Effect of Age and Growth Rate on Myocardial Irritability in Broiler Chickens (42861)

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Abstract. **Young broiler chickens are undergoing a period of extremely rapid growth and may be expected to be in a state of extreme endocrine and biochemical flux. These birds are also subject to a sudden death syndrome of unknown etiology. We hypothesized that an increased myocardial sensitivity in birds exhibiting early rapid growth may contribute to this syndrome. The objective of the current study was to investigate the interaction between early growth rate and age on myocardial irritability in young broiler chickens.**

This study utilized 74 male broiler chickens between 3 (Group A, 21–24 days) and 6 (Group B, 43–47 days) weeks of age exhibiting rapid (heavy weight, >425 g at 2 weeks) and slow (low weight, <350 g at 2 weeks) early growth. Physiologic parameters such as blood pressure, heart rate, and arterial blood gases in the awake, restrained birds were essentially unchanged across these groups. Myocardial irritability of the anesthetized bird (pentobarbital) as measured by the threshold to electrical fibrillation was significantly increased only in the Group A HW birds.

The results of this study suggest an increased myocardial irritability in large young broilers (3 weeks of age) that is no longer present in a similar group of older birds (6 weeks of age). These findings are consistent with the hypothesis that there is an increased myocardial sensitivity in birds succumbing to sudden death syndrome, with death due to myocardial fibrillation. Further research is needed to determine the mechanisms involve. [P.S.E.B.M. 1989, Vol 190]

Broiler chickens are subject to a sudden death syndrome (SDS) of unknown etiology. The syndrome is typically seen in otherwise asymptomatic, well grown, healthy birds of the flock, especially males (1, 2). Postmortem examination does not reveal specific lesions (1–4). The incidence of SDS begins within days of hatch and declines after 3 weeks of age (1, 2). Major difficulties in investigating this syndrome have been an inability to induce the syndrome or predict individual sensitivity to SDS and the lack of an experimental model. Physiologic studies of the embryonic chick heart have demonstrated ventricular sensitivity to exogenous catechols (isoproterenol) as early as the fourth embryonic day and adrenergic nervous control by the 16th day (5). There is a 10-fold decrease in the cardiac sensitivity to exogenous catechols by the third week *in ovo* that is not restored until the first week after hatch (5). Young broilers are undergoing a period of extremely rapid growth and may be expected to be in a state of extreme endocrine and biochemical flux. The objective of the current study was to investigate the interaction between early growth rate and age on myocardial irritability in young broiler chicken.

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Materials and Methods

This study utilized 74 male broiler chickens between 3 (group A, 21–24 days) and 6 (Group B, 43–47 days) weeks of age. The birds were all raised on a standard commercial diet with food and water supplied *ad libitum*. The two groups were from separate hatches from the same breeder flock. Each group was further subdivided at 2 weeks of age into heavy weight (HW, >425 g) and light weight (LW, <350 g) birds to assess the effect of early growth rate.

Each bird was tied to a heated surgical table in lateral recumbency with the wing outstretched. The brachial artery and vein of each bird was cannulated

with polyethylene tubing (PE60) using a local anesthetic (lidocaine HCl). Care was taken to soothe the bird by voice, touch, and hooding, as required, during the recovery. Thirty minutes later mean systemic arterial pressure (SAP) and heart rate (HR) measurements were made using a Statham pressure transducer (model P23ID; Gould, Hato Rey, Puerto Rico) and recorded with a Grass polygraph (model 7D; Grass Inc., Quincy, MA). Arterial blood gases were determined on 1.5 ml of blood using a Radiometer blood gas analyzer (model BME33/BGA3; Radiometer). The bird was then lightly anesthetized with pentobarbital (25 mg/kg iv). The thorax was then entered under the keel, avoiding the air sacs, and electrodes were attached to the right and left ventricles. The electrodes were connected to a Grass stimulator (model S44; Grass Inc.) and the electrical fibrillation threshold (EFT) was determined in response to increasing direct myocardial stimulation (300 pps, 50 msec, 1 sec stimulation, DC). The EFT was defined as the current in milliamperes requires to induce a sustained myocardial fibrillation. The EFT determination was the terminal measurement in all birds.

Results were analyzed for statistical differences between groups using analysis of variance and least significant difference. The effect of growth rate within age groups was evaluated for the EFT determination using Student's t test. Comparisons between age groups were not made. Statistical tests were performed using Crisp Interactive Statistical Package (Crunch Software Corp.). Differences were considered to be statistically significant at $P < 0.05$.

Table I. Body Weight for Birds within Each Age Group

Age (weeks)	Group A		Group B	
	LW (g)	HW (g)	LW (g)	HW (g)
2	250 ± 0.4[a] (n = 12)	375 ± 0.4[a] (n = 12)	219 ± 4.1[a] (n = 25)	425 ± 4.4[a] (n = 25)
3	490 ± 14[a] (n = 9)	750 ± 14[a] (n = 12)	—	—
6	—	—	1843 ± 82[a] (n = 20)	2199 ± 59[a] (n = 25)

[a] Significantly different within the same age group ($P < 0.05$).

Results

The weights of birds used in this study are summarized in Table I. There is no significant difference in the weights of the corresponding LW and HW groups between the Group A and Group B experimental groups at 2 weeks of age. LW and HW birds were statistically different within age groups at 2 weeks of age and this difference was sustained through 6 weeks ($P < 0.01$).

Table II presents the hemodynamic and blood gas results. There was no statistical difference between age groups or weight groups in mean SAP or HR. The Group B HW birds had a significantly ($P < 0.05$) lower arterial PO_2 (109.5 mm Hg ± 1.0, mean ± SEM) than either Group A groups (LW, 114.0 mm Hg ± 3.0; HW, 120.5 mm Hg ± 1.6), although arterial oxygenation was still above 100 mm Hg in all groups. No differences were apparent in arterial PCO_2 or pH in any group.

A total of 20 birds in Group A and 25 birds in Group B were evaluated for the electrical fibrillation threshold. Birds succumbing to respiratory failure and those which fibrillated immediately upon attachment of the electrodes to the heart prior to electrical stimulation were eliminated from the study. Group A birds were particularly sensitive to the anesthetic and this accounted for most of the difference in mortality between groups. The remaining birds were evaluated for EFT and the results are reported in Figure 1. The EFT for 3-week HW birds (400 mA ± 45) was significantly less than that of 3-week LW birds (700 mA ± 100) (Group A). There was no longer a significant difference between LW and HW birds at 6 weeks (Group B).

Table II. Effect of Growth Rate and Age on Mean SAP, HR, and Arterial Blood Gases

Variable	Group A		Group B	
	LW	HW	LW	HW
Mean SAP (mm Hg)	104.0 ± 2.5 (n = 9)	106.5 ± 4.5 (n = 12)	82.0 ± 10.8 (n = 25)	101.0 ± 8.4 (n = 25)
Mean HR (beats/min)	334 ± 16 (n = 9)	340 ± 16 (n = 12)	350 ± 15 (n = 14)	334 ± 10 (n = 19)
PaO_2 (mm Hg)	114.0 ± 3.0 (n = 8)	120.5 ± 1.6 (n = 12)	109.9 ± 4.4 (n = 19)	109.5 ± 4.4[a] (n = 25)
$PaCO_2$ (mm Hg)	28.5 ± 1.0 (n = 8)	27.9 ± 0.7 (n = 12)	26.2 ± 1.1 (n = 19)	27.2 ± 0.5 (n = 25)
pH	7.509 ± 0.130 (n = 8)	7.493 ± 0.017 (n = 11)	7.521 ± 0.020 (n = 13)	7.503 ± 0.014 (n = 21)

[a] Significantly different within the same age group ($P < 0.05$).

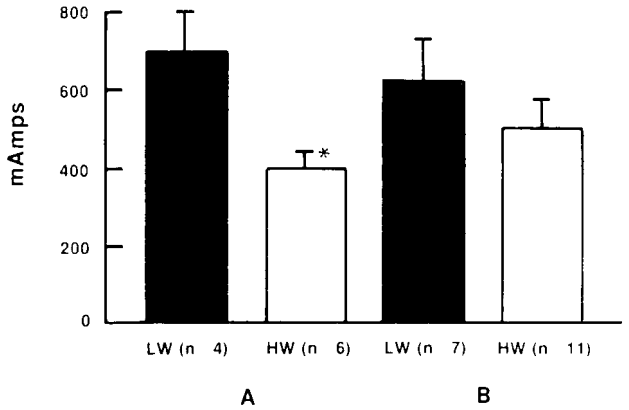

Figure 1. Effect of age and growth rate on the electrical fibrillation threshold. * Significantly different within the same age group ($P < 0.05$).

Discussion

Sudden death syndrome is a widespread disorder (2) and there is little question that the syndrome is a significant contributor to overall flock mortality (5, 6). SDS is seen in otherwise asymptomatic, well grown, healthy birds, especially males up to 5 weeks of age (2). The postmortem examination is not especially revealing. There may be some pulmonary congestion and some edema may be present (1, 7). The heart typically contains what has been shown to be postmortem blood clots (3) and the ventricles tend to be contracted, with dilated, blood-filled auricles (8). Some evidence of inflammatory cell infiltration of the heart and lungs may be apparent upon microscopic examination (9). However, this finding has been contested (4). These postmortem findings are generally consistent with agonal changes but do not rule out a cardiogenic death. However, the cause of any failure of the heart to maintain function, and the mechanisms involved, remains uncertain.

Summers *et al.* (10) have hypothesized that SDS involves a lactic acidosis similar to that seem in ruminants following carbohydrate overload. Although the results of the study do not generally support this hypothesis, the researchers propose that an acid base imbalance may be induced (10). We have been able to establish that arterial blood gas, pH, and oxygen content were consistent with normal values (11) and are not likely to account for these changes in myocardial irritibility. In addition, arterial blood pressure and heart rates, as measured in our laboratory, were similar to those reported for normal chickens (11). Julian and Leeson (14) were able to increase SDS mortality by feeding birds a high glucose diet, although the mechanism remains unclear. Some researchers have noted an increase in hepatic concentrations of copper and cardiac calcium in SDS birds (15). Increased cardiac calcium is consistent with an influx of calcium during fibrillation and heart failure. Biotin deficiency has been proposed as a causal agent in SDS (16). However, Steele and Edgar (6) have been able to establish that cardiac and liver biotin levels remain normal in birds affected with SDS and supplemental biotin also failed to alter the incidence of SDS (16). Others have found dietary micronutrients to have little effect (17).

Dietary fat has been implicated in a number of studies (17–20) where the incidence of SDS has been altered by modifying the level of fat in the diet or where body fat levels have been measured in birds affected with SDS. One potential mechanism for fat levels to influence SDS is through arachidonic acid metabolism and resultant prostaglandin levels in the heart. Rotter et al. (18) found reduced levels of arachidonic acid in the hearts and livers of SDS birds. They suggested that prostaglandin synthesis was inhibited in the hearts of SDS birds, increasing myocardial irritability. Buckley *et al.* (21) were unable to support these findings. They do, however, suggest an alternative mechanism based on fatty acid compositions in SDS cardiac tissue. They propose that an interaction of hypoxia and arachidonic acid metabolism may reduce prostaglandin synthesis in SDS.

We have looked at 3- and 6-week old birds exhibiting rapid and slow early growth. Physiologic parameters such as blood pressure, heart rate, and arterial blood gases were essentially unchanged across these groups. Arterial PO_2, although statistically lower in Group B HW than the corresponding LW group, remained well within the normal ranges (11).

Myocardial irritability as measured by EFT was significantly increased only in the Group A HW birds. This suggests that a period of increased vulnerability occurs around 3 weeks of age that is not longer present by 6 weeks. This is consistent with the observations of Riddell and Springer (2) that the incidence of sudden death syndrome in the field peaks between 2 and 4 weeks and declines thereafter. In addition, work, with the embryonic and neonatal chick heart suggests that cardiac adrenergic receptors are in a state of change during this period (5, 12). These reported findings are consistent with the hypothesis suggested by the results of the present study that there is an increased myocardial sensitivity in birds succumbing to SDS, with death due to myocardial fibrillation. The heart may be especially vulnerable to inappropriate biochemical modulation during a period of changing neural control. However, loss of adrenergic control alone cannot be invoked as a cause of SDS since dietary reserpine has been shown to have little effect on overall or SDS mortality (13).

The results of this study suggest an increased myocardial irritability in large young broilers that is no longer present in a similar group of older birds. It is

clear from the work of Higgins and Pappano (5) that neonatal chicken cardiac adrenergic receptors are in a state of flux. The rate of growth as a fraction of body weight is greater in the young Group A HW birds than the Group B HW birds. It is possible that rate of growth, rather than age, contributes most to the increased vulnerability shown in the Group A HW birds. It is possible that a nutritional imbalance alters normal myocardial sympathetic-parasympathetic control with an increased likelihood of myocardial failure. Further research is needed to determine the mechanisms involved.

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