



University of Tennessee, Knoxville
**TRACE: Tennessee Research and Creative
Exchange**

Chancellor's Honors Program Projects

Supervised Undergraduate Student Research
and Creative Work

Spring 5-1997

Athletic Heart Syndrome in the Sled Dog

Robert Francis Garza
University of Tennessee - Knoxville

Follow this and additional works at: https://trace.tennessee.edu/utk_chanhonoproj

Recommended Citation

Garza, Robert Francis, "Athletic Heart Syndrome in the Sled Dog" (1997). *Chancellor's Honors Program Projects*.

https://trace.tennessee.edu/utk_chanhonoproj/215

This is brought to you for free and open access by the Supervised Undergraduate Student Research and Creative Work at TRACE: Tennessee Research and Creative Exchange. It has been accepted for inclusion in Chancellor's Honors Program Projects by an authorized administrator of TRACE: Tennessee Research and Creative Exchange. For more information, please contact trace@utk.edu.

**Appendix D - UNIVERSITY HONORS PROGRAM
SENIOR PROJECT - APPROVAL**

Name: ROBERT GARZA

College: AGRICULTURE Department: ANIMAL SCIENCE

Faculty Mentor: DR. MASINCUPP

PROJECT TITLE: ATHLETIC HEART SYNDROME IN THE
SLED DOG.

I have reviewed this completed senior honors thesis with this student and certify that it is a project commensurate with honors level undergraduate research in this field.

Signed: X JRMasincupp, Faculty Mentor

Date: 5-12-97

Comments (Optional):

**Appendix E - UNIVERSITY HONORS PROGRAM
PLAN FOR INTERNATIONAL STUDY, WORK, AND TRAVEL**

Name: _____

College: _____ Department: _____

Faculty Mentor: _____

BRIEF DESCRIPTION OF PROPOSED INTERNATIONAL EXPERIENCE:

I have discussed the above plan with this student.

Signed: _____ faculty mentor

Date: _____

.....

ATTACH COPIES OF: Your travel schedule, addresses of institutions with which you will be affiliated, copies of relevant correspondence from host institutions.

I have met with this student and discussed the plan and attachments describing the proposed international study, work, and travel. The plan is well-conceived, and the student appears to be prepared adequately for this experience.

Signed: _____ Date: _____

Elizabeth Ousley, Advisor
Center for International Education
G102 Melrose Hall
phone: 974-3177
Email: EOUSLEY@UTK.EDU

Return this completed form to The University Honors Program, F101 Melrose Hall, 974-7875, at least 1 month prior to your departure.

Athletic Heart Syndrome in the Sled Dog

Robert Garza

May 12, 1997

University Honors Project

Athletic Heart Syndrome in the Sled Dog

R. F. Garza

ABSTRACT: Athletic heart syndrome is a medical condition common to the endurance athlete, whether human or canine. It is characterized by the presence of bradycardia, arrhythmia in the form of P wave amplitude increase, QRS wave voltage and amplitude increase, ST segment elevation, and QT segment lengthening. Also indicative of athletic heart syndrome is hypertrophy of the cardiac muscle, mostly the left ventricle and ventricular septum, and presence of systolic murmurs. These findings are concluded through the use of auscultatory means as well as electrocardiography. Causes of

athletic heart syndrome are most likely attributed to the physiologic response of the heart to endurance training, but genetics, hormonal responses, environment, and nutrition may all have an effect. Research in the field of canine athletic heart syndrome is severely lacking, and further elucidation of the causes and consequences of this medical condition in the canine endurance athlete may prove to be useful in both humans and canines in the related conditions of systolic anesthetic complications, training, nutrition, and sudden death syndrome.

Key Words: Athletic Heart Syndrome, Endurance Training, Sled Dogs

Introduction

The Siberian Husky is a breed of dog that has historically been known for its feats of endurance. The Husky has been used for centuries by humans to pull sleds in the Arctic regions of North America and Asia. Over the course of human intervention, the Siberian Husky, along with other sled dog breeds, has developed into a powerhouse of strength and endurance. This ability to run long distance inspired the modern sport of sled dog racing, which includes the Iditarod.

The Siberian Husky is a picture of the perfect sled dog athlete. This breed of sled dog pulls faster than any other and is known for its intelligence and loyalty to humans. The Husky ranges in size from about 23 to 51 kg and average a distance of over 100 miles per day. A modern race ranges from the three mile sprint

competitions to the 1,049 mile Iditarod. The dogs of the Iditarod, in particular, are ideal specimens for research. They are models of nutrition as well as models of athletic prowess. The feats of endurance of sled dogs place them in an athletic status alongside of marathon runners, swimmers, and long distance cyclists.

Human endurance athletes and canine endurance athletes have much in common. Both have to maintain a balanced diet and train frequently. Both must have a willingness to endure the physical stress of endurance events and be motivated to compete. But some of these athletes also have something else in common, a medical condition known as the athletic heart syndrome. The physical aspects of the athletic heart syndrome are comparable in humans and sled dogs. Unfortunately, little research is available on athletic heart syndrome in sled dogs, so it is imperative that the abundance of human data on this subject be applied to the canine endurance athlete.

Discussion/Results

Athletic Heart Syndrome

Human marathon runners were diagnosed with the medical condition known as athletic heart syndrome in America as early as 1968. Athletes periodically arrived at medical clinics complaining of chest pain, and doctors found symptoms indicative of heart disease. These symptoms were systolic murmurs, a slow pulse rate, arrhythmias, cardiac enlargement (upon X-ray), and abnormalities in electrocardiograms (Gott et al., 1968). However, upon catheterization and angiography, the patients were commonly found to have normal heart function (Desser et al., 1973). Thus, the need arose for further study and research into the functioning of the heart of the athlete.

It was evident that athletic heart syndrome needed to be distinguished from cardiac myopathy. The key was a thorough and accurate patient history. The history of athletic participation, particularly endurance training, became the landmark for determining the presence of the athletic heart syndrome from pathological

causes (Fecteau, 1984). Once it was known that this condition existed and was peculiar to endurance athletes, much research began on the subject. Sled dogs naturally became a topic of study for athletic heart syndrome as well.

Physical Conditions

Bradycardia. Athletic heart syndrome can be described as the physiological adaptation of the heart to endurance training. Several physical conditions, similar to those seen in cardiac care patients, are manifested in athletic heart syndrome. One characteristic is resting sinus bradycardia. Bradycardia is the decrease seen in the resting heart rate of the endurance athlete. National Football League Players are reported to have an 85% incidence of bradycardia (George et al., 1991). In one case, a teenage athlete was reported to have a heartbeat of 25 beats/min, where the normal in human non-athletes is 75 beats/min, a marked decrease of 66% (Chapman 1982).

Bradycardia is found to exist in the canine endurance athlete. Highly trained sled dogs are found to have a average resting heart rate of 102 beats/min as opposed to the average rate of the untrained sled dogs at 131 beats/min and the average rate of lightly trained sled dogs of 126 beats/min (Constable et al., 1994). The resting heart rate decreases as the level of training increases. Bradycardia occurs as the heart of the athlete develops, and is the most common characteristic of the athletic heart (Gott et al., 1968)

Arrhythmia. Another characteristic of the athletic heart syndrome is sinus arrhythmia. Both P wave and QRS wave amplitudes increase, QRS duration increases (George et al., 1991), and ST segment elevation frequently occurs in the endurance athlete (Huston et al., 1985). P wave amplitude increase are consistent with right atrial enlargement (George et al., 1991) while QRS amplitude elevations and voltage changes are consistent with left

ventricle enlargement and level of exercise. ST segment elevation is consistent with the increase in physical conditioning and is attributed to the increased tone of the muscle (Huston et al., 1985).

The arrhythmia found in sled dogs is equivocal to that found in human endurance athletes. QRS duration increased with the level of endurance training. A prolonged QT interval was only found to be present in heavily trained sled dogs and not found in untrained sled dogs, and amplitude of the QRS wave also increased as the level of training increased. No evidence is documented for change in the P wave or ST segment elevation. However, the changes in QRS duration and amplitude correlating with the increase in conditioning are proof of left ventricle hypertrophy in response to endurance training (Constable et al., 1994). These findings are indicative of the left ventricle enlargement that occurs in the athletic heart syndrome.

Hypertrophy. The heart of the endurance athlete is typically enlarged, has a greater volume than normal, and exhibits a marked increase in the diameter of the coronary arteries (Gott et al., 1968). However, there are two different kinds of exercise that can generate a change in the heart muscle, isometric and isotonic.

Isometric exercise is the exercise involving systolic pressure overload in the heart. These are the sports such as weightlifting, gymnastics, and wrestling. This type of exercise results in little or no increase in heart size or capacity, but there is hypertrophy of the left ventricular wall. The stroke volume (amount of blood ejected with each contraction) and the ejection fraction (percentage of blood ejected with each contraction) remains unchanged. There is only an increase in the thickness of the muscular walls, including the ventricular septum (Simpson and Morris, 1979). Left ventricular mass will begin to increase as early as one week after the onset of physical training (Huston et al., 1985). The more highly trained the isometric athlete, the thicker the increase of the size of the heart wall.

Isotonic exercise creates increased dimensions of the right and left ventricles with a minimal increase in wall thickness. This is seen in swimmers, runners, cyclists, and basketball players. The increase in ventricular

mass in these athletes is merely due to the increase in size of the chambers. The heart wall will thicken to proportionately match the increase in diameter of the chamber. The ejection fraction remains the same but the stroke volume is increased (Simpson and Morris, 1979). This type of exercise produces a chronic volume overload where the size of the heart chambers increase to meet the demand for sustained cardiac output. As the athlete experiences higher levels of endurance training, the heart experiences a larger increase in diameter due to isotonic training (Huston et al., 1985).

The sled dog is an example of the combined efforts of isometric exercise and isotonic exercise. Being an endurance runner, the sled dog is susceptible to isotonic conditioning in order for the heart to support the physiologic demand of long distance running. On the other hand, pulling the sled itself requires conditioning from isometric exercise whether it be the initial pull to overcome the static friction of the stationary sled or the effort that it takes to pull the sled uphill. In either case, isometric conditioning and isotonic conditioning are both necessary components of the training of the sled dog. The average weight of the mammalian heart is .59% of total body weight, while the average heart weight of the canine athlete is 1.00%. Electrocardiographic evidence also shows the left ventricular enlargement of the sled dog as well as the hypertrophy of the left ventricle and ventricular septum (Constable et al., 1994). The hypertrophy of the heart muscle, indicated by the increase in total mass of the heart and QRS duration, is due to the isometric training of the sled dog. However, most of this increase in mass is attributed to the increase in diameter of the heart and the increase in wall thickness resulting from isotonic training rather isometric training. Septal hypertrophy and ventricular hypertrophy is known to occur in both isotonic and isometric athletes. But, the heart of the isometric athlete increases only to the same degree as lean body mass (Huston et al., 1985), so the heart would not increase its percentage relative to the total body weight. The heart of the trained sled dog has been shown to have more mass than the untrained dog of similar size, breed, and shape. Thus, the heart of the sled dog illustrates the consequences of endurance training rather than that of the strength and non-endurance sports.

Murmurs. Cardiac murmurs are known to occur in 40% of young athletes (Gott et al., 1968), 93% of marathon runners, and 96% of basketball players (Simpson and Morris, 1979). Murmurs are produced by the turbulent flow of blood through the heart and great vessels. In athletic heart syndrome, murmurs are due to the increased stroke volume of blood that causes turbulent flow rather than the normal laminar flow of blood. Murmurs have been found to exist in 43% of athletes at rest. Also included in the conditions of the athletic heart syndrome are the presence of the third and fourth heart sounds, also called gallops. The first and second heart sounds are the normal sounds of the closure of the atrial and ventricular valves, respectively. The third sound is referred to as the “ventricular gallop” and is found in 93% of marathon runners, 96% of basketball players, and pediatric and adolescent age groups. It is produced by the early, rapid filling of the emptied ventricle. This sound is produced due to the large stroke volume of the athletic heart and the necessity with a more rapid flow than normal to refill it. The fourth heart sound, or atrial gallop, is also frequently heard in endurance athletes. Although normally associated with coronary artery disease and cardiac myopathy, it is heard in 71% of marathon runners and 56% of professional basketball players. The fourth heart sound is caused by the rapid movement of blood rushing in to refill the emptied atria (Simpson and Morris, 1979).

Heavily trained sled dogs have been found to have a 40% incidence of systolic murmurs. These murmurs most likely equal those found in human athletes, namely the sound from the increase in blood velocity across the aortic valve of the heart due to the increase in the stroke volume of the athletic heart. Because these dogs were free of heart disease, these murmurs are a consequence of endurance training and not a consequence of cardiac pathology (Constable et al., 1994). The presence of gallops, or the third and fourth heart sounds, has not yet been documented in the canine athlete. However, the presence of murmurs as a physiological response to heavy endurance training is evidence of the athletic heart syndrome existing in the canine athlete.

Additional Physiological Causes. There is a reported genetic link with athletic heart syndrome. One family of humans was reported to show athletic heart syndrome in three out of four children and the father. All were athletic, but none were long distance runners. Therefore, a correlation exists between family and left ventricular hypertrophy, suggesting that genetics may give a predisposition to athletic heart syndrome (Vieweg, 1975). Another study, however, concluded that the seeming correlation between genetics and athletic heart syndrome were probably due to non-cardiac features (George et al., 1991).

Endocrine factors may play a role in the development of the conditions associated with athletic heart syndrome. Acute exercise is reported to increase the levels of catecholamines, growth hormone, and testosterone in the body. What role these hormones might play in the development of athletic heart syndrome is unclear at this time. Cardiac enlargement has been shown to occur when the levels of thyroxine increase, but it is unclear if this is the mechanism that would stimulate exercise-induced cardiac enlargement. Estrogen has been shown to have an effect on cardiac growth as well (George et al., 1991). As for endocrine activities in the canine endurance athlete, little is known at this time.

Implications

Sled dogs trained for endurance events are the ideal research specimen for athletic heart syndrome. The conditions of this medical syndrome in humans are well documented, but the canine aspect of this research is severely lacking. Further research on athletic heart syndrome in both humans and canines may elucidate some of the cause of sudden death syndrome, which has perhaps falsely been attributed to athletic heart syndrome (Vanek, 1995). Sudden death syndrome has become a hot issue in regards to the sport of sled dog racing and is still an issue in human athletics. Although presently inconclusive, more study of athletic heart syndrome may

better explain the physiology of the endurance athlete. Research may also clarify the unexplained incidence of sudden asystole in anesthetized patients previously diagnosed with athletic heart syndrome (Abdulatif et al., 1987; Kreutz and Mazuzan, 1990). The endurance-trained canine heart could also reveal information related to training and nutrition of canines and other animals as well as advance knowledge in the human endurance athlete.

Literature Cited

- Abdulatif, M., M. Fahkry, M. Naguib, Y. A. Gyamfi, and I. Saeed. 1987. Multiple electrocardiographic anomalies during anaesthesia in an athlete. *Can. J. Anaesth.* 34(1): 284.
- Chapman, J. H. 1982. Profound sinus bradycardia in the athletic heart syndrome. *J. Sports Med. Phys. Fitness.* 22(1): 45.
- Constable, P.D., K. W. Hinchcliff, J. Olson, and R. L. Hamlin. 1994. Athletic heart syndrome in dogs competing in a long-distance sled race. *J. Appl. Physiol.* 76(1): 433.
- Dresser, K. B., A. Benchimol, and J. A. Schumacher. 1973. External pulse and vectorcardiographic abnormalities in the athletic heart syndrome. *Chest.* 64(1): 105.
- Fecteau, D. 1984. Athletic heart syndrome: Differentiating normal physiologic changes from pathologic changes. *Dimens. Crit. Care Nurs.* 3(3): 134.
- George, K. P., L. A. Wolfe, and G. W. Burggraf. 1991. The 'athletic heart syndrome': A critical review. *Sports. Med.* 11(5): 300.
- Gott, P. H., H. A. Roselle, and R. S. Crampton. 1968. The athletic heart syndrome: Five-year cardiac evaluation of a champion athlete. *Arch. Intern. Med.* 122(4): 340.
- Huston, T. P., J. C. Puffer, and W. M. Rodney. 1985. The athletic heart syndrome. *N. Engl. J. Med.* 313(1): 24.
- Kreutz, J. M. and J. E. Mazuzan. 1990. Sudden asystole in a marathon runner: The athletic heart syndrome and its anesthetic implications. *Anesthesiology.* 73: 1266.
- Simpson, A. G. and A. F. Morris. 1979. Athletic heart syndrome: Some recent observations. *Am. Correct. Ther. J.* 33(2): 53.
- Vanek, J. A. 1995. Sudden death syndrome in canine athletes focus of sled dog veterinary meeting. *J. Am. Vet. Med. Assoc.* 206(4): 413.
- Vieweg, W. V. 1975. Left ventricular hypertrophy in an athletic family; A variant of the athletic heart syndrome. *J. Sports Med. Phys. Fitness.* 15(2): 132.

**Appendix C - UNIVERSITY HONORS PROGRAM
SENIOR PROJECT - PROSPECTUS**

Name: ROBERT GARZA

College: AGRICULTURE Department: ANIMAL SCIENCE

Faculty Mentor: DR. MASINCUPP

PROJECT TITLE: ATHLETIC HEART SYNDROME IN THE

SLED DOG

PROJECT DESCRIPTION (Attach not more than one additional page, if necessary):

A literary research topic designed to explore the causes of athletic heart syndrome in the sled dog and its possible implications to the health of the sled dog as well as its implications in human medicine as well.

Projected completion date: 3-1-97

Signed: Robert Garza

I have discussed this research proposal with this student and agree to serve in an advisory role, as faculty mentor, and to certify the acceptability of the completed project.

Signed: X JRMasincupp, Faculty Mentor

Date: 5-12-97

Return this completed form to The University Honors Program, F101 Melrose Hall, 974-7875, not later than the end of your 3rd year in residence.

Appendix B - Sample 4 Year Honors Curriculum

This sample curriculum focuses only on elements and requirements of the honors program and does not attempt to include any requirements of individual academic majors.

Year	Fall	Spring	Summer
1	UH 117/118 1-2 honors courses*	UH 127/128 1-2 honors courses*	
2	UH338 1 honors course UH 337/347/357* Study Abroad**	UH 348 1 honors course UH 337/347/357* Study Abroad**	Study Abroad**
3	UH338 Study Abroad** Obtain Faculty Mentor	UH348 Study Abroad** Preliminary work on Senior Project	Study Abroad** Preliminary work on Senior Project
4	UH 458 Wrap up Senior Project	UH348 Submit approved Senior Project	

* 3-credit UH courses may count for all or part of the 4 honors course requirement for most students; one UH course required of Whittle Scholars during their 2nd year. One University Studies Course may be substituted for one of the 4 required honors courses.

** Optimal times to pursue international study or internship