of infants diagnosed as SIDS to determine if their vitreous chemistry was different from non-SIDS infants.

Materials and Methods

Since 1977, approximately 2100 postmortem vitreous specimens have been collected by the Orange County Sheriff-Coroner Office from routine autopsies. The specimens were collected with a 10-mL syringe and an 18-gauge needle from the lateral canthus of both eyes, combined and refrigerated until chemical analysis could be performed. The analysis was done on the Technicon Simultaneous Multiple Autoanalyzer Computer (SMAC), which provides twenty chemical analyses and four calculations. These constituents are: alkaline phosphatase (alk, ptase), lactate dehydrogenase (LDH), glutamic oxaloacetic transaminase (GOT), creatinine phosphokinase (CPK), glutamic pyruvate transaminase (GPT), total protein (TP), albumin (ALB), total bilirubin (T. BIL), calcium, phosphorus, sodium, potassium, chloride, carbon dioxide (CO₂), urea, uric acid, creatinine, cholesterol, triglycerides, and glucose.

Each of the significant constituents was segregated into hourly groups. For each hour a "normal" population, mean, and standard deviation was determined. Any value beyond two standard deviations from the mean was considered abnormal. Through the years, there were consistently elevated abnormal values related to SIDS. This SIDS profile consists of alk, LDH, CPK, GOT, calcium phosphorus, and potassium (Table 1).

The data from the SMAC analyses was stored on a PDP 11-43 computer and from the computer 59 cases of SIDS were collected and also 57 cases from zero to two years of age were collected as a comparative pediatric control group (Table 2).

SIDS Group

In general, the SIDS group represents the characteristic sudden unexpected crib death, "death at the scene," ranging from one to nine months in age with no significant sex distribution. The racial differences represents the population of Orange County. As a rule, it is axiomatic that all SIDS cases did not reach the hospital and did not receive any therapeutic measures. For the purpose of this study, the definition of SIDS is considered an exclusion of all definite disease entities leaving only cases between one to nine months with no gross or microscopic cause of death.

Control Group

There is very little "normal" in any autopsy population, and as a result this control group represents a variety of diagnoses. There are 20 infectious, 17 traumatic, 11 asphyxial, 5 congenital, and 3 cases of newborns with placental abnormalities. Almost all of these cases were hospital treated cases and as such would have some relationship to the chemistries involved. Nevertheless, this control group provides the only readily available reference group of pediatric cases for comparison with the SIDS group. It is somewhat reassuring to note that the normal SIDS and the normal control provide similar means for each chemistry involved, which would indicate that this pediatric reference group most likely does represent a reasonably accurate comparative pediatric population.

This control group was further analyzed by each subgroup (infectious, traumatic, and so forth) and each subgroup compared with the SIDS group. There does not appear to be any pertinent correlation between the subgroups and the SIDS group. That is the SIDS group shows seven out of eight constituents elevated. Alkaline phosphatase and high calcium were

TABLE 1—*SIDS profile.*

Hours	Case	Age, Sex, Race ^a	Alk Ptase	LDH	GOT	CPK	GPT	Calcium	Phos- phorus	Potas- sium
1 to 3	3	2 mo	6	435	82	287	11	7.0	4.2	12.9
1 to 3	10	2½ mo	15	1700+	343	4200+	27	7.3	3.4	10.5
1 to 3	13	m o 6 wk f w	7	932	124	3736	11	6.1	2.4	11.0
4	2	2 mo m w	4	59	20	79	12	6.2	2.1	10.0
4	17	2½ mo f w	20	500	2680	12000	33	6.7	3.7	10.0
4	30	...	7	675	91	966	2	7.1	2.2	13.0
5	3	4 mo f w	12	3136	452	3024	33	7.1	4.0	11.2
5	18	6 wk f w	19	1000+	277	2400+	45	6.9	3.4	10.6
5	22	8 wk f w	6	1820	239	57	...	6.6	2.5	9.2
6	3	3 mo m w	2	423	122	305	13	6.3	2.3	11.4
6	4	4 mo m w	6	...	56	520	20	6.1	3.7	...
6	10	7 wk m w	5	321	64	417	18	7.4	2.0	10.2
6	17	2 mo f w	22	3080	740	10800	125	6.9	3.7	10.0
6	23	2 mo m w	5	1200+	38	537	...	6.5	2.8	12.0
7	9	3 mo f w	19	600	...	27600	25	6.2	2.6	8.4
7	17	2 mo m w	7	927	201	414	23	7.7	3.9	12.9
7	27	6 mo f w	2	158	23	537	4	6.7	2.6	11.0
7	36	3 mo f w	49	4320	892	6496	55	7.6	4.9	12.8
8	9	21 da f w	6	220	82	295	13	7.3	0.8	13.1
8	11	4 mo m w	1990	5551	72	5.8	4.8	13.2
8	15	2½ mo m o	3	420	137	2030	20	6.0	1.5	10.8
8	35	5 mo m w	17	2000	316	640	6	7.3	3.5	12.6
9	25	23 da m w	10	...	289	...	17	6.6	2.8	10.2
9	28	2 wk m w	9	2100	402	4200+	5	5.9	1.9	8.2
9	51	3 mo f w	2	145	33	200	20	6.7	2.8	11.3
9	53	7 wk m w	8	1628	442	7944	32	5.0	3.0	10.6
9	55	10 mo m w	18	3500+	3500+	5020	31	8.0	5.4	16.1
10	15	16 mo m w	6	1224	104	854	14	2.7	3.0	9.8
11	20	4½ mo m w	18	2640	306	1380	37	6.7	3.4	12.0
11	23	3½ mo m w	4	141	29	413	2	7.5	3.6	14.6

TABLE 1—Continued.

Hours	Case	Age, Sex, Race ^a	Alk Ptase	LDH	GOT	CPK	GPT	Calcium	Phos- phorus	Potas- sium
11	31	4 mo f w	27	1000+	254	505	35	6.5	4.2	13.2
11	36	1 mo m w	21	3120	204	10100	13	6.4	3.9	...
11	44	3 mo m w	12	330	94	1754	24	6.5	2.6	11.2
11	46	2 mo f w	8	180	78	564	38	6.7	2.5	11.3
11	52	4 mo m w	8	4000+	1000+	2400+	19	6.8	3.5	15.7
12	28	8 mo m w	15	1500	516	1620	36	7.7	5.2	14.1
12	52	5 mo m w	...	427	57	1468	40	7.2	7.3	20.0
12	53	3 mo m w	5	1113	93	1863	55	7.1	3.5	12.6
13	22	1 mo m w	20	1952	718	10976	24	6.9	3.7	11.2
14	50	5 wk m w	48	2378	305	5400	22	6.5	3.4	14.8
17	21	2 mo f b	1	506	66	998	5	3.6	5.2	18.4
18	33	6 wk f w	50	5250	1024	5130	67	9.7	7.6	18.2
20	3	20 da m w	59	5900	1056	196	66	7.8	6.7	16.0
20	36	4 wk f w	29	1500	487	360	42	8.2	7.7	18.3
20	37	5 mo f w	38	2696	780	11520	9	3.5	2.2	14.2
22	10	10 wk f w	90	1500	606	3600	87	9.3	4.5	15.0
22	12	9 mo f w	6	78	94	81	61	6.2	7.9	10.0
23	27	3 mo m w	4	1248	89	4200	44	3.7	4.8	18.4
24	1	2 mo m w	28	2100	1141	4200	35	9.5	6.3	17.5
24	26	2 mo f w	26	896	307	500	29	3.3	2.7	16.6
25	2	1 mo m w	8	640	90	711	15	2.3	2.9	...
25	10	7 mo f w	4	876	202	6328	13	6.7	2.6	13.6
25	18	2 mo f w	2	145	58	487	12	6.6	4.1	13.2
26	9	2 mo m w	64	...	2095	...	76	6.1	6.9	22.5
28	11	3½ mo f w	12	1168	174	2236	5	7.7	4.4	15.6
32	14	2 mo f w	0	526	112	1508	14	7.2	8.2	23.2
33	7	1 mo f w	20	2519	624	250	13	6.4	4.2	10.6
34	7	11 wk m w	38	1800	232	4936	16	8.2	8.4	10.8
36	5	3 mo f w	21	600	64	975	15	6.8	6.4	10+

^ao = other.

TABLE 2—Control group profile.

Hours	Case	Age, Sex, Race ^a	Alk Ptase	LDH	GOT	CPK	GPT	Calcium	Phos- phorus	Potas- sium
1 to 3	5	18 mo m w	5	1250	52	7270	17	6.2	1.3	6.4
1 to 3	19	11 mo f w	16	500	381	1200	24	5.9	2.2	7.0
1 to 3	23	2 yr f w	4	714	136	3444	33	6.6	1.4	6.9
4	19	17 mo f w	2	21	9	...	1	5.8	1.4	8.3
4	24	19 mo m w	2	...	81	...	19	5.8	1.8	9.2
4	31	2½ yr m w	1	59	14	150	7	7.6	3.6	7.2
4	41	11 mo m w	5	65	28	8	7	6.0	0.9	7.5
5	9	22 mo f w	5	23	24	33	16	6.3	1.8	7.1
6	32	4 mo m w	4	823	29	210	10	...	2.6	12.6
6	18	7 mo m w	13	3000	531	7200	16	6.3	3.6	9.0
8	12	5 mo m w	6	1000	423	2400	33	7.2	3.0	11.2
8	34	5 mo m w	6	1480	311	5200	32	6.7	2.9	11.2
8	46	20 mo m w	20	820	287	3568	38	7.1	1.5	9.6
9	35	1½ da f w	24	1500	216	3600	51	...	3.9	12.9
10	3	10 mo m w	...	500	101	1200	11	6.4	2.4	10.2
10	24	7 da f w	14	1000	650	2400	18	7.0	2.0	10.0
10	30	21 mo m w	10	1750	35	4200	31	6.5	2.9	10.0
10	36	SB f w	...	7200	762	10800	114
11	24	2 yr m w	4	1000	352	2400	29	6.8	3.0	10.0
13	49	1 da f w	29	2560	225	3390	5	9.0	2.8	10.0
14	60	4 mo m w	17	3184	1032	18784	37	5.8	3.6	10.6
14	48	18 mo m w	0	114	8	13	5	7.3	3.3	11.6
14	34	1 yr m w	10	2225	...	1200	12	7.6	4.2	12.1
15	22	7 mo m w	5	455	113	280	31	8.0	4.6	15.3
15	77	1 mo m w	30	1800	201	3600	57	11.1
16	25	newborn f w	43	1200	100	3236	8	7.4	2.8	7.6
17	46	3½ mo f w	14	3816	776	14752	12	6.2	4.2	14.8
18	12	4 mo f w	2	286	56	1054	4	5.9	2.2	11.6
19	16	9 wk m w	3	940	50	493	2	6.5	2.7	12.4

TABLE 2—Continued.

Hours	Case	Age, Sex, Race ^a	Alk Ptase	LDH	GOT	CPK	GPT	Calcium	Phos- phorus	Potas- sium
19	18	2 yr f w	2	278	52	1181	0	6.8	2.1	13.2
19	25	18 mo m w	9	2255	518	8650	149	6.6	2.2	9.7
19	37	1½ yr f w	10	202	223	299	93	6.6	1.6	11.8
19	38	11 mo f w	18	357	42	201	7	7.5	5.0	13.6
19	44	2 mo f w	4	119	50	148	9	6.7	1.5	9.5
20	42	7 mo f w	7	502	139	54	2	6.5	2.2	9.7
20	50	2 yr m w	10	192	59	106	4	6.4	2.0	9.4
21	12	7 mo f w	3	162	98	717	20	7.0	2.2	11.4
21	20	5 mo m w	11	3250	760	15000	37	8.0	3.7	13.6
21	27	3 mo m w	4	1362	286	3600	35	6.1	2.9	11.7
21	36	1 yr m w	7	1464	359	2631	8	6.0	2.3	9.6
21	38	2 yr m w	10	576	105	208	10	6.9	3.0	13.5
21	40	7 mo f w	21	1486	13	6.4	2.8	11.7
22	33	18 mo f w	4	87	68	224	24	7.6	2.8	11.0
22	48	2 yr f w	5	1056	302	5512	8	6.6	4.7	16.8
22	13	8 mo f b	3	560	85	1452	20	6.3	2.4	12.6
23	48	9 mo m w	8	632	127	2704	1	7.1	2.5	12.0
23	17	3 mo f w	20	492	93	952	19	8.2	3.2	9.2
23	26	6 mo m w	3	744	133	3284	16	7.3	2.2	10.8
24	31	3 mo f w	...	1000	560	2400	16.2
24	40	1 yr f w	2	1236	277	1112	...	5.9	1.8	9.3
26	2	3 mo f o	1	23	46	...	18	6.4	1.3	9.1
26	17	16 mo f w	2	53	27	240	7	7.1	1.4	8.6
26	18	10 mo f w	11	600	500	1200	29	6.3	1.7	7.9
28	2	9 mo f w	10	1125	114	3600	7	7.5	5.3	14.4
31	9	10 da f w	44	3950	800	19200	44	7.0	4.0	13.4
34	10	17 mo f w	25	3448	864	223	81	9.2	5.5	18.4
35	6	SB f w	78	1500	828	3600	324	...	16.8	29.4

^ao = other.

excluded from the SIDS profile as they were indistinguishable from the pediatric control group while low calcium was included since there were no low calcium levels in the pediatric control group. Of these subgroups, only the newborn group shows a high alkaline phosphatase. The asphyxial group shows three out of eight elevated constituents. The traumatic, infectious, and congenital groups present four out of eight elevated chemistries while the newborn group has six out of eight elevated factors. No single group appears to parallel the SIDS group. In conclusion, there does not appear to be any chemical relationship between SIDS and any of the subgroups.

Statistical Analysis

In general, statistically speaking, laboratorians are accustomed to considering a biochemical constituent in terms of the 10 000 population with its mean and standard deviation. But when the sample is analyzed, even a large sample over 30, the opposite is true. Hypotheses are made about the population from the sample. Assumptions are made that the mean of the population lies about the sample mean within certain limits (confidence interval). But even with this confidence interval, there is an error factor that must be considered. For example, the 59 cases of SIDS considered here, at a 95% confidence level contains a 37% error, and to reduce this error to 1% would increase the sample size to 80 000. Clearly this goal is unattainable and compromises must be made in sample size and the inherent risk in accepting hypotheses about the sample.

The probability of X represents the occurrence (%) of X in each population [32]. In comparing the validity of the difference between two populations, a two population test is more appropriate than a simpler test since sample size becomes part of the calculations. Thus, the P (%) two-population test and the two-population Z test (mean) would be more appropriate than the P or Z test. The statistical analysis compares the probability (%), mean, and variance of the two populations and without exception the analysis indicates that all three parameters conclusively confirm the validity that the high SIDS group is statistically different from the normal control pediatric group (see Table 3). The high values of the control group need not be considered since they may represent the results of treatment or a disease process. The anova (variance) confirms the P population test and the two-population Z test in all except those constituents where the difference in variance between the two populations was too great to comply with the assumptions of the anova test, that is, the variances must be similar or the same.

Since alkaline phosphatase and high calcium fail to surpass three standard deviations they must be considered as more representative of the pediatric group and discarded from the SIDS profile. Low calcium, even though there are only six cases, becomes statistically significant because there is no comparative normal.

In summary, the statistical analysis of the high SIDS group compared to the normal control pediatric group would indicate that SIDS is not a variant of the pediatric group but a statistically valid separate population.

Discussion

As the statistical analysis indicates, the SIDS group is statistically different from the pediatric population. However the clinical significance of these findings is far from clear. The role of potassium is nebulous. Irrespective of SIDS, potassium represents a very labile component in that it is elevated in so many different entities. Potassium seems to follow most any lead, and with this general background, its significance with SIDS is equally indistinct. There is sufficient potassium in each individual to be lethal. Any slight shift from the intracellular to the extracellular compartment could lead to untoward results.

The role of the elevated enzymes also seems nonspecific, but the presence of so many en-

TABLE 3—Statistical data.^a

	<i>P</i> test	Two-Population Z Test	Z Test	\bar{X}^2
Potassium				
HS-HC	4.27	0.76	0.133	
HS-NC	6.07	9.3	3.04	291
Phosphorus				
HS-HC	3.14	0.78	0.875	
HS-NC	-3.37	10.8	3.64	365
GOT				
HS-HC	1.37	1.95	0.68	
HS-NC	13.3	4.9	46.6	352
GPT				
HS-HC	4.5	-5.67	0.75	
HS-NC	-4.14	2.41	2.68	237
CPK				
HS-HC	2.02	-0.54	-0.10	...
HS-NC	9.8	5.9	44.1	...
Calcium				
HS-HC	0.9	0.36	0.069	...
HS-NC	6.58	11.22	2.8	92
LS-NC	-10.25	-35.0	-6.7	50
LDH				
HS-HC	0.85	1.22	0.27	...
HS-NC	9.3	7.5	30.8	...
Alk Ptase				
HS-HC	0.73	2.4	0.49	...
HS-NC	-0.31	10.0	12.1	...

^aHS = High SIDS, HC = high control, GOT = glutamic oxalacetic transaminase, GPT = glutamic pyruvic transaminase, CPK = creatine phosphokinase, LDH = lactate dehydrogenase, and Alk Ptase = alkaline phosphatase.

TABLE 4—Age, sex, and race of SIDS and control groups.

	SIDS	Control
Age		
<i>n</i>	59	57
Months	1 to 9	1 to 24
Sex		
Male, <i>n</i>	32	29
Male, %	54	51
Female, <i>n</i>	27	28
Female, %	46	49
Race		
White, <i>n</i>	56	55
White, %	95	97
Other, <i>n</i>	2	1
Other, %	3	1.5
Black, <i>n</i>	1	1
Black, %	2	1.5

TABLE 5—Data base for Table 3.^a

	Number	Mean	Standard Deviation	Probability of X	Total No.	H Control P Test	N Control P Test
POTASSIUM							
SIDS							
H	51	13.5	3.3	0.90	56	4.27	6.07
N	5	9.3	0.9	0.10
Control							
H	32	13.0	3.75	0.57	56
N	24	9.1	1.45	0.43
PHOSPHORUS							
SIDS							
H	29	5.01	1.84	0.49	59	3.14	-3.37
N	30	3.03	1.11	0.51
Control							
H	12	4.73	3.2	0.22	54
N	42	2.35	0.73	0.78
GPT							
SIDS							
H	29	45.48	23.29	0.51	57	4.5	-4.14
N	28	11.46	5.91	0.49
Control							
H	8	114.13	91.79	0.15	54
N	46	16.35	11.24	0.85
GOT							
SIDS							
H	54	491.72	679.66	0.93	58	1.37	13.3
N	4	26.25	5.85	0.07
Control							
H	47	304.81	273.75	0.85	55
N	8	21.75	10.08	0.15
CALCIUM							
SIDS							
H	16	7.95	0.82	0.27	59	0.9	6.58
N	37	6.54	0.52	0.63
L	6	3.18	0.56	0.10	10.25
Control							
H	10	7.9	0.72	0.20	51
N	41	6.55	0.50	0.80
CPK							
SIDS							
H	52	3888.73	4895.44	0.91	57	2.02	9.8
N	5	122.6	69.5	0.09
Control							
H	40	4385.00	4873.00	0.77	52
N	12	131.50	85.30	0.23
ALK PTASE							
SIDS							
H	29	28.86	18.76	0.52	56	0.73	0.31
N	27	5.22	2.23	0.48
Control							
H	24	21.17	15.56	0.45	53
N	29	4.07	2.05	0.55

TABLE 5—Continued.

	Number	Mean	Standard Deviation	Probability of X	Total No.	H Control P Test	N Control P Test
LDH							
SIDS							
H	48	1850.00	1774.00	0.86	55	0.85	9.3
N	7	129.00	44.00	0.14
Control							
H	45	1490.62	1318.88	0.80	56
N	11	83.73	57.33	0.20

^aH = high, N = normal, L = low, GPT = glutamic pyruvic transaminase, GOT = glutamic oxalacetic transaminase, CPK = creatine phosphokinase, LDH = lactate dehydrogenase, and Alk Ptas = alkaline phosphatase.

zymes may represent a specific hepatic involvement while the elevated phosphorus and the low calcium might suggest milk intolerance or antibodies. Other immunologic problems have been reported such as the report by Raven [33] who suggested the inability of the infantile immune mechanism to cope with routine immunizations. It is not at all unreasonable to suppose that the transition between a gradually receding passively acquired maternal immunity and the awakening of a rudimentary infantile response may produce a void where minor infections may prove fatal.

Tables 4 and 5 indicate that there are two distinct populations of SIDS cases: those with high vitreous chemistries and those with chemistries consistent with the pediatric control. It appears then that SIDS may not represent a single disease entity, but a group of diseases. However, the high profile is so consistent that in our opinion all the information pertaining to a given case should be reviewed before diagnosing a case as SIDS if the high profile is absent.

It is not possible with this study to attach a clinical significance to these chemistries. It seems too early. However, as Sturner [30] has defined a parameter in PEPCK, the authors hope that additional biochemical constituents may further delineate this entity of SIDS with the hope that the future investigations will provide additional pieces to the puzzle for a clearer understanding of the clinical process.

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Address requests for reprints or additional information to
 Robert G. Richards
 Orange County Sheriff-Coroner
 Forensic Science Services
 P.O. Box 449
 Santa Ana, CA 92702