



## EVALUATION OF RIGOR MORTIS AND ITS APPLICATION TO ASSESSMENT OF THE POSTMORTEM INTERVAL IN DOGS

**Khaled A. Abdou**

Dept. of Forensic Med. and Toxicology, Fac. of Vet. Med. (Beni-Suef), Cairo University

---

### **ABSTRACT :**

Rigor mortis is Latin for "stiffness of death". It is used medically to describe the stiffness of skeletal muscles that appears soon after death. The use of rigor mortis, course, intensity and duration in the estimation of early postmortem interval in dogs was the aim of the present work. Apparently healthy 15 mature stray dogs were classified into three equal groups (n=5). Dogs in the three groups were killed, in the first group, by I/V injection of air (control), in the second group by 10-g strychnine per Os for each dog to produce hyperactivity and in the third group by 40-mg/thiocolchicoside/kg b.wt. (I/V) to produce complete muscular relaxation before death. Rigor mortis, course, intensity, development, and duration were recorded by palpation the cadaver's muscles in dogs of each group every half an hour for 48 hours postmortem at an ambient temperature of 38 °c. The time required for rigor mortise's first phase (delay phase) was 3-4, 2-2.5 and 3.5-4 hours, for the second phase was, 11-12, 6.5-7.5 and 11-12 hours, and for the third phase was 21-23, 15-16 and 23-24 hours postmortem in the three groups respectively. The time of rigor mortis, course and sequences was estimated for each group. These results cited an index for the sequences and the time range required for rigor mortis completion in dogs.

---

### **INTRODUCTION :**

The changes that occur at the time of death in the body will depend very much on the manner of death. Think of the body as an interconnected and interdependent web of structures and functions, these are normally kept in harmony by very resilient homeostatic processes. However, if one or several

vital parts or functions are lost or damaged, then homeostasis will not be maintained and the individual as a whole will die. When someone dies, some of the component cells in their tissues remain alive for up to several days afterwards, reminding us that the distinction between life and death is not completely clear-cut. Rigor mortis or postmortem stiffening is the first and most

considerable change occurred in the cadaver or corpse. It developed at a variable period after death and succeeded the state of primary muscular flaccidity (Gordon et al., 1988). Contraction of all the voluntary and involuntary muscles, smooth muscles of the internal organs, iris muscles and stiffening of the joints characterized this phenomenon (Gordon & Shapiro, 1975 and Gracey, 1986). Small masses of muscle involved completely much more rapidly than large masses. The size of different joints and muscles controlling them determined the development of rigor (Mason, 1983 and Gordon et al., 1988). High environmental temperature, fatigue or exhaustion before death, age, muscular development, anatomical location of the muscles, species and sex are several factors influenced the onset and duration of rigor mortis (Bendall, 1960; Gunn, 1978; Alvi, 1980; Honikel et al., 1981; Armstrong et al., 1982; and Gordon et al., 1988). The sequences of rigor mortis development regarded as a valuable aid in determining the early post-mortem interval, the rough time of death, the position of the body after death and the cause of death (Gordon and Shapiro, 1975, Eikelenboom, 1982; Gracey, 1986 and Krompecher et al., 1983). Many authors, describe the course and mode of rigor's onset in human cadavers (Gonzales et al., 1954; Smith and Smith, 1955; Polson, 1965 and Simpson, 1974)

The present study was conducted to evaluate the use of rigor mortis, course, intensity and duration in the estimation of early postmortem interval and to cite an index for the sequences and the time range required for rigor mortis completion in dogs because of the lack of literatures about this work in animals although its importance in veterinary forensic practice.

## **MATERIALS AND METHODS :**

Apparently health 15 mature stray dogs weighing 7-10 kg were obtained from Beni Suef Governorate streets. They were kept under good conditions for three days before the experiment, then were classified into three equal groups (n=5). Dogs in the three groups were killed, in the first group (GI), by I/V injection of air and was kept as control, in the second group (GII) by 10 g of strychnine per Os for each dog to produce hyperactivity and in the third group (GIII) by 40 mg/thiocolchicoside/kg b.wt. (I/V) to produce complete muscular relaxation before death. The rigor mortis, course, intensity, development and duration were recorded by palpation the cadaver's muscles of dogs in each group every half an hour for 48 hours postmortem, which ideally should correspond to the usual time of rigor mortis in its three phases (Krompecher et al., 1983). The experiments were carried out at an ambient temperature of 38°C. The statistical analysis for the obtained results was carried out after Snedecor and Cochran (1971).

## **RESULTS :**

Concerning the results of this investigation we could classify rigor mortis into three phases, the first phase, when a well marked rigidity was established in the muscles, the second phase, when the complete rigidity was established and the third phase, where the rigor begin to pass off. In the first group, dogs were killed by I/V injection of air and were kept as control, the time required for the first phase was 3-4 hours, for the next phase was 11-12 hours while for the last phase was 21-23 hours postmortem. In the second group, dogs were killed by strychnine

to produce hyperactivity before death, the time required for the first phase was 2-2.5 hours, for the second phase was 6.5-7.5 hours postmortem and for the third phase was 15-16 hours postmortem. The second group showed significant decrease in the time of the three phases of rigor mortis compared with that in the control group. In the third group, dogs were killed by thiocolchicoside (muscle relaxant) to produce complete muscular relaxation before death, the time required for the first phase was 3.5-4 hours, for the second phase was 11-12 hours and for the third phase was 23-24 hours postmortem. The third group showed non significant increase in the time of the three phases of rigor mortis compared with the control group (Table & Figure 1).

Rigor mortis course and sequences was as the same in the all cadaver with only few differences were showed despite of the cause of death. Rigor mortis was began in all muscles of the cadaver (voluntary & involuntary) after death but it first noticed in the lower lip (depressor labii mandibularis) followed by the upper lip (levator labii

maxillaris) and in the small muscles of the eyelids followed by the jaw muscles, first in the lower mandible and in the cheek muscles (masseter muscle). The eyeballs appear retracted inside due to the affection of the iris muscle. Next it appeared in the muscles of the neck and thorax followed by the muscles of the thoracic limb (the stiffness first appeared in the fetlock joint, carpal joint, and at last in the shoulder joint). After this, rigor mortis was noticed in the trunk muscles followed by the muscles of the hind limb (the stiffness first appeared in the fetlock joint and hock joint). At last, it appeared in the tail and perineum region. Rigor mortis was developed with the time until complete or a well-marked rigidity was established in the all palpated muscles. A decrease in rigor mortis intensity began to appear and it pass away leaving the body in the same order as it came gradually till complete relaxation when the body tissues autolysis began in different groups. Time of rigor mortis well noticed, complete developed and when its intensity decreased and it pass off were recorded in Table, (2).

Table (1): Rigor mortis duration in the three groups (Comparison of rigor mortis progress rate)

Groups		Phase I hours	Phase II hours	Phase III hours
GI (control)	Range	3 - 4	11 - 12	21 - 23
	X±SE	3.6±0.19	11.5±0.22	21.8±0.34
G II	Range	2 - 2.5	6.5 - 7.5	15 - 16
	X±SE	2.3±0.12***	6.80 ± 0.19***	15.40 ± 0.19***
G III	Range	3.5 - 4	11 - 12	23 - 24
	X±SE	3.80 ± 0.122	11.7 ± 0.20	23.60 ± 0.187

Results represented by range & X ± SE

\*\*\* Significant at P<0.001

**Table (2): Development of rigor mortis in different regions and muscles in different groups in relation to postmortem intervals (hrs)**

Groups	Phases	Lower lip	Upper lip	Eye lids	Lower mandible	Upper mandible	Cheek muscles	Neck muscles	Thorax muscles	Fore limb	Trunk muscles	Hind limb	Tail & perineum
G I ©	Phase I	2 - 2.5 2.2±0.12	2 - 2.5 2.2±0.12	2 - 2.5 2.2±0.12	2 - 2.5 2.2±0.12	2 - 3 2.5±0.16	2.5 - 3 2.7±0.12	2.5 - 3 2.8±0.12	2.5 - 3 2.8±0.12	3 - 3.5 3.3±0.12	3 - 3.5 3.3±0.12	3.5-4 3.7±0.12	3.5-4 3.8±0.12
	Phase II	6 - 6.5 6.5±0.12	6 - 6.5 6.5±0.12	6 - 6.5 6.5±0.12	6.5 - 7 6.5±0.16	6 - 7 6.2±0.19	7 - 7.5 7.3±0.12	8 - 8.5 8.2±0.12	8 - 8.5 8.4±0.10	9 - 9.5 9.2±0.12	9.5 - 10 9.8±0.12	10-11 10.5±0.22	11-12 11.6±0.19
	Phase III	12 - 13 12.3±0.19	12 - 13 12.3±0.19	12 - 13 12.3±0.19	12 - 13.5 13.0±0.27	12 - 13.5 12.4±0.29	12 - 13.5 12.3±0.29	14 - 15 14.2±0.19	14 - 15 14.5±0.22	15 - 17 16.2±0.37	16 - 18 17.4±0.40	18-21 19.6±0.51	21-23 22.0±0.45
G II	Phase I	1 - 1.5*** 1.2±0.12	1 - 1.5*** 1.2±0.12	1 - 1.5*** 1.3±0.12	1.5 - 2 * 1.7±0.12	1.5 - 2 1.9±0.2	1.5 - 2** 1.9±0.2	1.5 - 2** 1.9±0.2	2 - 2.5 2.40±0.2	2 - 2.5*** 2.2±0.12	2 - 2.5*** 2.4±0.2	2-2.5*** 2.4±0.2	2-2.5*** 2.4±0.2
	Phase II	5 - 5.5*** 5.2±0.12	5 - 5.5*** 5.3±0.12	5 - 5.5*** 5.4±0.09	5.5 - 6** 5.9±0.09	6 - 6.5 6.4±0.09	6 - 6.5*** 6.4±0.09	6.5 - 7*** 6.7±0.12	6.5 - 7*** 6.8±0.12	7 - 7.5*** 7.1±0.09	7 - 7.5*** 7.2±0.12	7-7.5*** 7.3±0.12	7-7.5*** 7.4±0.09
	Phase III	6 - 7*** 6.4±0.24	6 - 7.5*** 6.7±0.3	7 - 7.5*** 7.3±0.12	7.5-8.5*** 8.0±0.16	8.5 - 9*** 8.7±0.12	8.5 - 9*** 8.8±0.122	9 - 10*** 9.5±0.16	9 - 10*** 9.7±0.2	10-11.5*** 10.7±0.25	12 - 13*** 12.6±0.25	14-15*** 14.6±0.24	15.5-16*** 15.8±0.12
G III	Phase I	2 - 2.5 2.1±0.09	2 - 3 2.4±0.19	2 - 2.5 2.4±0.01	2 - 3 2.6±0.19	2 - 3 2.7±0.12	2.5 - 3 2.8±0.13	2.5 - 3 2.8±0.12	2.5 - 3 2.9±0.09	3 - 3.5 3.2±0.12	3 - 4 3.4±0.19	3-4 3.6±0.19	3-4 3.7±0.2
	Phase II	7 - 7.5** 7.2±0.12	7 - 7.5 7.4±0.45	7 - 8* 7.5±0.16	7 - 8** 7.6±0.19	7 - 8 7.7±0.45	7 - 8 7.8±0.19	7.5 - 8.5 8.1±0.24	8.5 - 9 8.7±0.12	9 - 10.5 9.5±0.22	9 - 10.5 9.7±0.19	11-11.5** 11.3±0.12	11-12 11.7±0.2
	Phase III	12.5 - 13 12.8±0.12	12.5 - 13 12.8±0.12	12.5 - 13* 12.9±0.09	12.5 - 14 13.5±0.32	14 - 15*** 14.6±0.19	15-15.5*** 15.3±0.12	15.5-16.5*** 16.1±0.19	16.5-17*** 16.8±0.12	17.5 - 18** 17.8±0.12	18 - 19.5** 19.1±0.29	20-22* 21.0±0.31	23-24* 23.4±0.19

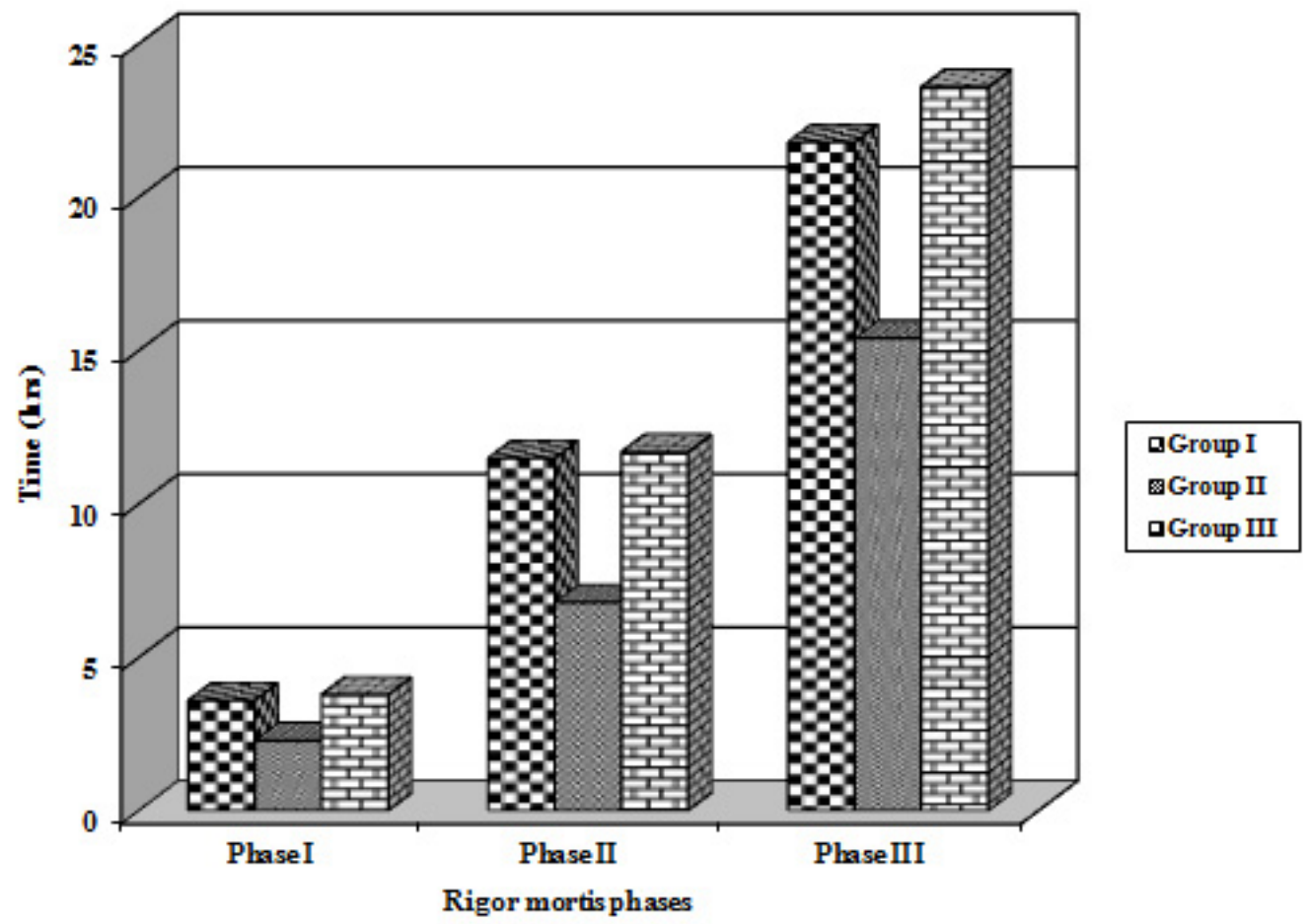
Results represented by range &  $\bar{x} \pm SE$  \* Significant at  $P < 0.05$  \*\*Significant at  $P < 0.01$  \*\*\* Significant at  $P < 0.001$

Phase I = First noticed well marked rigor mortis

Phase II= Development of complete rigor mortis rigidity

Phase III = Decreased the intensity and rigor mortis pass off.

**Fig. (1) : Duration of rigor mortis phases in different groups**



## **DISCUSSION :**

Rigor mortis is a well-known phenomenon, and is due to a complex chemical reaction in the body. In the living body muscles can function both aerobic and anaerobic. In the dead body muscle cells can only function anaerobically. When muscle cells work anaerobically the end product is lactic acid. In the living body, lactic acid can be converted back, by means of excessive oxygen uptake after an anaerobic exercise. In the dead body this can not happen, and the breakdown of glycogen in the muscles leads irreversibly to high levels of lactic acid in the muscles. This leads to a complex reaction where actin and myosin fuses to form a gel. This gel is responsible for the stiffness felt in the body. This stiffness will not be over before decomposition begins. As rigor mortis is due to a chemical reaction, the reaction time is due to temperature and the initial concentrations of lactic acid. High metabolic activity in the time just before death, for example when running, leads to higher levels of lactic acid, and shorter time for the rigor mortis to develop (Sherwood, 1997).

In the first group (control), where dogs were killed by I/V injection of air, the marked rigidity was established 3-4 hours, continued for 11-12 hours then begin to pass off and the resolution begin in 21-23 hours postmortem. Rigor mortis sets in as muscle cells run out of the energy substance called ATP (adenosine triphosphate). Even when a person is clinically dead, some cells within their tissues continue to survive for a while. After the circulation of blood ceases, surviving muscle cells resort to anaerobic glycolysis but eventually they become unable to make any more ATP. You will probably recall that in healthy muscle cells ATP is

involved in unlocking the cross bridges at the end of the power-stroke and energizing them ready for the next contraction. Calcium ions also leak into the compromised muscle cells, moving regulatory proteins away from the molecular cross-bridges between the myofilaments. The myofilaments then become locked in position as a result of these changes, and the skeletal muscles no longer give' or stretch when parts of the body are moved. Rigor mortis wears off as the tissues begin to decompose - proteolytic enzymes in the lysosomes of the muscle cells escape and begin to dissolve the myofilaments (Rutishauser, 1994). The stiffening, shortening and opacity of the muscles in rigor mortis are like those occurred during physical contraction of muscles but irreversible. In this reaction rigor mortis might depend on the conversion of glycogen to lactic acid. This reaction apparently detected to account for the gelation and coagulation of sarcoplasm (Polson, 1965 and Cassens, 1966). In rigor mortis contraction, the muscles, passed into rigor quickly due to run out of ATP supply and unbreaking of myosin-actin linkage formed in the last step of the contractile cycle. In rigor mortis ATP was turned over very slowly. The level of ATP began to fall and lost due to exhaustion of the system resynthesis it and due to the actions of ATP ase, myokinase and deaminase enzymes (Bendall, 1979). The maximum duration of rigor mortis in rabbit is consisted of three phases, the first phase "delay phase" in which the extensibility of the muscle remained constant and high it took about 11 hours. The next phase "rapid phase", in which the extensibility decreased rapidly. The last phase "post-rigor phase" in which the extensibility again become constant but at lower level. It was lasting for many

hours without change in extensibility until the putrefaction began ( Bendall, 1960).

In the second group where dogs were killed by strychnine, the marked rigidity was established 2-2.5 hours, continued for 6.5-7.5 hours then the rigidity begin to pass off gradually until complete resolution in 15-16 hours postmortem. The onset, duration and the end of rigor mortis were rapid and shorter than that of control. The reduction of time and course of rigor mortis may be attributed to the struggling before and during death, which caused the reduction of glycogen content of the muscle before death. This result was in agreement with that reported by Gracey (1986). The duration of rigor mortis was reduced by exhausting the animals before death by means of insulin or strychnine in convulsive dose in rabbit, horse, and beef muscles but the main difference between those investigators were in the duration and not in the overall pattern of rigor mortis (Bendall, 1960; Lawrie, 1953; Marsh, 1954 and Howard & Lawrie, 1956 &1957). Kenny and Tarrant (1984), mentioned that the level of glycogen might be lowered as a result of ante-mortem exhaustion, physical exercise, insulin, tetany or by any other physiological stress which led to short period of rigor mortis

In the third group injected by muscle relaxant, a well-marked rigidity was established 3.5-4 hours postmortem and continued 11-12 hours. Then the rigidity began to pass off gradually until complete resolution in 23-24 hours postmortem. The onset and duration of rigor mortis in the third group were delayed and longer than that of control group. This could attribute to the restoring ATP and glycogen from rapid destruction due to absence of struggling at

time of death. The treatment of well-fed animal with myanesisin (muscle relaxant) almost showed long period before rigor with long duration of rigor mortis ( Marsh, 1952).

Concerning to the course and sequences of rigor mortis development, rigor mortis was established in the muscles of the head, neck, thorax, and thoracic limb. Followed by the muscles of the trunk region, hind limb then by the tail and perineum region muscles respectively. The great individual deviation in rigor development within the same species depending on the physiological state of the individual animal (Lawrie, 1960 and Lawrie et al., 1963). Nosztray et al. (1983) and Gordeon et al. (1988) observed differences in rigor mortis duration in the individual muscles of the same species. These differences attributed to difference in the initial levels of creatine phosphate and ATP. The striking differences among animal species in the time course of rigor mortis development caused by three differences. The variations in membrane resistance against autolytic process or increasing acidification, the deviations in post-mortem release of Ca<sup>2+</sup> and other ions by muscle protein and the differences in the relation between the velocities of the glycolytic ATP resynthesis and its breakdown (Partmann, 1961 & 1963). Various investigators mentioned different time interval for the mode of onset, rate of progression and disappearance of rigor mortis (Gordon et al., 1988).

In conclusion, the author tried to cite an index for the sequences and the time range required for rigor mortis completion in dogs as a result of the absence of literature recorded the onset and disappearance of rigor mortis in different animal species. The time of rigor onset and its duration was a

helpful guide for suggesting the cause of death. The progressive development of rigor mortis is regarded as the most valuable postmortem change in determining the postmortem interval. Repeated measurements of the intensity of rigor mortis allow more accurate estimation of the time since death than the single measurement.

## REFERENCES

- Alvi, A. S. (1980): The influence of sex status on meat quality Characteristic in sheep. *Fleischwirtsch.* 60: 2037.
- Armstrong, R. B.; Saubert, I. V.; Seeherman, H. J. and Taylor, C. R. (1982): Distribution of fiber types in locomotory muscles of dogs. *Am. J. Anat.*, 163 (1): 87.
- Bendall, J. R. (1960): The structure and Function of Muscle: Post-Mortem Changes in Muscle. Vol. 3, (Bourne, G. H. Ed.). Academic Press New York.
- Bendall, J. R. (1979): Relations between muscle pH and important biochemical parameters during the post-mortem changes in mammalian muscles. *Meat Sci.*, 3: 143.
- Cassens, R. G. (1966): The Physiology and Biochemistry of Muscle as Food: General Aspects of Post-Mortem Changes. Ed. By Briskey et al., 1966.
- Eikelenboom, G. (1982): Einsetzbarkeit des Rigormeters bei Rinderschlachtier-korpern. *Pers. Mitt.*
- Honikel, K. O.; Hamid, A.; Fischer, Chr. And Hamm, R. (1981): Influence of postmortem changes in bovine muscle on the water - holding capacity of beef. Post-mortem storage of muscle at various temperatures between 0 and 30 c. *J. Food Sci.*, 46: 23.
- Howard, A. and Lawrie, R. A. (1956): Studies on beef quality: Part 2. Physiological and biological effects of various pre-slaughter treatments. CSIRO Australian Div. Food Preserv. Tech. Paper No. 2.
- Howard, A. and Lawrie, R. A. (1957): Studies on beef quality: Part 5. Further observations on biochemical and physiological response to pre-slaughter treatments. CSIRO Australian Div. Food Preserv. Tech. Paper No.
- Gonzales, T. A.; Vance, M.; Helpern, M. and Umberger, C. J. (1954): Legal "Medicine Pathology and Toxicology" Second edition. Appleton-Century-Crofts INC. New York.
- Gordon, I.; Shapiro, H.A. and Berson, S.D. (1988): Forensic Medicine: A Guide to Principles. Third edition. English Language Book Society, Bailliere Tindall.
- Gordon, I. And Shapiro, H. A. (1975): Forensic Medicine: A Guide to Principles. Churchill Livingstone. Edinburgh London and New York.
- Gracey, J. E. (1986): Meat Hygiene. Eighthth edition. English Language Book Society, Bailliere Tindall.
- Gunn, H. M. (1978): Differences in the histochemical properties of skeletal muscles of different breeds of horses and dogs. *J. Anat.*, 127 (3): 615.
- Kenny, A. W. and Tarrant, P. V. (1984): Meat quality in beef heifers slaughtered at oestrus. Proc. 30 th. Europ. Meet. Meat Res. Work. Bristol p 99.
- Kobayashi, M., Takatori, T., Iwadate, K., and Nakajima, M. (1996): Reconsideration of the sequence of rigor mortis through postmortem changes in adenosine nucleotides and lactic acid in different rat



- muscles. *Forensic Science International*, 82, 243-253.
- Krompecher, T., Beregerieux, C., Brandt-Casadevall, C., and Gujer, H.R. (1983): Experimental evaluation of rigor mortis: VI effect of various causes of death on the evolution of rigor mortis. *Forensic Science International*, 22,1-9.
- Lawrie, R.A. (1953): The onset of rigor mortis in various muscles of the draught horse. *J. Physiol.*, 121: 275.
- Lawrie, R.A. (1960): Post-mortem glycolysis in normal and exudative longissimus dorsi muscle of the pig in relation to so called white muscle disease. *J. Comp. Pathol.* 70: 273.
- Lawrie, R. A.; Pomeroy, R. W. and Cuthbertson, A. (1963): Studies on the muscles of meat animals. III. Comparative composition of various muscles in pigs of three weight groups. *J. Agric. Sci.*, 60: 195.
- Marsh, B. B. (1954): Observations on rigor mortis in whale muscles. *Biochim. Biophys. Acta.* 9: 127.
- Marsh, B. B. (1954): Rigor mortis in beef. *J. Sci. Food Agric.*, 5: 70.
- Mason, J. K. (1983): *Forensic Medicine for Lawyers*. Butter-worth London Boston Durban Singapore Sydney Toronto Wellington.
- Parekh, S.R. and Patel, B.D. (1972): Epidemiologic survey of Indian childhood cirrhosis. *Indian Paediatrics*, 9: 431-38.
- Partmann, W. (1963): Aktivierung der ATP. Spaltung in warm und kaltbutermuski durch gefrineren und mechanische verletzung. *Z. Ernährungswiss.*, 2:7
- Partmann. W. (1963): Postmortem changes in chilled and frozen muscle. *J. Food Sci.*, 28: 15.
- Rutishauser, S. (1994): *Physiology and anatomy: a basis for nursing and health care*. Edinburgh: Churchill Livingstone (Chapter 37: Death and dying, pp 625-631).
- Schafer, A.T. (1997): Absence of rigor mortis. *Lancet*, March 15, 349 (9054), 810.
- Sherwood, L. (1997): *Human physiology: from cells to systems (3<sup>rd</sup> edition)*. Belmont, CA: Wadsworth Publishing Company (page 232).
- Simpson, K. (1975): *Forensic Medicine*. Seventh edition. The English language Book Society. Edwaed Arnold (Publishers) Ltd.
- Smith, S. and Smith, F. F. (1955): *Forensic Medicine*. Tenth edition. A textbook for students and practitioners. J. and A. Churchill LTD. London.
- Snedecor, G.W. (1971): *Statistical methods* 14 th ed. the Iowa states collage press Amer., Iowa.

## تقييم التيبس الرمي و استخدامه فى تقدير الوقت بعد الموت فى الكلاب خالد عباس حلمى عبده

قسم الطب الشرعى و السموم - كلية الطب البيطرى - جامعه القاهرة (فرع بنسوييف).

تعد دراسة التيبس الرمى واحدة من أهم المظاهر والتغيرات الميتية فى الإنسان والحيوان ومن ثم فإن معرفة ميكانيكية ظهوره وحدته ومدى استدامته تعد من الأمور الهامة فى تحديد الوقت المنقضى على الموت ، ونظراً لقلّة الأبحاث فى هذا الاتجاه فقد تمت هذه الدراسة وذلك فى الكلاب لايضاح هذه الظاهرة .

تم استخدام خمسة عشر كلباً قسمت إلى ثلاثة مجموعات ، قتلت الأولى عن طريق الحقن بالوريد بواسطة الهواء ، وتم استخدامها كمجموعة ضابطة ، و فى المجموعة الثانية تم استخدام الاستركنين فى قتل تلك الحيوانات لإحداث تشنجات قبل وبعد الموت ، وفى المجموعة الثالثة تم قتل الحيوانات باستخدام مادة ثيوكولشيكوسيد ، والتي تتسبب فى ارتخاء العضلات . تم إجراء هذه التجربة فى درجة حرارة ٣٨ درجة مئوية. كما تم تتبع التيبس الرمى وتدوين النتائج التى تم الحصول عليها . ومن خلال النتائج المسجلة لوحظ أن المجموعة الأولى قد استغرقت المرحلة الأولى فيها ٣-٤ ساعة والمرحلة الثانية ١١-١٢ ساعة والمرحلة الثالثة ٢١-٢٣ ساعة . وفى المجموعة الثانية استغرقت المرحلة الأولى ٢-٢,٥ ساعة ، والمرحلة الثانية ٦,٥ - ٧,٥ ساعة والمرحلة الثالثة ١٥-١٦ ساعة. وفى المجموعة الثالثة استغرقت المرحلة الأولى ٣,٥ - ٤ ساعة ، والمرحلة الثانية ١١-١٢ ساعة ، والمرحلة الثالثة ٢٣ - ٢٤ ساعة. كما تم تسجيل خطوات ظهور واختفاء التيبس الرمى فى جثث الكلاب فى المجموعات الثلاث .

اتضح من هذه الدراسة أن توقيت ظهور واختفاء التيبس الرمى فى عضلات جثث الكلاب يختلف باختلاف سبب الموت ، الأمر الذى يدل على أنه يمكن الاعتماد على التيبس الرمى كواحد من العناصر الهامة فى تحديد وقت وسبب الموت فى الكلاب .