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ORIGINAL ARTICLE

A Survey on Electrocardiographic Parameters in Broiler Chickens Following the Intracerebroventricular Injection of Melatonin

Majid Taati[1*], Hassan Norouzian[2], Abbas Raisi[2]

[1] *Department of Basic Sciences, Faculty of Veterinary Medicine, Lorestan University, Khorramabad, Iran.*

[2] *Department of Clinical Sciences, Faculty of Veterinary Medicine, Lorestan University, Khorramabad, Iran.*

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Abstract

Objective- The pineal gland and its principal hormone melatonin play an important role in the circadian organization in birds. However, little is known about the role of melatonin in cardiac activity of broiler chicken. The aim of this study was to assess the impact of intracerebroventricular injection of melatonin in electrocardiographic parameters, heart rate and mean electrical axis in broiler chickens.
Design- Experimental study.
Animals- Forty male Ross broiler chickens
Procedures- Ross broiler chickens received intracerebroventricularly (ICV) melatonin. All ECGs were standardized at 1 mV = 20 mm, with paper speed of 50 mm/sec. Leads I, II, III, aVR, aVL and aVF were recorded.
Results- Injection of melatonin (200 nmol) increased the duration of T wave and QRS complex and declined the heart rate as compared to the control group (p<0.05). Mean electrical axis, calculated from leads II and III, did not differ between groups and it was between -93° and -99° in all chickens.
Conclusion and clinical relevance- Exogenous melatonin intracerebroventricularly results in sinusoidal bradycardia in broiler chickens.

* Correspondence to: Majid Taati; Department of Pathobiology, Faculty of Veterinary Medicine, Lorestan University, Khorramabad, Iran. E-mail: Taati.m@lu.ac.ir

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1. Introduction

Melatonin (N-acetyl-5-methoxytryptamine), mainly synthesized in the pineal gland, regulates several physiologic functions, including immunity, reproduction and sleep.[1] In natural environment, light intensity and duration are the most effective factors in secretion of melatonin.[2] In birds, melatonin also controls various biological activities such as circadian rhythm, feeding pattern, thermoregulation and neuroendocrine functions.[3] Melatonin may influence cardiovascular pathophysiology.[4] Classic melatonin membrane receptors (MT1 and MT2) are present in the heart and throughout the vascular system. Furthermore, melatonin-specific receptors are widely distributed in the central nervous system, especially the suprachiasmatic nucleus (SCN) of the hypothalamus.[5] Increasing evidences suggest a possible modulation of cardiac autonomic control by the SCN, a circadian center.[6] Studies have also shown that melatonin supplementation may reduce blood pressure.[7] In human beings, cardiovascular activity has a distinct circadian variation; blood pressure, heart rate, and vascular tone decrease at night.[8,9] Pineal melatonin synthesis is limited to the dark part of the light-dark cycle and the amplitude of the circadian melatonin rhythm is inversely related to length of photoperiod and to some extent also to light intensity.[1] To the authors' knowledge, the role of the melatonin in cardiac activity of chickens has not been studied yet. So, we designed a study to investigate the melatonin induced effects on cardiac activity in broiler chickens.

2. Materials and Methods

Animals

Forty male Ross broiler chickens (Dorbar Hatchery, Borojerd, Iran) were reared in a temperature-controlled room at 33° C, according to the recommendation of Ross 308 management guide catalogue, with continuous lighting until 3 weeks of age. Birds were provided with a mash diet (21% protein and 2,869 kcal/kg of metabolizable energy) and water ad libitum. When the birds reached 2 weeks of age, the temperature of the animal room was maintained at 22 ± 1° C in addition to the continuous lighting condition. All procedures in this work were conducted in agreement with the National Institutes of Health Guide for care and use of laboratory animals.

Drugs

Melatonin was purchased from Sigma Chemical Co. (St. Louis, MO, USA). Melatonin was dissolved in ethanol and further diluted in saline to a final volume (final concentration of ethanol, 4%).

Surgical preparation

At 3 weeks of age, broilers were anesthetized intramuscularly with ketamine (30 mg/kg) and xylazine (1 mg/kg)[10] and a 23-gauge thin walled stainless steel guide cannula was stereotaxically implanted into the right lateral ventricle, according to the technique applied previously by Zendehdel *et a*l.[11] The stereotaxic coordinates were AP= 6.7, L = 0.7, H = 3.5-4 mm below the duramater with the head oriented as described by Zendehdel *et al*.[11] The cannula was secured with three stainless steel screws placed in the calvaria surrounding each guide cannula, and then acrylic dental cement (Pars acryl) was applied to the screws and guide cannula. Anorthodontic # 014 wire (American Orthodontics) trimmed to the exact length of the guide cannula was inserted into it while the chicks were not being used for experiments. Lincospectin (50 mg/kg) (Razak Co. Tehran, Iran) was applied to the incision to prevent infection. The birds were allowed a minimum of 5 days recovery prior to injection.

Experimental design

In this experiment the birds were infused ICV with 0, 50, 100 and 200 nmol of melatonin in a volume of 10 µl into the right lateral ventricle (n = 10 for each group). Injections were made with a 29-gauge, thin walled stainless steel injecting cannula which extends 1.0 mm beyond the guide cannula. This injecting cannula was connected through 30-cm-long PE-20 tubing to 10 µl Hamilton syringe. Solutions were injected over a period of 30 sec. A further 30 sec period was allowed to permit the solution to diffuse from the tip of the cannula into the ventricle. All experimental procedures were performed during a 0200 p.m. to 0500 p.m. period.

Electrocardiography (ECG) recordings

Because of the thickness of poultry chest muscle, it is impossible to obtain any recording by using chest leads. Therefore, we recorded standard bipolar (I, II, III) and augmented unipolar (aVR, aVL, aVF) leads.[12] Alligator clip electrodes were attached to the skin at the base of the right and left wings and gastrocnemius muscle of the right and leftlimbs.[12] Good clip-to-skin contact was established by using electrode gel. The chickens were placed on a wooden table and calmed down by covering them with a cloth for 5 min before recording.[12,13] The ECGs were recorded by a digital electrocardiograph (Kenz 110, Japan) and all recordings were calibrated to 1 mV/20 mm, with a paper speed of 50 mm/sec.[12] The ECG was recorded while animals were conscious. All procedures took place in an isolated quiet room in order to minimize the stress for birds. The duration and amplitude of waves on the trace were measured in lead II [12] and mean electrical axis (MEA) of ventricular depolarization in the frontal plane was calculated using leads II and III.[12] Measurement of heart rate was also taken in lead II.[12]

Statistical analysis

All results were analyzed using SPSS/PC program. The difference among groups before or after injection was separately analyzed using one-way ANOVA. Moreover, to determine the difference in each group between before and after administration, paired sample t-test was applied. All measurements are expressed as mean ± SEM.

3. Results

Data of the ECG findings of chickens following the intracerebroventricular injection of melatonin are depicted in Table 1 and Figures 1 and 2. In lead I, traces were very low amplitude. The P wave was positive in leads I-III, aVL and aVF, and negative in lead aVR. The QRS polarity was negative in I-III and aVF leads, being positive in aVR and aVL leads. The Q wave was not seen in any leads and QRS complex was in the form of rS in all leads. T wave was positive in leads I, II, III and aVF and negative in leads aVR and aVL. T wave was in the form of P-T, except that chickens treated with 200 nmol of melatonin had P waves separated from the T. Although the duration of T waves and QRS complexes was significantly increased following the injection of melatonin with doses of 200 nmol, the heart rate of these chickens (294 beats/min) decreased compared to the control (331-351 beats/min) ($p<0.05$, Table 1). Administration of melatonin did not significantly influence other ECG parameters.

Table 1. The durations and amplitudes of waves and the heart rate in lead II in the control and melatonin-treated chickens (mean ± SEM).

Groups	ECG values				
	QRS (sec)	**QRS (mV)**	**T(T+P) (sec)**	**T (mV)**	**Heart Rate**
Control (vehicle)	0.045±0.034	0.21±0.017	0.095±0.067	0.13±0.01	333±2.8
Melatonin (50nmol)	0.055±0.0061	0.23±0.024	0.10±0.014	0.16±0.019	323±3.5
Melatonin (100nmol)	0.058±0.004	0.24±0.023	0.12±0.014	0.14±0.02	327±4.6
Melatonin (200nmol)	0.065±0.004*	0.25±0.0196	0.13±0.015*	0.15±0.01	294±3*

*indicates significant different with control group ($p<0.05$).

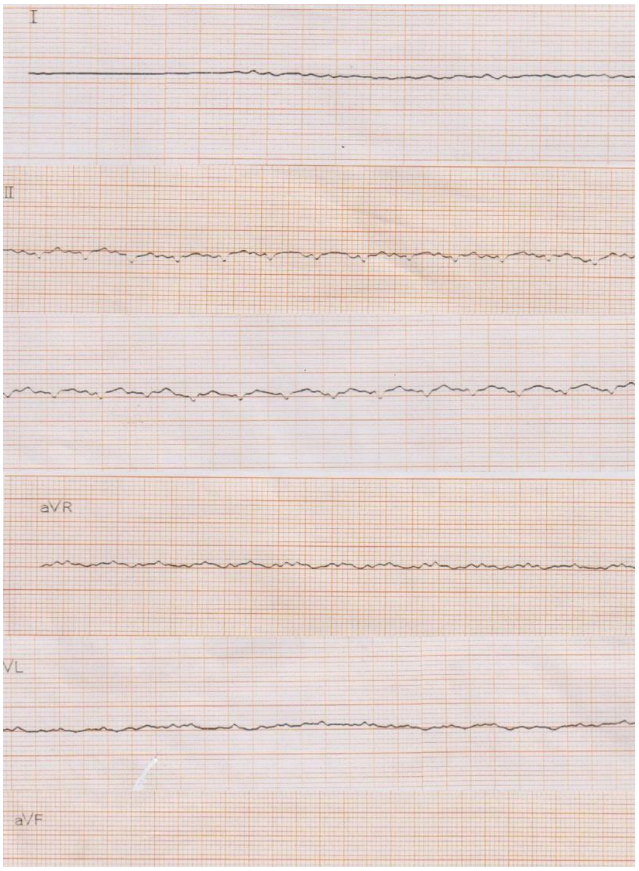

Figure 1. Electrocardiogram of control group (ICV injected normal saline), standardization; 1 mV = 20 mm, chart speed: 50 mm per second.

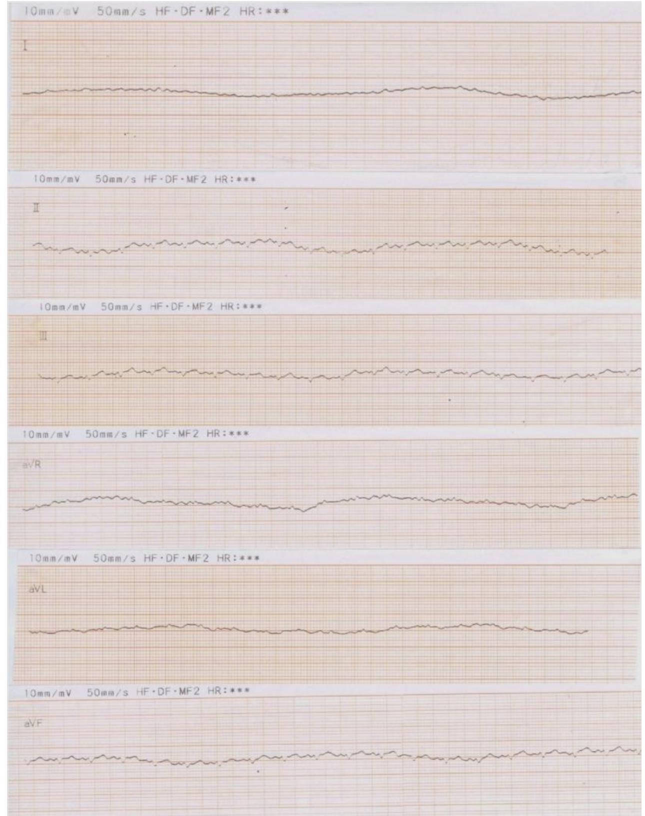

Figure 2. Electrocardiogram of melatonin (200 nmol) group (ICV injected melatonin), standardization; 1 mV = 20 mm, chart speed: 50 mm per second.

4. Discussion

The ECG is relatively a simple technique, used routinely for monitoring cardiac functions. The results of the present study showed that morphological patterns of PQRS-T deflections in all leads obtained from control and treatments were predominantly in agreement with the previous studies on broiler chickens.[12-14] The HR, duration of QRS complex and T (P+T) wave altered after ICV injection of melatonin. The T wave is usually seen in the form of P-T in chickens. In this study, the T wave was seen in the form of P-T in control group. With a rapid HR, P wave and preceding T wave as well as T wave and preceding QRS complex may intermix.[15] Previous studies have suggested when P-on-T phenomenon is not associated with high HR, it may indicate alterations in ventricular repolarization or may be related to ascites.[16] In the current study, P-on-T phenomenon is related to the high HR.

In our study, mean amplitude of QRS complexes (0.135 mV) approximately was the same as our previous study on broiler chicken,[10] however, it was lower than the results obtained by Çınar *et al*.[12] The discrepancy may be related to the different species and weight of chickens in those two experiments. ICV injection of melatonin or chlorpheniramine could not significantly change the amplitude of QRS complexes. An increased voltage in the QRS complexes may be indicative of heart muscle hypertrophy.[16] Additionally, it has been noticed that the QRS complex amplitude increases in birds developing ascites syndrome.[17, 18]

ICV infusion of melatonin with a dose of 200 nmol significantly increased the duration of QRS complex as compared to the control groups. This effect is related to the presence of a bradycardia (290-298 bpm). The amplitude of T (T+P) wave changed between 0.08 and 0.12 mV in the current study. Melatonin did not significantly affected the amplitude of T (T+P) wave. Elevated and peaked T waves were identified as a sign of hyperkalemia in ducks.[12] The same T pattern can be recorded in shocked raptors and after electrocution as a result of hyperkalemia.[19] ICV application of melatonin (with dose 200nmol) significantly increased the duration of the T wave, (Tables 1). Indeed, these alterations in the duration of the T wave were due to bradycardia (294 bpm), caused by melatonin. The mean MEA in the present study was -96°, which is characteristic of the avian ECG and implies the negative polarity of QRS complex in II, III and aVF leads.[20] The mean cardiac axis represents the net direction and magnitude of cardiac electrical force. In essence, the QRS electrical axis is useful because it helps to determine the position of the heart in the chest, patency of electrical pathways and integrity of muscle mass. Despite some criticisms, a number of studies suggest that morphological aspect analysis of the electrical axis (amplitude and duration changes by ECG) is one of the primary tools to detect ventricular hypertrophy and sudden cardiac death.[21] We first speculated that ICV injection of melatonin may promote deviations in MEA by inducing arrhythmias in chickens, but the difference between mean MEA in the groups was not statistically significant.

In accordance with previous studies in chickens,[12] the heart rate in this study ranged from 314 to 351 in the control groups. Melatonin with only 200nmol significantly reduced the HR. In our study, for the first time it was shown that melatonin, given intracerebroventricularly, modulates the heart rate and can induce bradycardia in broiler chickens. As it was previously shown in dogs [22], the results show that melatonin acts as a negative chronotropic agent in chicken. Previous studies have suggested that melatonin possibly influences autonomic cardiovascular regulations in animals. Pinealectomy induced hypertension in rats, whereas melatonin administration decreased blood pressure and heart rate in pinealectomized and normal rats.[23] Melatonin modified the turnover rates of cardiac catecholamine in Syrian hamsters.[24] It also inhibited the central sympathoadrenomedullary outflow in rats.[25] Besides, in human beings, it is probable that melatonin influences autonomic cardiovascular modulation.[7] Nishiyama *et al*. showed that melatonin administration increases cardiac vagal tone in healthy men.[8] Melatonin-specific receptors are widely distributed in the central nervous system, especially the suprachiasmatic nucleus (SCN) of the hypothalamus.[26] Cardiac autonomic activity can be controlled by the central nervous system at various levels and increasing evidence further suggests a possible modulation of cardiac autonomic control by the SCN, a circadian center.[27] Melatonin might have exerted the autonomic effect through the SCN.

It is obvious that the effects of melatonin on cardiac activity may be advantageous in broiler chickens, when a fast growth and intensive breeding contributes to development of diseases such as sudden death syndrome.[28] It has been revealed that when an animal encounters a noxious stimulus, two major physiological responses to stress, the activation of the sympathetic nerve system and ACTH release, induce tachycardia.[12,29-31] Concurrently, it is postulated that melatonin secretion could be a defense mechanism against numerous types of stress and may be involved in physiological adaptation to the given stress. Experimental studies have demonstrated the suppressive effect of melatonin on sympathetic nervous system.[25] Hence, it is possible to hypothesize that in a stressor condition, increased melatonin secretion can act to alleviate the adverse effects of histamine in heart activity of broiler chickens. Melatonin addition to the diet of chickens was reported to ameliorate some of the immunosuppression associated with exposure to heat stress.[32] The role of stress as an influencing factor on cardiac arrhythmias in broilers is well understood.[16] The beneficial effects of intermittent lighting programs on broiler production have been studied in the incidence of sudden death syndrome (SDS).[33] The incidence of sudden death syndrome has been numerically lower in melatonin treated chickens.[34] The exact pathogenesis of SDS is not thoroughly understood, however, cardiac arrhythmia, being common among fast growing broilers, is an important part of the story.[16] So it is possible to hypothesize that in a stressor conditions, increased melatonin secretion can be utilized to alleviate the adverse effects of stress in heart activity of broiler chickens.

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Conflict of Interests

The authors declare they have no conflicts of interest.

References

1. Yonei Y, Hattori A, Tsutsui K, Okawa M, Ishizuka B. Effects of melatonin: basics studies and clinical applications. *Anti-Aging Medicine*, 2010; 7(7): 85-91.
2. Olanrewaju HA, Thaxton JP, Dozier WA, Purswell J, Roush WB, Branton SL. A review of lighting programs for broiler production. *International Journal of Poultry Science*, 2006; 5(4): 301-308.
3. Li QQ, Zhao XL, Xu HL, Zhao BY, Zhu Q. Research progress on melatonin receptor in poultry. *Energy Procedia,* 2011; 11: 2252-2257.
4. Tengattini S, Reiter RJ, Tan DX, Terron MP, Rodella LF, Rezzani R. Cardiovascular diseases: protective effects of melatonin. *Journal of Pineal Research,* 2008; 44(1): 16-25.
5. Adamah-Biassi EB, Zhang Y, Jung H, Vissapragada S, Miller RJ, Dubocovich ML. Distribution of MT1 melatonin receptor promoter-driven RFP expression in the brains of BAC C3H/HeN transgenic mice. *Journal of Histochemistry&Cytochemistry*, 2014; 62(1):70-84.
6. Van Eekelen AP, Houtveen JH, Kerkhof GA. Circadian variation in cardiac autonomic activity: reactivity measurements to different types of stressors. *Chronobiology International*, 2004; 21(1): 107-129.
7. Pechanova O, Paulis L, Simko F. Peripheral and central effects of melatonin on blood pressure regulation. *International Journal of Molecular Sciences*, 2014; 15(10): 17920-17937.
8. Nishiyama K, Yasue H, Moriyama Y, Tsunoda R, Ogawa H, Yoshimura M, Kugiyama K. Acute effects of melatonin administration on cardiovascular autonomic regulation in healthy men. *American Heart Journal*, 2001; 141(5): 13A-17A.
9. Hower IM, Harper SA, Buford TW. Circadian rhythms, exercise, and cardiovascular health. *Journal of Circadian Rhythms*, 2018; 16: 7.
10. Rafiei M, Taati M, Alavi S, Nayebzadeh H, Zendehdel M. Effects of intracerebroventricular injection of histamine and H1, H2 receptor antagonists on electrocardiographic parameters in broiler chickens. *Iranian Journal of Veterinary Research*, 2011; 12(3): 192-198.
11. Zendehdel M, Taati M, Jonaidi H, Amini E. The role of central 5-HT 2C and NMDA receptors on LPS-induced feeding behavior in chickens. *The Journal of Physiological Sciences*, 2012; 62(5): 413-419.
12. Çinar A, Belge F, Donmez N, Ta A, Selçuk M, Tatar M. Effects of stress produced by adrenocorticotropin (ACTH) on ECG and some blood parameters in vitamin C treated and non-treated chickens. *Veterinarskiarhiv,* 2006; 76(3): 227-235.
13. ÇınarA, DonmezN. The effect of ration supplemented with zinc on ECG in broilers. *Turkish Journal of Veterinary and Animal Sciences*, 2001; 25(1): 81-85.
14. Taati M, Babapour V, Kheradmand A, Tarrahi MJ. The role of central endogenous histamine and H1, H2 and H3 receptors on food intake in broiler chickens. *Iranian Journal of Veterinary Research*, 2009; 10(1): 54-60.
15. Machida N, Aohagi Y. Electrocardiography, heart rates, and heart weights of free-living birds. *Journal of Zoo and Wildlife Medicine*, 2001; 32(1): 47-55.
16. Olkowski AA, Wojnarowicz C, Nain S, Ling B, Alcorn JM, Laarveld B. A study on pathogenesis of sudden death syndrome in broiler chickens. *Research in Veterinary Science*, 2008; 85(1): 131-140.
17. Odom TW, Hargis BM, Lopez CC, Arce MJ, Ono Y, Avila GE. Use of electrocardiographic analysis for investigation of ascites syndrome in broiler chickens. *Avian Diseases*, 1991: 738-744.
18. Owen RL, WidemanJr RF, Leach RM, Cowen BS, Dunn PA, Ford BC. Physiologic and electrocardiographic changes occurring in broilers reared at simulated high altitude. *Avian Diseases*, 1995: 108-115.
19. Espino L, Suárez ML, López-Beceiro A, Santamarina G. Electrocardiogram reference values for the buzzard in Spain. *Journal of Wildlife Diseases*, 2001; 37(4): 680-685.
20. Lumeij JT, RitchieBW. Cardiology. In: Ritchie BW; Harrison GJ. Harrison LR. Eds. *Avian medicine:*

principles and application. 1st ed. HBB International, Delray Beach, 1999; 695-722.

21. Simko F, Pechanova O, RepovaBednarova K, Krajcirovicova K, Celec P, Kamodyova N, Zorad S, Kucharska J, Gvozdjakova A, Adamcova M, Paulis L. Hypertension and cardiovascular remodelling in rats exposed to continuous light: protection by ACE-inhibition and melatonin. *Mediators of Inflammation*, 2014.
22. Neshatgharamaleki M, Seyyedsaadat M. Melatonin and dogs heart performance (electrocardiogram and enzymes changes). *Annals of Biological Research*, 2012; 3(6): 2615-2618.
23. Chuang JI, Chen SS, Lin MT. Melatonin decreases brain serotonin release, arterial pressure and heart rate in rats. *Pharmacology*, 1993; 47(2): 91-97.
24. Viswanathan M, Hissa R, George JC. Suppression of sympathetic nervous system by short photoperiod and melatonin in the Syrian hamster. *Life Sciences*, 1986; 38(1): 73-79.
25. Wang M, Yokotani K, Nakamura K, Murakami Y, Okada S, Osumi Y. Melatonin inhibits the central sympatho-adrenomedullary outflow in rats. *The Japanese Journal of Pharmacology*, 1999; 81(1): 29-33.
26. Arendt J. Physiology of the pineal: role in photoperiodic seasonal functions. Melatonin and the mammalian pineal gland. 1st ed. Chapman & Hall, London, 1995; 110-158.
27. Taylor EW, Jordan D, Coote JH. Central control of the cardiovascular and respiratory systems and their interactions in vertebrates. *Physiological Reviews*, 1999; 79(3): 855-916.
28. Bottje WG, Wideman RF. Potential role of free radicals in the pathogenesis of pulmonary hypertension syndrome. *Poultry and Avian Biology Reviews*, 1995; 6: 211-231.
29. Knigge U, Warberg J. The role of histamine in the neuroendocrine regulation of pituitary hormone secretion. *European Journal of Endocrinology*, 1991; 124(6): 609-619.
30. Miyazaki S, Onodera K, Imaizumi M, Timmerman H. Effects of clobenpropit (VUF-9153), a histamine H3-receptor antagonist, on learning and memory, and on cholinergic and monoaminergic systems in mice. *Life Sciences*, 1997; 61(4): 355-361.
31. Bealer SL. Central neuronal histamine contributes to cardiovascular regulation. *Physiology*, 1999; 14(3): 100-105.
32. Abbas AO, Gehad AE, Hendricks GL, Gharib HB, Mashaly MM. The effect of lighting program and melatonin on the alleviation of the negative impact of heat stress on the immune response in broiler chickens. *International Journal of Poultry Science*, 2007; 6(6): 651-660.
33. Scott TA. Evaluation of lighting programs, diet density, and short term use of mash as compared to crumbled starter to reduce incidence of sudden death syndrome in broiler chicks to 35 d of age. *Canadian Journal of Animal Science*, 2002; 82(3): 375-83.
34. Clark WD, Classen HL. The effects of continuously or diurnally fed melatonin on broiler performance and health. *Poultry Science*, 1995; 74(11): 1900-1904.

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چکیده

بررسی پارامترهای الکتروکاردیوگرافیک به دنبال تزریق داخل بطن مغزی ملاتونین در جوجه‌های گوشتی

مجید طاعتی[1]، حسن نوروزیان[2]، عباس رئیسی[2]

[1] *گروه علوم پایه، دانشکده دامپزشکی، دانشگاه لرستان، خرم‌آباد، ایران.*
[2] *گروه علوم درمانگاهی، دانشکده دامپزشکی، دانشگاه لرستان، خرم‌آباد، ایران.*

هدف- غده پینئال و هورمون اصلی آن، ملاتونین، نقش مهمی در سازمان‌دهی دوره‌های شبانه‌روزی فعالیت‌های پرندگان دارد. بااین‌حال اطلاعاتی در مورد نقش ملاتونین در فعالیت قلبی جوجه‌های گوشتی وجود ندارد. هدف از این مطالعه بررسی تأثیر تزریق داخل بطن مغزی ملاتونین بر پارامترهای الکتروکاردیوگرافیک، تعداد ضربان قلب و محور الکتریکی متوسط قلب در جوجه‌های گوشتی بود.

طرح- مطالعه تجربی.

حیوانات- ۴۰ قطعه جوجه‌ی گوشتی از نژاد راس.

روش کار- جوجه‌های گوشتی از نژاد راس ملاتونین را به‌صورت داخل بطن مغزی دریافت کردند. ثبت الکتروکاردیوگرام‌ها با استاندارد سرعت حرکت ۵۰ میلی‌متر در ثانیه برای کاغذ ثبت‌کننده و یک میلی ولت به ازای هر ۲۰ میلی‌متر انجام گرفت. لیدهای I، II، III، aVR ،aVL ،aVf ثبت شدند.

نتایج- تزریق ملاتونین به میزان ۲۰۰ نانومول به هر جوجه به‌طور معناداری در مقایسه با گروه کنترل مدت‌زمان موج T و کمپلکس QRS را افزایش داده و تعداد ضربان قلب را کاهش داد. محور الکتریکی متوسط قلب که با استفاده از لیدهای II و III محاسبه گردید بین ۹۳- تا ۹۹- درجه بوده و تفاوتی را در بین گروه‌ها نشان نداد.

نتیجه‌گیری و کاربرد بالینی- می‌توان نتیجه گرفت که ملاتونین خارجی تزریق‌شده به طریق داخل بطن مغزی موجب کاهش ضربان قلب (برادیکادی) در جوجه‌های گوشتی می‌شود.

واژه‌های کلیدی- ملاتونین، الکتروکاردیوگرام، جوجه گوشتی