

TECHNICAL NOTE

Dolores Serrat More,¹ M.D. and Maria Castellano Arroyo,¹ M.D.

Biochemical Changes of the Synovial Liquid of Corpses with Regard to the Cause of Death. 2: Alkaline Phosphatase, Lactic Acid Dehydrogenase (LDH), and Glutamic Oxalacetic Transaminase (GOT)

REFERENCE: Serrat More, D. and Castellano Arroyo, M., "Biochemical Changes of the Synovial Liquid of Corpses with Regard to the Cause of Death. 2: Alkaline Phosphatase, Lactic Acid Dehydrogenase (LDH), and Glutamic Oxalacetic Transaminase (GOT)," *Journal of Forensic Sciences*, JFSCA, Vol. 30, No. 2, April 1985, pp. 547-551.

ABSTRACT: We studied the activity of various enzymes in the synovial liquid of 100 corpses with regard to the cause of death finding that the alkaline phosphatase and glutamic oxalacetic transaminase (GOT) are increased in cranioccephalic trauma, possibly as a result of the important cellular lysis which goes with them; and lactic acid dehydrogenase (LDH) is increased in the pulmonary processes, almost certainly with relation to the great quantity of this enzyme in the lung.

KEYWORDS: pathology and biology, death, synovial liquid

There are many works that relate the enzymatic concentrations in different organic fluids with the data of death and the period of agony. Of these, those carried out by Enticknap [1] are of great interest, as they determine the transaminases (glutamic oxalacetic transaminase [GOT], glutamic pyruvic transaminase [GPT]), lactic dehydrogenase (LDH), acid phosphatase, alkaline phosphatase, and the amylase in the blood; and Schmidt [2] who carried out a similar work obtaining very similar results. From these investigations it is deduced that the GOT and GPT increase progressively until 50 h postmortem and the LDH until 60 h. The alkaline phosphatase increases until 48 h have passed but decreases after this time and the acid phosphatase increases but not much. The amylase is found 48 h postmortem with values three or four times higher than in life. The fact that the results obtained by different authors have coincided makes the interpretations taken from them more valid.

The activity of the esterases (especially cholinesterase) has also been measured by various authors, Edson et al [3], and all of them admit that it is kept constant during a long period of time postmortem, however it can be modified by the time which the agony lasts, its activity increasing in sudden deaths and its average value decreasing in long agonies.

¹Associate professor and professor, respectively, Department of Legal Medicine and Toxicology, Zaragoza University, Zaragoza, Spain.

Sawaguchi and Funao [4] studied the activity of various enzymes and isoenzymes such as malate dehydrogenase (MDH), lactic acid dehydrogenase (LDH), sorbitol dehydrogenase (SDH), succinate dehydrogenase, alpha-hydroxybutyric acid dehydrogenase, aldolase dehydrogenase, alkaline phosphatase, and adenosine triphosphate in blood and tissues (heart and liver) by spectrophotometer, observing that the activity of the same is increased in sperm until 6 h after death.

Klein et al [5] studied the activity of alcohol dehydrogenase (ADH) postmortem in liver finding a decrease of the same; they interpret this as being more influenced by the decrease of temperature than by the time which has passed.

Hinojal et al [6] studied a group of seric enzymes (GOT, GPT, alkaline phosphatase, LDH, cholinesterase, and hydroxybutyric acid dehydrogenase [HBDH]) relating their activity with the cause of death, the agony period, and the time of death. The results obtained show a narrow relation between the rates of GPT, alkaline phosphatase, cholinesterase, and LDH with the time of death. The decrease of the gamma glutamyltransferase is related to the time the period of agony lasts. And they also find higher figures of cholinesterase when the death occurs suddenly and quickly.

Materials and Methods

For our work we have used 100 samples of synovial liquid, coming from corpses of which 63 were men and 37 women, with ages between 14 and 93 years, and with two different origins:

(1) the first group of 53 liquids was obtained from the Forensic Anatomic Institute (Instituto Anatomico Forense) in Zaragoza and

(2) the second group of 47 liquids was obtained from the "Hospital Clínico Universitario" in Zaragoza.

These liquids have been grouped according to the cause of death of the subject into six groups:

Group 1: Individuals who have died from cranioencephalic trauma and cerebral hemorrhages (24 cases).

Group 2: Death from asphyxial mechanisms (ten cases).

Group 3: Death as a result of tumoral processes (18 cases).

Group 4: Cardiac causes: myocardial infarction, cardiac insufficiency, and so forth (19 cases).

Group 5: Pulmonary causes: embolisms, acute edema of the lung, and so forth (14 cases).

Group 6: Miscellaneous, including: hepatic cirrhosis, comas, and so forth (15 cases).

We wish to point out that in none of the cases was the existence of articular pathology of any kind shown.

In these liquids we have studied a series of enzymes: alkaline phosphatase, lactic acid dehydrogenase (LDH), and GOT, using the apparatus Serum Multianalyzer Automatized 12/60 for the analysis, which uses chromometer methods.

The results obtained were submitted to a variance analysis (ANOVA) on one path. To check the equality of variance, Bartlett's test for homogeneity of variances was carried out. In those cases where this test showed an inequality, the transformation of the variable \bar{x} in $\sqrt{\bar{x}}$ was done to stabilize them and afterwards a second ANOVA was done.

Results and Discussion

Alkaline Phosphatase

On studying Table 1 we see a significant decrease of the values of the alkaline phosphatase in the deaths whose cause was by any kind of tumor (13.61 U/L) with regard to the deaths by cranioencephalic trauma (32.33 U/L) and pulmonary deaths (29.21 U/L).

TABLE 1—Alkaline phosphatase ANOVA after the transformation of the mean \bar{x} into $\sqrt{\bar{x}}$ to stabilize the variance.^a

Cause of Death	Mean \bar{x} , U/L	$\sqrt{\bar{x}}$	Standard Deviation SD	No. of Cases
TCE	32.33	5.18	2.40	24
Asphyxia	18.70	4.20	1.08	10
Tumors	13.61	3.21	1.86	18
Cardiac	18.95	4.09	1.54	19
Pulmonary	22.21	5.04	2.02	14
Mixed	17.20	3.98	1.22	15
		INTERCLASS	INTRACCLASS	
	Sum square $\sqrt{\bar{x}}$	49.93	318.85	
	G.1	5	94	
	Variance	9.99	3.39	
	F.Snedecor = 2.94	Significant $P < 0.05$		

^aWe have calculated the Newman Keul's statistic to find between what groups of death causes there are significant differences. We have found differences between the TCE and the tumor groups.

These results correspond with what Navarro et al [7] refer to in serum and pericardial liquid where they see a rise of the proportions of this enzyme in those subjects whose deaths were caused by cranial trauma (266.89 U/L), with regard to the asphyxia (146.14 U/L), rise which relates to the important cellular lysis which accompanies them.

Similar behavior is found in the serum by Enticknap [1] and Schmidt [2].

Lactic Acid Dehydrogenase (LDH)

The results are seen in Table 2. On studying it, we find a decrease of the proportion of this enzyme in the tumor group (62.78 U/L) with regard to the asphyxia (201.9 U/L) and deaths as a result of pulmonary causes (160.0 U/L).

TABLE 2—Lactic acid dehydrogenase (LDH) ANOVA after the transformation of the mean \bar{x} into $\sqrt{\bar{x}}$ to stabilize the variance.^a

Cause of Death	Mean \bar{x} , U/l	$\sqrt{\bar{x}}$	Standard Deviation SD	No. of Cases
TCE	137	10.52	5.23	24
Asphyxia	201.9	13.03	5.98	10
Tumors	62.78	7.12	3.98	18
Cardiac	134.4	10.18	5.71	19
Pulmonary	160	12.22	3.37	14
Mixed	146	10.82	5.57	15
		INTERCLASS	INTRACCLASS	
	Sum square $\sqrt{\bar{x}}$	313.60	2340.02	
	G.1	5	94	
	Variance	12.72	24.89	
	F.Snedecor = 2.52	Significant $P < 0.05$		

^aWe have calculated the Newman-Keul's statistic to find between what groups of death causes there are significant differences. We have found differences between the tumor group and the asphyxia and pulmonary groups.

There are several authors who have referred in live subjects to increases of the enzyme in different pulmonary case histories, that is, in serum, King [8]; in lung infarction and in pulmonary embolism, Garcia Viudez [9]; and in cerebrospinal fluid (LCR) and in decompensated hypoxemia situations, Martinez Barrero [10].

These increases of LDH in pulmonary illnesses might be attributed to the fact that the lung is rich in this enzyme, especially in Fractions 2 and 3 (F-2 and F-3) as King [8] comments on.

In the corpse, Navarro et al [7] do not find in serum nor pericardial liquid any variations of this enzyme with regard to the different causes of death. Luna [11], although he does not find variations between the global value of LDH and causes of death, refers to an increase of Fraction 2 (F-2) of this enzyme in bronchopulmonary and pulmonary embolism processes in pericardial liquid, as well of Fraction 4 (F-4) in pulmonary embolic processes and trauma and hemorrhages of the central nervous system.

It is deduced that it is more useful clinically and thanatologically to determine the isoenzymes of LDH than to know their global figure.

Glutamic Oxalacetic Transaminase

On looking at Table 3, we see a significant increase of GOT for the group of deaths caused by cranioencephalic trauma (39.21 U/L) with regard to those of the mixed group (4.80 U/L).

In the live subject, King [8] refers to increases of GOT in the LCR of subjects affected by cerebrovascular accidents.

In the corpse, Luna [11], in pericardial liquid, and Navarro et al [7], in serum and pericardial liquid, do not find variations of this enzyme with regard to the cause of death.

Starting from the fact that GOT is an enzyme that appears in any tissular cytolysis process and that the tissular destruction is greater in the cranioencephalic trauma group than in the mixed one, we think that this could be the origin of the significant increase in the first one.

Conclusion

Summing up, we conclude that alkaline phosphatase and GOT are found increased in cranioencephalic trauma, possibly as a result of the important cellular lysis which goes with them; and the LDH is increased in the pulmonary processes, almost certainly with relation to the great quantity of this enzyme in the lung.

TABLE 3—*Glutamic oxalacetic transaminase (GOT) ANOVA after the transformation of the mean \bar{x} into $\sqrt{\bar{x}}$ to stabilize the variance.*^a

Cause of Death	Mean \bar{x} , U/l	$\sqrt{\bar{x}}$	Standard Deviation SD	No. of Cases
TCE	39.21	4.79	4.12	24
Asphyxia	11.10	2.48	2.34	10
Tumors	10.78	1.92	2.74	18
Cardiac	30.53	3.99	3.92	19
Pulmonary	17.86	3.43	2.57	14
Mixed	4.80	1.08	1.97	15
		INTERCLASS	INTRACCLASS	
	Sum square $\sqrt{\bar{x}}$	174.86	984.76	
	G. I	5	94	
	Variance	34.97	10.48	
	F. Snedecor = 3.34	Significant $P < 0.05$		

^aWe have calculated the Newman-Keul's statistic to find between what groups of death causes there are significant differences. We have found differences between the TCE and mixed groups.

References

- [1] Enticknap, J. B., "Biochemical Changes in Cadaver Serum," *Journal of Forensic Medicine*, Vol. 7, 1960, pp. 135-146.
- [2] Schmidt, "Valeur des examens effectués post-mortem," *Actua*, Vol. 20, 1970, p. 13.
- [3] Edson, E. F., et al, "Stabilité des cholinesterases sanguines après morte," *Medicine, Science and the Law*, Vol. 4, No. 2, 1962, pp. 252-267.
- [4] Sawaguchi, A. and Funao, T., "Postmortem Change of Serum Enzymes and Isoenzymes," *Journal of the Tokyo Women's Medical College*, Vol. 44, No. 9, 1974, pp. 833-840.
- [5] Klein, H. et al, "Intérêt de l'activité du alcool-déhydrogenase et de la transaminase des acides glutâmico-oxalacetic du foie humain après mort," *Deutsche Zeitschrift Fuer Die Gesamte Gerichtliche Medizin*, Vol. 52, No. 4, 1962, p. 615.
- [6] Hinojal, R., Fernandez, P. et al, "Estude sur l'agonie a travers la enzymes seriques (GOT, GPT, Phosphatase Alcalina, 8-GO, Colinesterapia y HBDH). La date de la mort par rapport aux enzymes seriques," *Comunicaciones al I Congreso de Medicina Legal de Oporto*, Oporto, 22-25 June 1983, in press.
- [7] Navarro, J. A. et al, "Comportement biochimique du serum et du liquide pericardique dans differents types de mort," *Actas de XII Congres de L'Academie Internationale de Medicine Legale et Medicine Sociale*, Viene, May 1982.
- [8] King, J., *Enzimología Clínica Práctica*. Acribia, Zaragoza, Spain, 1968.
- [9] Garcia Viudez, J. A., "Diagnóstico diferencial del infarto de miocardio con la embolia pulmonar," *Medicina Clínica*, Vol. 70, No. 9, 1978, pp. 417-424.
- [10] Martinez Borrero, F. et al, "La actividad enzimática, sérica y humoral en afecciones hipoxémicas y metabolicas descompensadas," *Medicina Clínica*, Vol. 65, No. 8, 1975, pp. 407-409.
- [11] Luna, A., "Comportamiento bioquímico del líquido pericardico en función de la causa de muerte y del tiempo posible de agonía," doctoral thesis, Granada, 1979.

Address requests for reprints and additional information to
 Dolores Serrat More, M.D.
 Department of Legal Medicine
 Facultad de Medicina
 C/Domingo Miral
 Zaragoza, Spain