

# Roundheart Disease:

Spontaneous Turkey  
Cardiomyopathy

Although first described and identified over 45 years ago<sup>7</sup>, we still cannot totally prevent roundheart. We do however have a better understanding of what the requirements are of the developing embryo and young poults. The condition is related to early rapid growth and the incidence is increased by lack of oxygen to the heart muscle. At some point the oxygen demand exceeds the oxygen supply. The highest mortality occurs in poults between 2 to 3 weeks of age. Losses can, on occasion, still occur up to 14 weeks of age. This disease occurs mainly in males, but an increased incidence has been reported in turkey hens<sup>8</sup>.

### Appearance of Roundheart

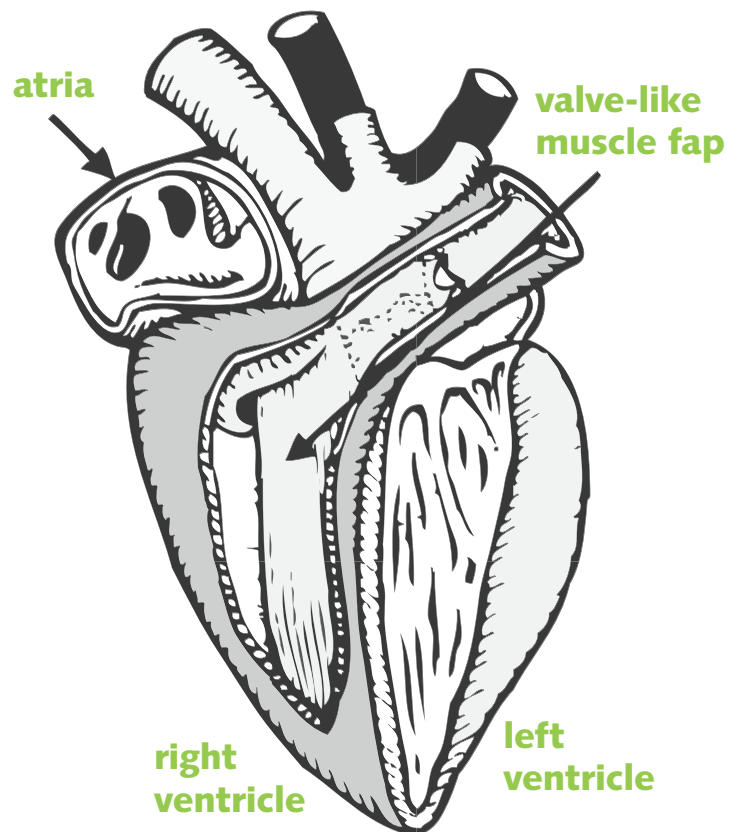
Poults which arrive at the farm appear healthy and active. By 1 to 4 weeks of age however, they have difficulty breathing and become stunted. On post-mortem the most striking feature is the large “round” heart. The ventricles or lower chambers of the heart become enlarged and flabby.

### Normal Anatomy and Physiology (How the System Works)

Birds have developed an amazing circulatory system designed to maximize oxygen extraction and delivery to all tissues. The cone shaped heart is divided into four chambers. The two upper chambers called atria function to fill and hold blood. The two lower chambers called ventricles function as pumps. When the heart contracts, the right ventricle pumps deoxygenated blood to the lungs to be oxygenated and the left pumps this oxygen rich blood to the body. The left ventricle is 4 times thicker than the right. This increased thickness ensures that the oxygenated blood reaches all tissues as quickly as possible. Birds do not have valves on the right side of the heart separating the upper and lower chambers of the heart the way mammals do. Instead they have a flap of muscle which functions like a valve. When the flap opens, blood flows from the upper chamber into the lower chambers. The flap then closes and seals the chambers. Next, the heart contracts and blood is pushed forward through the lungs to become oxygenated.

The heart muscle cells (myocytes) continue to multiply in number during the first 2 to 3 weeks of a poult's life. A lot of oxygen is required to fuel this process. The role of the myocytes is to maintain heart function (contractility). The oxygen within the bloodstream (hemoglobin) is transferred to the heart muscle cells via a carrier called myoglobin.

Why turkeys are so susceptible to roundheart is not known. However we do know that turkeys have less myoglobin than chickens and significantly less than mammals<sup>9</sup>.



## Development of Roundheart

The myocytes respond to changes in pressure and volume (the amount of work the heart has to do to pump the blood) and to lack of oxygen, toxins, infectious or nutritional insults in only a very limited way: which is to enlarge to meet the increased demand placed on them<sup>5</sup>. An increased workload or lack of oxygen eventually leads to death of the myocytes, and at this point the ventricles (bottom chambers) start to dilate. The muscle flaps or valves separating the top and bottom chambers of the of the heart cannot close completely. Consequently when the heart contracts, some of the blood will be pumped forward towards the lungs and the body like normal but some will also “backwash” towards organs such as the liver and the lungs. This causes the liver to become enlarged and congested with blood. Fluid leaks out of the liver into the abdomen. Birds lack a diaphragm and consequently any fluid in the abdomen will travel into the lungs which become wet and congested.

In addition, a much smaller volume of poorly oxygenated blood is sent to the tissues. These are the two main reasons that affected birds become stunted and have difficulty breathing

**We feel the same effects if we start at sea level and hike up into the mountains. Muscles and organs that are chronically starved for oxygen, cannot perform and, with time, begin to deteriorate.**

## Why Does It Happen?

Although the specific causes of roundheart are not well defined, one of the critical initiating events is felt to be when the oxygen supply cannot keep pace with the oxygen demand. It is uncertain whether this is the result of a deficit which occurred at one single point in time (ie: during incubation or during brooding) or is the result of cumulative insults.

### Oxygen is critical for the developing embryo at two specific times<sup>10</sup>:

- During the first five days of incubation when the oxygen conductance depends on the total pore area (size x number of pores) and shell thickness. Anything that impedes conductance such as pores being blocked by chemicals used in sanitation or cleaning or increased shell thickness as occurs in early production will affect oxygen delivery to the embryo.
- After 25 days of incubation oxygen consumption of the embryo exceeds the conductance properties of the eggshell. Gas conductance through the shell depends on loss of moisture. If this is low due to high humidity, the supply of oxygen to the embryo is not adequate.

There are several critical areas within the hatchery. Proper ventilation during the whole period of incubation is important but especially between 25–28 days. During this time the level of oxygen in the air cell drops from 21% to 14%, which stimulates pipping. Consideration should be given to use of supplemental oxygen or reducing the number of eggs per tray.

Oxygen levels are also important during processing, if poult are held overnight in the hatchery and also during transport to the farm.

**Oxygen levels are also critical during the first three weeks of brooding when poult are growing rapidly and have a high demand.**

There are many on farm factors which can contribute to insufficient oxygen reaching the heart muscle cells (ie: demand is greater than supply) such as:

1. *Brooder stoves with yellow flame exhausting carbon monoxide* – This gas binds with hemoglobin interfering with its ability to carry oxygen in the bloodstream.
2. *Nitrates present in the drinking water* also bind with hemoglobin interfering with its function.
3. *Inadequate ventilation causing high levels of carbon dioxide within the rings* – In addition to compromising the heart muscle cells, high levels of carbon dioxide cause the poults to be dopey and inactive. This is because CO2 interferes with the conversion of glycogen to glucose, the useable form of energy for the poult. It has been shown that early poult mortality increased in houses where any one or a combination of the following conditions were present: oxygen less than 20.3%, carbon dioxide greater than 2,500 ppm or carbon monoxide greater than 20 ppm 2.  
The incidence of roundheart is typically higher during the winter months when both hatcheries and brooding barns run “tighter” in an attempt to save heating costs.

**Table 1:** Effect of Poult Age and Outside Environmental Temperature on Air flow (CFM per 10,000 Turkeys) to Control Moisture<sup>4</sup>

Age in Days	Outside Temperature			
	-10°C 14°F	0°C 32°F	10°C 50°F	20°C 68°F
1	400	430	490	1000
7	825	900	1000	2000
14	1550	1725	2000	5300
21	2100	2400	2800	6500
28	3100	3500	4200	10,000

Most important in the table above is the dramatic increase in ventilation required from one week to the next and how that is amplified with increasing outside temperatures. For example: from day 1 until day 7, the ventilation rate doubles and in another 7 days, it has doubled again.

4. *Temperature fluctuations* – both excessive cold or excessive heat increases the metabolic rate and requirement of the poult for oxygen. Drops in temperature during the night can be a common contributing factor to roundheart. It must always be remembered that litter temperature is critical not air temperature. The barn should be preheated prior to poult placement so that the floor and litter are warm. For every 1.8°F or 1°C rise in body temperature there is a 10% increase in O2 required to run the same basic processes.
5. *High protein* – starter diets above 28 %.

**Table 2:** Effects of oxygen and/or protein level in the starter ration on the incidence of roundheart<sup>6</sup>.

Oxygen Level	Type of Diet	
	Slow: 17% Protein	Fast: 26% Protein
Normal: 20.26%	Base Level	3 times base level
Low: 16.3%	4 times base level	11 times base level

6. *Factors which accelerate growth* – such as lighting programs of constant 23 hours bright light<sup>1</sup> or in new barns with no disease challenge.
7. *Damage to the lungs* – Lung lesions interfere with oxygen exchange and reduce the lung surface area. Minimize lung damage via dust, ammonia and aspergillosis. Poults should not pant.
8. *Rickets* – affected poults do not breathe normally due to the pain from soft ribs.
9. *High levels of salt* in feed and/or water cause an increase in blood volume. This causes the heart muscle to work harder leading to muscle cell enlargement and eventually dilation. Be careful giving electrolytes to young poults, especially during hot weather.

Although there are many factors during incubation, processing, transportation and brooding, the basic defect leading to roundheart is not being able to provide sufficient oxygen to the heart muscle of the embryo/poult to meet its needs at that stage of development or growth. Poults with compromised heart muscle may leave the hatchery appearing normal. Not all of these poults will develop roundheart. Those exposed to additional management and nutritional stressors have a much greater likelihood of developing roundheart.

## On Farm Management Checklist to Minimize Roundheart<sup>4</sup>

- Barn pre-warmed. 24 hrs in cold weather.
- Brooder stoves running cleanly. Check color and/or CO levels.
- Check drinking water source for nitrates if previous problems
- Room temperature and ring temperatures on target prior to poult placement.
- Check minimum/maximum thermometer daily to prevent temperature fluctuations. Large swings will increase the metabolic rate of the poult and increase the need for oxygen.
- Same day placement if possible. Do what's best for the birds.
- Avoid continuous lighting program. Check time-clock.
- On problem farms monitor CO2 levels
- Rickets prevention
- Consider recirculating fans

## References

1. Classen, H.L., C. Riddell, F.E. Robinson, P.J. Shand, and A.R. McCurdy. 1994. Effect of lighting treatment on the productivity, health, behavior and sexual maturity of heavy.
2. Frame, D.D. 1991. Roundheart disease in Utah turkey flocks. Proc. 40th West. Poultry Dis. Conf, pp. 95-96.
3. Gazdzinski, P. Cuddy Feather File. Spring 1998. Spontaneous Turkey Cardiomyopathy (Round Heart Disease) Etiology and Prevention.
4. Huffman, H., P.Eng. Ontario Ministry of Agriculture and Food.
5. Julian, R.J., 1990. Cardiovascular disease. In; F.T.W. Jordan (ed) Poultry Diseases, 3rd Edn., pp. 345-353. Balliere Tindal.
6. Julian, R. J., S. M. Mirsalimi, L.G. Bagley, and E. J. Squires. 1992. Effect of hypoxia and diet on Spontaneous Turkey Cardiomyopathy (Round Heart Disease) . Avian Diseases 36 : 1043-1047.
7. Magwood, S. E., and D. F. Bray.1962. Disease condition of turkey poults characterized by enlarged and rounded hearts. Can. J. Comp. Med. Vet. Sci. 26 : 268-272.
8. McCausland Consulting Services. 1992. Spontaneous Cardiomyopathy in Turkey Flocks. Proc. Poultry Serviceman's Workshop. Lake Louise Alberta. pp. 1-5.
9. O' Brien, P. J., M. O'Grady, L.J. McCutcheon, H. Shen, L. Nowack, R. D. Horn, S. M. Mirsalimi, R. J. Julian, E. A. Grima, G. W. Moe, and P. W. Armstrong. Myocardial myoglobin deficiency in various animal models of congestive heart failure. J. Mol. Cell. Cardio. 24 : 721-730. 1992.
10. Rahn, M. 1981. Gas exchange of avian eggs with special reference to turkey eggs. Poultry Sci. 60 : 1971-1980.

### © Hybrid Turkeys

Unless otherwise specified, the information provided here is the property of Hybrid Turkeys. Before reproducing or publishing this material in any manner, please obtain approval by contacting the Hybrid Turkeys' head office in Kitchener, Ontario, Canada.

[info.hybrid@hendrix-genetics.com](mailto:info.hybrid@hendrix-genetics.com)

[www.hybridturkeys.com](http://www.hybridturkeys.com)