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University of Alberta

Growth Performance, Carcass Characteristics, and the Incidence of Ascites in Broiler Chickens in Response to Short Term Feed Restriction, Temperature Fluctuations, and Litter Oiling.

by

Ruth Helen McGovern ©

A thesis submitted to the Faculty of Graduate Studies and Research in partial fulfillment of the requirements for the degree of Master of Science

in

Animal Science

Department of Agricultural Food and Nutritional Sciences

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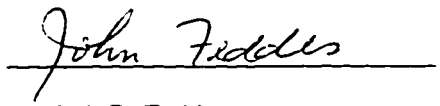

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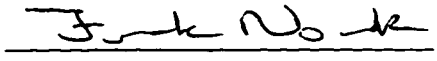

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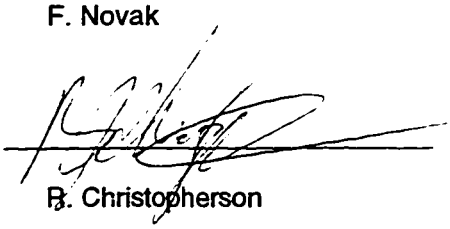

B. Christopherson

ABSTRACT

The ascites syndrome currently seen in fast growing broiler chickens is associated with an increase in the weight, volume, and area of the right ventricle of the heart. Three experiments were conducted to determine: 1. the effects of feed restriction and litter oiling on the growth performance, carcass characteristics, and the incidence of ascites, 2. the consistency and repeatability of using image analysis technology to quantify right ventricle area for assessment of the degree of right ventricular dilation, and 3. the effects of temperature fluctuations and litter oiling (for the removal of respirable airborne dust particles) on the growth performance, carcass characteristics, and the incidence of ascites.

In the first experiment, the effects of feed restriction and the removal of airborne respirable dust on growth performance, carcass traits and the incidence of ascites was evaluated with 800 male broilers studied in two 6 wk periods. Feed restriction reduced the incidence of ascites, but also reduced gain. Removal of respirable dust reduced the RVA, but did not reduce mortality. In the second experiment, determination of the RVA appears to be an accurate method of assessing the severity of ascites, if the placement of the heart slice is standardized with each cross section of heart scanned atria side up with natural ventricular positions. In the third experiment, the effect of temperature fluctuations and the dust removal to broiler straw litter to remove airborne respirable dust on growth performance, carcass characteristics, and the incidence of ascites was evaluated with 1200 male broilers studied in two 6 wk periods. Litter oiling to remove respirable dust at high levels increased the percentage of mortality due to ascites, but did not effect the overall mortality. Diurnal temperature fluctuations caused higher mortality in broilers and had significantly larger RVWT.

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1. INTRODUCTION

1.1 Background

1.1.1 Introduction

Ascites has caused significant mortality in broiler chickens raised at high altitudes for many years. Ascites in broiler chickens is characterized by accumulation of fluid in the abdomen which is caused by a cascade of events related to the need to supply high levels of oxygen to the tissues. The incidence of ascites caused by right ventricular failure in broiler and roaster chickens in Canada, England, the United States, and other areas of the world where broilers are grown at low altitude, has increased over the past several years and coincides with a continuing genetic and nutritional improvement in feed efficiency and rate of growth (Julian *et al.*, 1986). Maxwell and Robertson (1997) found, in the World Broiler Ascites Survey taken in 1996, information on 18 countries (50% poll) from four continents, the average incidence of ascites was 4.7% across the globe. Canadian condemnation records indicate that the incidence of ascites at processing plants between 1986 and 1994 has risen from 3.5% to 19%, respectively.

Deaths due to ascites have been estimated recently to cost the poultry industry in excess of $100 million annually in the United States and $500 million globally. With a total world broiler placement of approximately 12.9 billion broilers and a conservative estimate of 4.2% incidence of ascites in 21 countries, the losses due to ascites, for almost 560 million ascitic birds at a wholesale cost, according to last year's prices, total about one billion US dollars worldwide.

The incidence of ascites at moderate and low altitudes has increased in the 1970s and there was marked increase in the 1980s. The incidence at high altitudes has also continued to increase. These increases in the incidence of ascites has led to many reports describing the lesions and discussing the possible causes of the ascites syndrome. Research on ascites syndrome has been increasing over the past 10 years and is now carried out in many parts of the

world (Julian, 1993). Research has shown that the accumulation of fluid in the peritoneal cavities is the result of increased intravascular pressure in the portal system of the liver and capillaries of the organs in the abdominal cavity. This portal hypertension arises from right ventricular valvular insufficiency resulting from right ventricular hypertrophy (RVH), which is a response to pulmonary hypertension (Julian, 1993).

1.1.2 State of the Broiler Industry

A number of specialized poultry breeding companies have been in existence since the late 1940s to early 1950s, breeding chickens for meat consumption. Over that period of time, the age to slaughter and the amount of feed required to produce a given quantity of chicken meat has been more than halved, and in the United States per capita consumption of broiler meat has risen from about 4.0 kg in 1950 to over 31.8 kg in 1990. The body weight of broilers (i.e., the average of both sexes) has increased from about 2 000 g at 56 d in 1976 to about 3 100 g in 1991. Thus, 56 d broiler body weight (BW) has increased over that 15 yr. period by approximately 1 100g (73 g/yr.). Because broiler flock weights increase by about 65 to 70 g /d during the normal marketing ages, this increase has effectively reduced marketing age to a given BW by about 1 d/yr. over that time frame. With the advent of further processing, however, market weights in the United States have been steadily increasing, so the actual marketing age has decreased somewhat less than 1 d/yr. over that period. The increase in BW, probably 85 to 90% has come about due to genetic selection for increased BW, applied by commercial broiler breeding companies.

1.1.3 Recent Trends

Meat-type chickens are selected for growth rate and muscle mass, which have increased up to 5% per year over the past 30 years. These birds have also been selected for feed conversion, so that the digestive system and muscle mass have developed at the expense of the

heart and lungs (Julian, 1990). As a percentage of the body weight, the lungs of a meat-type chickens are smaller than those of other chickens (Julian, 1989). Breeding for increased muscle mass has not led to a concomitant increase in the size of the heart and respiratory system, and so the modern broiler has increasing difficulty maintaining oxygen demand for maintenance and growth (Shlosberg *et al.*, 1991).

One long-term solution for the ascites syndrome is breeding for resistance to ascites, a trait that can be bred in parallel with other desirable traits. Short-term solutions involve changes in management to minimize losses. Factors such as feed restriction, avoiding excessive exposure to cold, minimizing levels of dietary sodium, and having with an awareness of the hazards of raising broilers at high altitudes are some of the management strategies which help the producer avoid severe losses to the ascites syndrome (Shlosberg *et al.*, 1991).

1.2 The Ascites Syndrome

1.2.1 Introduction

Ascites is also referred to as Edema Disease, Pulmonary Hypertension, Congestive Heart Failure, and Water Belly. Ascites in broiler chickens is characterized by accumulation of fluid in the abdomen which is caused by a cascade of events related to the need to supply high levels of oxygen to the tissues. Fluid in the abdomen is in part plasma and lymphatic fluid that has seeped from the liver, and this occurs as the end result of a cascade of events ultimately triggered by oxygen inadequacy.

For several reasons, the need to provide more oxygen to the tissues leads to increased heart stroke-volume, and ultimately to hypertrophy (a morbid enlargement) of the right ventricle. Such heart hypertrophy, coupled with malfunctions of the heart valve, leads to increased pressure in the venous supply, and to pressure build-up in the liver, and often the characteristic fluid leakage (Leeson *et al.*, 1995).

While the events leading up to the onset of clinical signs of ascites have been fairly well established, the exact reasons for cardiopulmonary insufficiency have not been adequately quantified. Because a number of major factors can influence the degree of oxygen demand by the bird, the treatment and/or prevention is not straightforward. The most obvious solution is to reduce growth rate, thereby reducing the major oxygen demand by the tissues, although this has obvious adverse economic implications, and hopefully will be considered as a short-term solution to this major industry problem (Leeson *et al.*, 1995).

1.2.2 Definition

Ascites is an increase in the amount of lymph (fluid) normally found in the peritoneal spaces. Lymph is a clear, colorless liquid somewhat similar to blood plasma from which it is derived. Lymph may contain a few red blood cells and numerous lymphocytes as well as inorganic salts, glucose, nonprotein nitrogenous substances, and some proteins. The quantity of protein in lymph is considerable less than in plasma, but the content of simple chemical substances, crystalloids, is about the same. Birds have eight coelomic cavities. Depending on the cause of the ascites the greatest quantity of fluid is usually found in the ventral hepato-peritoneal spaces, cardiac coelomic space (pericardial sac), and intestinal peritoneal space. Fluid may also be found in the right dorsal hepato-peritoneal space. Very small amounts of fluid may be found in the coelomic spaces surrounding the lung, particularly in birds with lung edema, but these are narrow spaces and tend not to become distended (Julian, 1987). The normal amount of fluid in the coelomic cavities is not recorded but, except for the pericardial sac, any visible accumulation is likely to be abnormal. Normal meat-type chickens frequently have 1 to 3 mL of fluid in the pericardial sac at 6 to 8 weeks of age but any quantity over 4 mL is likely to be abnormal (Julian, 1993).

1.2.3 Diagnosis

It is likely that ascites will be a problem in any fast-growing bird with a high oxygen demand. Ascites is associated with hypertrophy of the right ventricle and an imbalance in the right-left ventricle weight. Heart morphology and the evidence of abdominal fluid are necessary for adequate diagnosis. Depending upon environmental conditions, ascites is most commonly diagnosed at 4-5 wks of age, although conditions have been recorded in day-old birds (Muirhead, 1987) and there are even suggestions that ascites may be due to the oxygen status of embryos during incubation (Maxwell *et al.*, 1987).

Ascitic birds are often seen panting even though there is no apparent heat stress. At necropsy the skin and tissues are congested and may be red or quite dark. Older birds may show cyanosis (blue coloring), especially around the comb and wattles and death is seen to occur spontaneously, especially when birds are excited. Panting is likely the result of abdominal fluid accumulation causing physical restriction of the large abdominal air sacs (Figure 1-1), and a reduction in tidal volume per respiration. Gurgling sounds often accompany panting. The abdomen is dilated with fluid in chickens that have an increased respiration rate and reduced exercise tolerance (Julian, 1993).

Opening of the abdominal cavity reveals amber or clear fluid (lymph) that resembles plasma. Wideman (1988) suggested that the presence of clotted plasma proteins on the surface of the liver indicates this to be the origin of the fluid. The liver may be swollen and congested or firm and irregular with edema and have fibrin adherent to the surface. It may be nodular or shrunken, it may be white with edema under the capsule and have a thickened capsule. For whatever reason, plasma that is normally held within the low-pressure liver venous system is unable to return to the heart in sufficient volume. Classically, the right ventricle is grossly dilated, and can reach 40% of total ventricle weight, compared with a normal value of 20%. Varying degrees of lung damage are seen, most often the lungs appear to be pale or gray (Leeson *et al.*, 1995). The lungs are extremely congested and edematous (Julian, 1993).

1.2.4 Physiology

1.2.4.1 Basic Series of Events Leading to Ascites

Ascites is not a disease, it is a condition in which the body cavity accumulates ascitic fluid, leading to carcass condemnation or death. Typically, the series of events that lead to the development of the ascites syndrome begin when broilers first start to gain body weight or muscle mass. Rapid growth rate in conjunction with high basic metabolic rate create an increased demand for oxygen leading to an oxygen deficit. Increasing the demand for oxygen may exceed the cardiopulmonary capacity to supply sufficient oxygen (Witzel *et al.*, 1990). At this point, there is an increase in exertion of the heart in an attempt to meet the oxygen demand of the body. Cardiac output is increased to increase blood flow. Increased pulmonary arterial pressure is due to the increased blood flow and often an increased resistance to blood flow. Right ventricular hypertrophy and dilation are the result of the increased workload, which in turn leads to valvular insufficiency, right ventricular failure, liver congestion, edema, and ascites (Julian *et al.*, 1986) (Figure 1-2).

1.2.4.2 Four Physiological Changes causing Fluid Accumulation

Ascites is a sign or lesion that may result from one or more of four physiological changes that cause an increased production or decreased removal of peritoneal lymph (Julian, 1990).

Ascites may result from:

1) obstruction of lymph drainage.

Blockage of lymph drainage prevents the return of fluid to the blood vascular system by the lymph channels. The lymphatic system includes both the lymphoid tissue of the body and the lymphatic vessels associated with the lymphoid tissue. It serves as a

system for draining tissue fluid that parallels and augments the venous circulation and therefore assists in the control of interstitial fluid pressures (Figure 1-3).

The smallest lymphatics begin blindly between cells as lymphatic capillaries, which collect the tissue fluid that is not absorbed by the venous system. When the tissue fluid enters the lymphatic vessels, it is known as lymph, which consists of fluid originally derived from the blood and is on its way back to the blood (Sturkie, 1986).

The lymphatic capillaries form complex networks throughout most tissues. These networks finally combine to form small lymphatic vessels. All of the lymph collected from the body eventually returns to the venous system by way of the thoracic duct, right ventricular duct (if present), and tracheal ducts. These ducts enter the cranial vena cava or jugular veins (figure 1-4) (Sturkie, 1986).

In mammals, neoplasia (new or abnormal body tissue) is the main cause of obstruction of the thoracic duct or other lymphatics. Increased venous pressure at the outlet of the thoracic duct may also impair lymph drainage. Broiler chickens with right ventricular failure from pulmonary hypertension develop ascites very rapidly, possibly because of interference with lymph return by high venous pressure where the thoracic duct opens into the vena cava (Julian *et al.*, 1989).

2) decreased plasma oncotic pressure.

Decreased vascular oncotic pressure allows fluid to move out of vessels more easily. Capillaries are tiny tubes with thin-walled vessels which are only large enough in diameter to accommodate a single file of erythrocytes. The wall acts as a selectively permeable membrane that permits water, oxygen, and nutrients to leave the blood for tissue cells and permits waste products from tissue cells to enter the blood (Sturkie, 1986).

Much of the fluid that passes out of the capillaries into tissue spaces again returns to the blood by passing back through the capillary walls. Some fluid remains in the tissues as tissue fluid, and the excess fluid normally is removed by lymph vessels (Figure 1-5). Larger connections called arteriovenous anastomoses or shunts permit

more blood to flow through the capillaries alone. This increased blood flow aids in sudden necessary shifts in blood volume as in increasing oxygen loading at the lungs. The speed at which the heart works affects blood pressure. As the heart rate increases during exercise, blood pressure increases (Sturkie, 1986).

Fluid moves in and out of the capillaries in order to keep the surrounding tissues fresh, and supplied with gases and nutrients. Red blood cells are too large and inflexible to freely move between the interior of the capillary tube and the exterior tissues. The red blood cell is transporting oxygen atoms and must release the oxygen into the plasma before it rushes back to the heart and lungs to pick up more oxygen.

Fluid moves into and out of the capillaries with the help of two types of pressures. Hydrostatic pressure is caused by the heart pushing on the fluids in the blood vessels resulting in fluids being pushed out of the capillaries. Osmotic (oncotic) pressure pulls fluid back into the capillaries as water moves from an area of high concentration in the tissue to an area of low concentrations that now exists in the capillaries.

Compared with mammals, birds have low concentrations of plasma protein. Plasma protein concentration is lower in newly hatched birds than adult birds and is directly related to dietary protein (Julian, 1993). Plasma protein and plasma albumen are lower in young broilers than in leghorns on the same diet (Bowes *et al.*, 1989). Except for the ascites of cachexia (a morbid state of the body system, the result of disease or of intemperate habits) there are no specific examples, in birds, of ascites caused by alterations in oncotic (similar to osmotic but includes colloidal effect) pressure. The loss of high protein lymph from the liver in ascites may lower plasma protein and increase the outflow of lymph. Broilers with ascites from right ventricular failure have lower plasma protein than normal broilers (Julian, 1993).

3) increased vascular permeability.

Vascular damage allows high protein fluid to leak out of the capillaries (mainly from the liver). Endothelial injury increases vascular permeability and allows fluid plasma protein to escape. This changes the oncotic balance and draws fluid into the

area. Free radicals damage the endothelium as do a variety of bacterial infections, chemical toxins, and viral infections (Julian, 1990). Endothelial damage may result in the accumulation of a small quantity of fluid in affected tissues or peritoneal spaces (Julian, 1991).

4) increased hydraulic pressure in the blood vascular system.

The term hydraulic is used to describe changes that occur as the result of liquid in motion. Increased fluid pressure in the capillary bed and sinusoids of the liver, or other permeable parts of the vascular system, is the most important cause of fluid accumulation in tissue and peritoneal spaces (Julian, 1990). Increased vascular hydraulic pressure may be involved in forcing fluid out or may prevent reabsorption of tissue fluid, as occurs in portal hypertension following right atrioventricular valvular insufficiency (Jordan, 1990). Intravascular pressure can rise because of increased blood-flow or increased resistance to flow, but interference with venous return is the common cause.

1.2.4.3 Increased Hydraulic Pressure

Ascites may be caused by several mechanisms, but in mammals and birds the most common cause is increased hydraulic pressure in the vena cava and portal system as the result of right ventricular failure. The two main causes of increased hydraulic pressure are liver damage and valvular insufficiency and right ventricular failure

1.2.4.3.1 Liver damage causing increased hydraulic pressure and ascites

Ascites can be the direct result of liver damage rather than right ventricular failure. Liver damage, causing the interference with venous return, and portal hypertension are reported to result in pulmonary hypertension in rats and humans. Although the mechanism is not

understood, it may be induced by chemical mediators inducing vasoconstriction in the lung (Julian, 1993).

The sinusoidal system of the liver is closely associated with the peritoneal spaces. Liver edema results in ascites. Liver fibrosis obstructing efferent hepatic vessels and increasing sinusoidal pressure is the most common cause of ascites in humans. Right ventricular failure with increased venous pressure is the most common cause in animals and birds (Julian, 1990). Early right ventricular failure results in edema, swelling and mild nodular changes in the liver. Chronic right ventricular failure results in liver atrophy and fibrosis, it is important that both the heart and liver are examined to determine the pathogenesis of ascites caused by increased hydraulic pressure (Julian, 1993).

1.2.4.3.2 Valvular insufficiency and right ventricular failure causing increased hydraulic pressure and ascites

Heart disease resulting in increased venous pressure can be divided into:

(I.) primary right heart or valvular disease and

(II.) right ventricular failure secondary to increased pulmonary arterial pressure (pulmonary hypertension).

(I.) Primary heart and valvular diseases.

These include three types: (1) congenital heart diseases,

(2) right atrioventricular valve lesions, and

(3) degenerative cardiomyopathies (Julian, 1993).

(1) Congenital heart diseases such as intra-atrial and intra-ventricular septal defects which result in left to right shunts, cause heart failure by the same mechanism as pulmonary hypertension. Congenital heart defects result in a low

incidence of ascites in broiler chicks usually within the first 2 weeks (Julian, 1990).

(2) Right atrioventricular valve lesions most frequently include valvular endocarditis (an inflammatory disease of the heart, ending in the deposit of fibrin upon the valves) (Julian, 1990) which may occur secondary to infection. Ascites caused by increased venous pressure from valvular insufficiency because of right atrioventricular valvular endocarditis occurs sporadically in broiler chickens, and in one survey of mortality in England caused 4.5% of the ascites cases. Endocardiosis of the right atrioventricular valve has been reported in normal birds and in ascites from right ventricular failure but in these cases it was probably secondary to right ventricular dilation and hypertrophy and was not the cause of valvular insufficiency (Julian, 1993).

(3) Degenerative and dilatory cardiomyopathies include spontaneous turkey cardiomyopathy (round heart disease), and other toxic, autoimmune, nutritional, and infectious cardiomyopathies, which result in ascites when the right ventricle is affected and right ventricular failure occurs (Julian, 1993).

(II.) Right ventricular failure secondary to increased pulmonary arterial pressure (pulmonary hypertension).

The second category of heart disease resulting in increased venous pressure is that associated with ascites caused by pulmonary hypertension-induced right ventricular failure. This results in the pulmonary hypertension syndrome (PHS).

Recent work on right ventricular failure and ascites caused by pulmonary hypertension in meat-type chickens indicates that right ventricular hypertrophy (RVH), right ventricular failure and ascites are a response to increased workload by the right ventricle as the result of pulmonary hypertension (Julian, 1987). Hypertrophy of the right ventricular wall is directly related to pulmonary hypertension and the ratio of the right ventricle to the total ventricle mass can be used as a measure of the increased pressure

load on the right ventricle. There are a number of possible causes of pulmonary hypertension (Julian *et al.*, 1986) (Figure 1-6), but fast-growing roasters and broilers may have a sufficiently high oxygen requirement that primary or spontaneous pulmonary hypertension may develop because of insufficient blood capillary capacity in their lungs (Julian *et al.*, 1987). Primary pulmonary hypertension is defined as occurring in the absence of any known organic dysfunction. Secondary pulmonary hypertension occurs in hypoxaemic (deficient oxygenation of the blood) broilers, in broiler with hypervolaemia or in broilers with lung pathology (Julian, 1987).

Primary Pulmonary Hypertension

If growth is slowed by restricting feed or by feeding mash, fewer cases of ascites will develop, probably because of the reduced metabolic rate and lower oxygen requirement. The right ventricular hypertrophy that preceded right ventricular failure-ascites in broiler and roaster chickens at low altitudes is the result of pulmonary hypertension. Primary pulmonary hypertension has not been reported in birds or mammals, and although it is recognized in people, the cause is unknown (Julian *et al.*, 1986).

1.2.4.4 Blood Pressure

Blood pressure is directly influenced by five factors:

1. elasticity of the artery,
2. diameter of the artery,
3. viscosity of the blood,
4. heart rate, and
5. volume of blood.

Arteries which are flexible and pliable maintain a lower blood pressure. Lower blood pressure reduces the deterioration of the organs. High blood pressure is accompanied with the hardening of the arteries. The diameter of the arteries can cause higher or lower blood pressure. For example, adrenaline causes vasoconstriction and higher blood pressure. Thicker blood tends to resist flowing. The resistance to flow causes the heart to work harder and causes an increase in blood pressure. The speed at which the heart works affects the blood pressure. When broilers are stressed, the heart rate increases and the blood pressure increases. A dramatic change in the volume of blood can cause a change in blood pressure. If blood volume is increased by drinking fluids then blood pressure may also increase (Kilback, 1990).

1.2.4.5 Pulmonary Hypertension Factors

Right ventricular failure may be caused by developmental heart disease, degenerative or inflammatory disease of the myocardium or valves, or acquired defects. Of these, the response of the right ventricle to increased partial pressure would appear to be by far the most frequent in broiler chickens. Increased partial pressure may result from increased pulmonary blood flow, organic vascular obstruction, pulmonary vasoconstriction, and chronic left atrial hypertension. Pulmonary hypertension is likely caused by a combination of factors, which can be divided into two major categories, increased blood-flow and increased resistance to blood-flow (Figure 1-7).

Increased Blood Flow

- increased oxygen requirement
- incomplete oxygen saturation of hemoglobin
- anaemia
- increased blood volume
- intraventricular and internal septal defects

Increased Resistance to Blood Flow

- polycythaemia
- increased red blood cell rigidity
- megalocytosis
- pulmonary vascular disease
- emboli obstructing vessels
- endothelial cell hypertrophy
- interstitial edema

1.2.4.5.1 Increased Blood Flow

The most important cause of increased blood flow (cardiac output) is increased body oxygen requirement resulting in hypoxia which occurs, for example, with exercise in human and animal athletes and in which both heart-rate and stroke-volume combine to increase blood-flow (Julian, 1990). There is now sufficient evidence to confirm that pulmonary hypertension syndrome is related to metabolic oxygen requirement at both high and low altitudes and that anything that increases the oxygen requirement increases the incidence of ascites caused by pulmonary hypertension (Julian *et al.*, 1987).

It has frequently been suggested in poultry industry literature that hypoxia resulting from lung damage caused by noxious fumes from poor ventilation, respiratory disease and stress are the cause of the increased incidence of ascites (Maxwell, 1990; Wideman, 1988). Main contaminants that irritate and damage the lungs are dust and ammonia (Leeson *et al.*, 1995). However, there is no evidence from research that noxious fumes or stress cause ascites (Julian, 1993).

Hypoxia may be a common cause of increased partial pressure in broiler chickens and is easily explained by increased pulmonary blood flow, organic vascular obstruction (due to an increased hematocrit), or vasoconstriction in broilers raised at high altitude, where there is

reduced oxygen tension or in experimentally induced hypoxia caused by reduced air capillary ventilation in broilers with severe rickets (Julian *et al.*, 1986).

Incomplete oxygen saturation of hemoglobin is the most important cause of pH at high altitude, but increased blood-flow because of tissue hypoxia from hypoxic-hypoxaemia is not likely to be the major reason that pulmonary hypertension occurs.

Anaemia reduces the oxygen-carrying ability of the blood and would result in increased blood-flow. However, it would also reduce growth rate and oxygen requirement.

Increased blood volume may be a major factor in some forms of pulmonary hypertension syndrome. High dietary salt increases blood volume and may be the cause of pulmonary hypertension induced by NaCl, but salt also increases RBC rigidity which increases resistance to flow. Sodium causes hypertension-induced left ventricular hypertrophy in people.

The right ventricle of birds has developed as a volume pump, not as a pressure pump, since it rarely has to respond to changes in pressure. It responds very rapidly to an increased workload. The right ventricle wall is quite thin, with a normal ratio of 1:4 to the left ventricle, and it gives the appearance of being attached to the side of the left ventricle, extending for only about 3/4ths of the length of the left ventricle. The right ventricle has very little chamber if the heart of a previously healthy bird is allowed to go into rigor before being opened. As a result there is normally no space at the level of the free edge of the right atrio-ventricular valve when the heart is trandected.

The right atrio-ventricular valve in birds is formed mainly by a continuation of the muscle of the right ventricular wall, and when the wall muscle hypertrophies, which it does very rapidly in response to increased partial pressure, the valve muscle hypertrophies to the same extent (Julian *et al.*, 1986). This thickening of the valve may interfere with its effectiveness and lead to rapidly developing valvular insufficiency and ascites. Alternatively, dilation of the right ventricle may be enough to cause valvular insufficiency. Broilers that develop right ventricular failure-ascites stop growing, and the longer they survive the less they weigh compared with their pen-mates.

1.2.4.5.2 Increased Resistance to Blood Flow

Polycythaemia causes increased blood volume and viscosity and markedly increases the resistance to blood flow through the lung. Polycythaemia is induced by hypoxic conditions and cold (although it is not clear if it does this by causing hypoxaemia or by reducing plasma volume) (Julian, 1993).

Blood flow dynamics are markedly affected by erythrocyte deformability. Because of the small blood capillary size and its lack of ability to expand to accept increased flow, reduced RBC deformability may be a significant cause of increased resistance to flow and pulmonary hypertension in the lungs of meat-type chickens. The RBCs of meat-type chickens are more rigid than those of leghorn chickens (Julian, 1993). Increased corpuscular hemoglobin concentration, increased dietary NaCl, and blood parasites reduce RBC deformability (Julian, 1993).

Megalocytosis is an increase in red blood cell size. Increased red blood cell size has been suggested as a cause of increased resistance to flow (Julian, 1987), but this has not been confirmed. Erythrocyte hemoglobin concentration increases with age as cells become smaller and less deformable. Young broiler chickens have more large red blood cells than leghorns (Julian, 1990) probably because the increased demand of rapid growth results in a high proportion of young cells. The deformability of large abnormal and large young avian red blood cells has not been investigated (Julian, 1993).

Pulmonary arteriole vasoconstriction may play a significant role in increasing vascular resistance to blood flow through the lung in hypoxic conditions in birds as it does in mammals. The effect is much more pronounced in some mammals than others, but the definitive evidence for hypoxia-induced vasoconstriction is lacking in birds (Julian, 1993).

Increased numbers of cartilaginous and bony masses have been reported in the lungs of broilers with ascites. Large numbers of these nodules could obstruct blood-flow but there is no evidence that the increase number are the cause of pulmonary hypertension syndrome (Julian, 1990). It is more likely that the physiological changes associated with pulmonary hypertension

induced hypoxia in the lung favor the development and retention of nodules that would have been removed in normal birds (Julian *et al.*, 1989).

The endothelial cells in birds are phagocytic and can be activated by biological and chemical materials (Julian, 1993).

It is frequently stated that lung damage is the cause of the increased incidence of ascites in broiler chickens but there is little evidence to support this theory. Since injury to the respiratory system slows growth, the effect on oxygen exchange or blood-flow would have to be severe before pulmonary hypertension would occur. In birds, the lung is more rigid than in mammals and blood capillaries are unable to expand significantly (Julian, 1993).

1.2.4.6 Right Ventricular Hypertrophy

The right ventricular failure that results in ascites is preceded by hypertrophy of the right ventricular wall and dilation of the chamber and frequently by thinning of the left ventricle wall. Increased pulmonary arterial pressure (partial pressure) can be produced in mammals and birds by the hypoxia of high altitude. In mammals, hypoxia provokes pulmonary vasoconstriction, which increases the resistance to flow and results in increased pulmonary vasoconstriction in fowl and ducks (Black *et al.*, 1980).

Right ventricular hypertrophy (RVH) is a response to an increased workload and eventually leads to right ventricular failure if the volume or pressure load persists. Hypertrophy of the right ventricular wall can be directly related to partial pressure, and the ratio of the right ventricle to the total ventricle mass can be used as a measure of the increased pressure load on the right ventricle. The hypoxia of high altitude that leads to increased partial pressure, right ventricular failure, and ascites is more common in broiler chickens than leghorns and in male broilers than female broilers (Julian *et al.*, 1986).

Some broilers that die from right ventricular failure have marked RVH with little dilation and little or no ascites. These broilers are of normal size for their age and are frequently found

dead on their back. Most chickens that die from right ventricular failure have marked dilation of the right ventricular chamber with hypertrophy of the wall, but occasionally there may be thinning of the wall, particularly in older chickens (Julian *et al.*, 1986). At high altitude, growing broiler chickens are more susceptible to the partial pressure that leads to the right ventricular failure than leghorns. Males are more susceptible than females, probably because of their higher metabolic requirement for oxygen (Julian *et al.*, 1986).

1.2.5 Heart Morphology

The heart of birds has a thick-walled left ventricle and thin-walled right ventricle (Figure 1-8). The right atrioventricular valve is composed of a muscle flap made up mainly of muscle fibers from right ventricle wall. The anatomy of this valve makes it very susceptible to valvular insufficiency (Julian *et al.*, 1987). The thin right ventricle responds very rapidly to increased workload by dilation (stretch) and hypertrophy (response to stretch). When the right ventricle wall hypertrophies, it leads to valvular insufficiency and right ventricular failure (Julian, 1987).

1.3 Management

1.3.1 Feed Restriction

An especially stressful period in the life of a broiler is during the first three weeks of life. Muscle and bone growth (somatic growth), expressed as a percentage of body weight, is greatest during this time. Feed restriction may slow somatic growth, allowing it to remain in phase with the functional growth of organs in the cardiopulmonary and renal systems. In addition, slower growing birds have reduced oxygen needs. By slowing the rate of gain during the starting period, the cardiopulmonary systems of the bird may not be taxed as severely as they might be by slowing the rate of gain during the finishing period. Enlargement of the right ventricle, and

subsequent physiological abnormalities resulting from the rapid growth rates are thus avoided. Birds may be able to enter the grower and finisher periods with stronger cardiopulmonary systems (Arce *et al.*, 1992).

The efficacy of the various feed-restriction regimes may be linked to the fact that broilers have a maximum ratio of feed intake per unit of metabolic weight at 10 to 20 days of age (Albers *et al.*, 1990). At that age, broilers need maximum amounts of oxygen to metabolize feed, particularly fats. Therefore, if uncontrolled feeding is permitted, they are prone to develop anoxemia, thus starting the chain of physiopathological events leading to ascites. It is interesting that feed restriction leads to inhibition of adipocyte proliferation and decreased fat deposits (Cartwright *et al.*, 1986), which theoretically should lead to increased metabolism of ingested fats by oxygenation and a greater susceptibility to ascites.

Feed is the most expensive item in broiler production. One method to reduce this cost is to restrict feed intake early in the life of the birds. Restricting feed has been initiated in an attempt to reduce not only the feed cost, but also to reduce the amount of fat deposited in broilers before market age. Acar *et al.* (1995) demonstrated that birds maintained optimum body weight, had a reduced fat deposition, and improved feed efficiency following an early feed restriction. Birds can recover from growth deficit resulting from a limited nutrient intake ("compensatory" or "catch-up" growth) (Acar *et al.*, 1995). Feed restriction is beneficial, not only because it reduces the feed costs, but because feed restriction can also reduce mortality due to ascites and leg problems.

1.3.1.1 Methods

Manipulation of diet composition and/or feed allocation system can have a major effect on the incidence of ascites. In most instances, such changes to the feeding program influence ascites via their effect on growth rate. There are several methods of feed restriction to successfully reduce the incidence of ascites including:

(1) composition of the feed or nutrient reduction programs,

(2) limiting the number of hours of access to feed, and

(3) skip-a-day programs.

1.3.1.1.2 Composition of Feed or Nutrient Reduction

Ascites is more common when high-energy diets are used, especially when these are pelleted. Mash feed, in contrast to a pelleted feed, could prevent a high incidence of ascites when given for the first 28 days of life (Shlosberg *et al.*, 1991). The reason behind this is that mash feed slows down growth, and therefore oxygen demand, because of its lower nutrient density. When fed in the first 4 wks, mash reduced mortality without downgrading market parameters (Shlosberg *et al.*, 1991). This feeding regime lowers the energy intake of birds at a time when feed intake per unit of metabolic weight is at its highest (Albers *et al.*, 1990), thus reducing oxygen requirements and, consequently, susceptibility to ascites.

It has also been suggested that dietary compositions that improve feed conversion ratio, such as high energy density, a high protein:energy ratio, and pelleted diets, all stimulate feed intake, protein accrual, and oxygen consumption, also increase the incidence of ascites (Schelle, 1993). Pellets dissolve in the crop almost immediately after consumption. The degradation of integral feed particle (not ground into fines or powder) in the upper intestine maybe slower than degradation of a fine powder, thus increasing peristalsis and improving feed utilization (Nir *et al.*, 1995).

Schelle (1993) also suggested that improved feed conversion may lead to a shortage of oxygen. Results from Nir *et al.* (1995) showed that the method of grinding feed (roller mill compared with hammer mill) improved feed utilization more than feed form (pellet versus mash), without any effect on mortality from ascites. The higher mortality due to feed form may be associated with lower activity. Behavior could be one of many factors contributing to the higher sensitivity of pellet-fed broiler chickens to ascities (Nir *et al.*, 1995). The slower growth rate of

mash fed birds appeared to contribute to their lower mortality compared with that of pellet-fed birds (Munt *et al.*, 1995). Munt *et al.* (1995) concluded that, despite the slower growth rate, the production of free choice fed (provides birds with the same feed ingredients as a mash formulation offered to the birds in separate whole grains) broiler chickens contribute to their greater profitability.

Energy restriction has been shown to result in a reduction in metabolic energy loss. This leads to a reduced requirement for maintenance, and if growth resumes to a normal or above normal rate (compensatory growth), feed efficiency would be substantially improved, leading to an economic advantage. Shlosberg *et al.* (1991) showed that mortality could be reduced from 2.32% to 0.9% by subjecting broilers to feed restriction from 6 to 11 days. The highest incidence of ascites occurred when the highest energy level was fed, regardless of either energy protein ratio or fat content of the diet. The greatest incidence of ascites occurred in the fastest growing birds (Leeson *et al.*, 1995).

1.3.1.1.2 Limited Access to Feed

Lighting programs are one method of restricting access to feed. Currently, there is interest in discontinuous lighting patterns for broilers because the altered growth pattern may be associated with a reduction in the incidence of skeletal abnormalities and mortality from Sudden Death Syndrome (Classen *et al.*, 1991). Sudden death in broilers may also be due to ascites, which can occur secondarily to right ventricular failure. It is possible that the lighting effects on ascites also reduce the stress associated with continuous lighting.

The increasing photoperiod lighting system (Figure 1-9) was beneficial in broiler flocks in reducing overall mortality, Sudden Death Syndrome mortality, and improving feed efficiency, with no reduction in body weight at market age (Blair *et al.*, 1993). The incidence of vargus leg abnormalities was reduced with the increasing light pattern, and the birds appeared to be more

mobile than those on constant light as judged by their ability to jump up onto a raised platform to feed (Blair *et al.*, 1993).

The benefits may stem from the reduced feed intake and resultant reduction in growth rate during the early phase of growth, coupled with physiological benefits associated with the light-dark cycle (Classen and Riddell, 1990). A reduced consumption of electricity and gas was an additional benefit with increasing photoperiod. Increasing light programs are beneficial in broiler flocks in reducing overall mortality, Sudden Death Syndrome mortality, mortality due to ascites, the incidence of leg deformities, and improving feed efficiency, with no reduction in body weight at market age (Blair *et al.*, 1993).

1.3.1.1.3 Skip-a-day Programs

If skip-a-day programs are instituted during the starting period, negative effects on subsequent body weight gain are minimal (Arce *et al.*, 1992). Feed restriction using skip-a-day methods or daily restriction methods have different effect on broiler performance and body composition. Plavnik and Hurwitz (1988) demonstrated that restricting feed intake of broilers at an early age exerts a negative effect on subsequent lipid deposition and a positive effect on lean tissue growth.

1.3.1.2 Compensatory Growth

Early feed restriction relies on the phenomenon called "compensatory growth" or "catch-up" growth (Yu and Robinson, 1992). Catch-up growth is defined as a recovery from a growth deficit resulting from a limited nutrient intake. Upon refeeding, birds demonstrate catch-up growth; however, the important point is that they are able to attain the same final body weight as controls at the market age (Acar *et al.*, 1995). Plavnik *et al.* (1986) pointed out that compensatory growth was obtained after short periods of restriction, whereas longer periods

diminished recovery and may have resulted in delay in achieving normal weight or in a permanent stunting of the animal. Broiler producers typically ship their birds at 42 days of age. Longer periods of feed restriction do not allow broilers time to experience compensatory growth.

The factors regulating feed intake are related in part to the anatomical adaptation of the gastro-intestinal tract. e.g. its increase in weight and in digestive enzyme activity. Protein might be a limiting nutrient during recovery period after a period of malnutrition. Plavnik and Hurwitz (1989) also suggested that essential amino acid requirements were higher in order to maximize growth in the first few weeks following feed restriction. If it is the case, the absence of compensatory growth and the limitation of protein deposition during the recovery period is due to an inadequate supply of dietary protein as noted by Yu *et al.* (1990). Also, if it is the case, high protein diet during the first few weeks might overcome the problem of the absence of compensatory growth.

Jones and Farrell (1992) stated that the success of feed restriction was associated with the success of maintaining BW stasis during the feed restriction period. Using skip day feeding, body weight was decreased by fasting and then was increased by a repletion period. Plavnik and Hurwitz (1988) suggested that compensatory growth was obtained after short periods of feed restriction, whereas longer periods diminished recovery and might result in delay in achieving normal body weight or in a permanent stunting of the animal.

1.3.1.3 Body Composition

Ad libitum feeding is an artificial condition, an outcome of modern management, and restriction feeding brings the animals back to their natural conditions (Balley *et al.*, 1992). Food scarcity or abundance is accompanied by adaptive mechanisms. Selection for body weight is accompanied by an increase body fat and abdominal fat. Early feed restriction lowers the percentage of abdominal fat at market age, which contribute to carcass yield and quality (Plavnik and Hurwitz, 1988). This lower quantity of carcass fat probably also contributes to the better feed

conversion efficiency of the restricted birds. The reduced amount of abdominal fat may be the result of the reduced number of adipocytes due to the early inhibition of adipocyte hyperplasia caused by the severe energy restriction (Plavnik and Hurwitz, 1985).

Restricted chicks could reach BW similar to the *ad libitum* chicks at 39 days of age when chicks were fed free access to feed and starved on the alternate day for 6 days (Balley *et al.*, 1992). Restriction for more than 6 days improved feed efficiency with no compensatory growth (Santoso *et al.*, 1993a). Nutrient restriction during the early life of chicks was investigated for reducing abdominal fat and body fat at market age without loss in general performance characteristics (Plavnik and Hurwitz, 1990). However, in the majority of cases fat deposition increases resulted from compensatory growth (Summers *et al.*, 1992).

A meat type-bird makes investments into supply organs such as gastro-intestinal tract and liver immediately after hatching to direct growth into the rapid development of the demand organs such as muscles and feathers. Therefore, the growth of breast muscle, as a large demanding tissue, becomes an important issue under the influence of feed restriction regimes (Acar *et al.*, 1995).

1.3.1.4 Feed Efficiency

Cumulative feed efficiency is better in young birds (Santoso *et al.*, 1993). This phenomenon could be explained by the principle of diminishing increments and food consumption during growth. As the animal grows larger, its maintenance cost in comparison to weight gain increases and, therefore, the energetic efficiency of growth decreases. As the broilers grow, beyond some point, broiler chickens deposits increasing proportions of fat relative to protein and water (Santoso *et al.*, 1993b). Consequently, the energy density of gain increases which causes the increasing inefficiency in the refed groups, which initially has been lower than in the control, exceeds that of the latter when body weights exceeds 2,500 g (females) or 3,000 g (males).

It is difficult to determine, however, whether an improvement, following short-term periods of feed restriction, resulted from the reduced needs for maintenance energy due to a lower body weight or reduced basal need following restriction (Plavnik and Hurwitz, 1985). The reduced energy requirement during feed restriction could result from the reduction of both basic metabolic rate and the specific dynamic action.

1.3.1.5 Conclusion

It is emphasized that feed restriction alone cannot be considered a complete preventative for the ascites syndrome; maintenance of satisfactory air quality and temperature are also critical. Once optimal management practices have been insured, restricted feeding can be highly effective in further reducing the incidence of ascites in commercial broiler flocks (Arce *et al.*, 1992).

Obvious disadvantages of feed restriction programs to control ascites syndrome include reduced weight gain and increased feed conversion leading to economic loss. If a feeding program could be developed that decreased the incidence of ascites syndrome without compromising productive performance, it would be of great economic benefit to producers (Arce *et al.*, 1992).

Final body weight gains are frequently, but not always, reduced slightly by feed restriction during the starting period. However, a modest weight reduction of birds in some feed restriction treatments appears to be an acceptable trade-off for the decrease in incidence of ascites (Arce *et al.*, 1992). Limiting growth rate by feed restriction may reduce the late mortality as well as lessen losses due to carcass condemnation and mortality associated with ascites (Shlosberg *et al.*, 1991; Acar *et al.*, 1995).

Feed restriction programs are unlikely to succeed without good management. For example, if litter quality is not maintained, subclinical coccidiosis may result from litter pecking on fasting days (Arce *et al.*, 1992). Adequate feeder space and rapid feed distribution are

important to prevent "piling up" when birds are re-fed after a period of restriction. While it may seem a simple matter to provide feed for a certain number of hours per day, in practice many growers cannot successfully accomplish feed restriction programs (Arce *et al.*, 1992).

1.3.2 Litter Oiling

1.3.2.1 Lung Function

The lungs of birds are rigid and molded into the thoracic cavity. They cannot expand as mammalian lungs do. The capillaries can dilate only very little to allow for increased blood flow. The lungs of chickens grow much less rapidly than the rest of the body and lung capacity is not proportional to the very rapid growth of muscle in fast growing broiler chickens (Jordan, 1990).

It has been suggested that the broiler, enhanced through selection for rapid BW gain and increased gain efficiency, is approaching its metabolic limit for growth in relation to oxygen consumption (Scheele *et al.*, 1991a). Therefore, these broilers may not be able to adapt to increased oxygen demands related to environmental stress. This theory is supported by the finding that the lung per unit BW is 20 to 33% lower for a modern layer breed than for its wild predecessor the Red Jungle Fowl. Even though layer breeds have an increased surface area for gas exchange, the net result was 28% thicker blood-gas barrier when compared with the Red Jungle Fowl. This increase had led to a 25% lower anatomical oxygen diffusion capacity of the blood-gas tissue barrier per unit BW in the domestic layer. Because layers are known to grow slower than broilers, it is speculated that this difference would be even greater for modern broiler strains (Anthony *et al.*, 1994).

Chickens that develop ascites following right ventricular failure seem to die from respiratory failure because of the pressure of ascitic fluid on air sacs, or from lung edema. Chickens that die without developing ascites appear to die from respiratory failure from lung

edema. It is postulated that the lung edema is caused by increased interstitial and air capillary fluid as a result of pulmonary hypertension, from increased pressure in the usually low pressure vascular system of the lung because of the much increased pressure capacity of the hypertrophied right ventricle (Julian *et al.*, 1989).

Because of continued selection pressures on growth, the incidence of ascites in broilers grown at low altitudes is likely to continue to increase (Huchzermeyer *et al.*, 1988). The high oxygen demand of rapid growth may increase pulmonary arterial pressure and the workload of the heart (Julian, 1987). Induced hypoxia increased the hemoglobin concentration, packed cell volume, and red blood counts (Maxwell *et al.*, 1990). Results were similar to those of aged broilers with ascites. Lactate dehydrogenase increased in the hearts of both hypoxic and ascitic birds, indicating reduced oxygen utilization. Fast growing male broilers, which require large quantities of oxygen, have insufficient pulmonary diffusion capacity. Hyperthyroidism has been stated to increase the oxygen requirement (Julian, 1990). If broilers exhibiting borderline lung capacity encounter anything that interferes with oxygen transport, breathing ability, or cardiac output, they could develop hypoxia (Anthony *et al.*, 1994).

Although ascites can occur throughout the year, it is mainly associated with cold weather, when poultry house ventilation is reduced to minimize heating losses. Low ventilation rates result in higher levels of noxious gases (NH_3, CO_2, and CO) and dust (Anthony *et al.*, 1994; Feddes *et al.*, 1992).

1.3.2.2 Respirable Dust

Dust may have several effects on health and performance of livestock, such as irritation of the respiratory tract and lowering resistance to respiratory diseases. While dust is not the primary health problem in turkey barns, it can contribute to mortality by increasing susceptibility to other problems. Dust can negatively affect rate of gain, thereby decreasing revenues of turkey operations. Inhaled particles high in protein content may cause allergic reactions. Furthermore,

when airborne pathogens are inspired, infection may result. High concentrations of non-pathogenic organisms may also be harmful. Viable particles such as spores and bacteria are generally small enough to be respirable. These particles may exist as solitary particles, or they may be attached to larger dust particles (Feddes *et al.*, 1992).

If viable particles reach the lungs, lesions in the terminal air sacs may result (Feddes *et al.*, 1992). Fungal and bacterial infections are thought to be opportunistic, that is, they flourish when the turkey is predisposed to other health problems and, in turn, cause further weakening and possibly death in affected turkeys. Maximum daily dust concentrations were 6 particles/mL for broilers (Feddes *et al.*, 1992), 13 particles/mL for laying hens (McQuitty *et al.*, 1985), 27 particles/mL for broiler breeders (O'Connor *et al.*, 1988), and 100 particles/mL in pullet rearing facilities (Glennon *et al.*, 1989). Turkeys that are exposed to dust levels around 25 particles/mL, 44 particles/mL late in the growth cycle, over a growth period may be expected to suffer ill effects from the dust (Feddes *et al.*, 1992).

1.3.2.3 Application of Canola Oil to the Litter

Poultry house aerosols consist of a wide range of particle sizes from 0.5 μm to 100 μm and shapes. Particles that are < 5 μm are categorized as respirable. These particles are not trapped by the upper respiratory tract, they are able to penetrate to the terminal air sacs of the lungs (Feddes *et al.*, 1992). The addition of canola oil to litter has a significant affect on lowering dust concentration. Canola oil, when applied to the litter, binds the < 5 μm respirable dust particles. Thereby, canola oil is able to reduce the amount of respirable dust in the air and therefore the amount of dust inhaled by the birds.

1.3.3 Temperature Fluctuations

Very fast growing meat-type chickens may develop primary pulmonary hypertension because of their high oxygen requirement (Julian *et al.*, 1987). Factors such as cold, which increase requirement, also increase cardiac output and blood flow and may result in increased pulmonary arterial pressure and cause both a volume and a pressure overload on the right ventricle. A drop in environmental temperature from 20 °C to 2 °C for example, almost doubles the oxygen requirement in adult female White leghorn hens (Gleeson, 1986). May and Deaton (1974) showed that cold increased the heart weight of chickens, probably as a result of the increased work-load on both the ventricles. Chicks have a very narrow thermoneutral zone and any temperature below an optimum increases the oxygen requirement (Olson *et al.*, 1972).

Cold temperatures have a strong effect on right ventricular hypertrophy, right ventricular failure, and ascites, as well as, on heart weight. Cold caused a significant hypertrophy of the right ventricular and total ventricle of chickens which survived to 57 days and which did not have ascites at processing (Julian *et al.*, 1989). The effect of growth and diet on ascites are less marked in some strains of broilers than the effect of cold, perhaps due to a lung volume and strain association.

High temperature (above 30 °C) reduces food intake, which in turn reduces pulmonary hypertension syndrome, although Huchzermeyer *et al.* (1988) have shown that oxygen requirement increases above 22 °C. If broilers continue to eat and grow well at 25 °C to 30 °C the incidence of pulmonary hypertension syndrome would increase as it does with cold exposure.

The recent increase in ascites both at high and low altitude has been noted to be more marked in cold weather. Although some workers have pointed to the possibility of poor ventilation causing low environmental oxygen, or noxious fumes causing lung damage in cold weather, there is no evidence to support this pathogenesis. Recent research shows that the increased metabolic rate induced by cold causes a marked increase in oxygen requirement and cardiac output resulting in pulmonary hypertension. Increased oxygen requirement is probably

the most important factor in cold-induced pulmonary hypertension syndrome. Cold may also increase blood viscosity, perhaps by decreasing blood volume (Julian, 1993).

1.4 Techniques of Assessing Cardiac Size and Function

Pulmonary hypertension can arise when an increase in cardiac output (CO) exceeds the capacity of the pulmonary vasculature to accommodate sufficient blood flow at a normal pulmonary arterial pressure (PAP), or when an elevated pulmonary arterial pressure is necessary to overcome an increased pulmonary vascular resistance (PVR) caused by vasoconstriction or lung damage (Owen *et al.*, 1995a; Wideman and Kirby, 1995). The increased work performed by the right ventricle in sustaining an elevated pulmonary arterial pressure triggers specific right ventricular hypertrophy and dilation; consequently, an increased right:total ventricular (RV:TV) weight ratio is the earliest gross symptom observed during the pathophysiological progression (Hernandez, 1987; Julian, 1993).

Following the onset of pulmonary hypertension, hypoxemia and hypercapnia (the presence of excessive amounts of carbon dioxide in the blood) develop when the rate of blood flow through the pulmonary vasculature becomes too rapid to permit normal gas exchange (Wideman and Kirby, 1995). Hypoxemia reduces the contractility of avian cardiac muscle (Maxwell *et al.*, 1990), which may account for the generalized cardiac enlargement, flaccidity, and congestive heart failure occurring during the terminal stages of the pathophysiological progression (Julian, 1993).

There are currently two methods of assessing cardiac size and function right ventricular weight ratio and electrocardiographic evaluation. The pulmonary artery clamp (Wideman and Kirby, 1995) is a new technique used to initiate pulmonary hypertension.

1.4.1 Right Ventricle Ratio

Wilson *et al.* (1988) showed ascitic birds to have a RV/TVx100 (right ventricle/total ventricle weight) to be around 50 versus 20 for normal birds. In addition to hypertrophy, the right ventricle also showed a dilation because volume was 0.6 mL compared to 0.1 mL in control birds. Maxwell *et al.* (1986) quantitated in detail the effect of ascites on various physiological parameters (Table 1-1). The right ventricle to total ventricle ratio is a more objective method of assessing RVH and is the recommended way to measure the response to increased partial pressure (Julian *et al.*, 1986).

Ascites birds were smaller, and there were an increase in both packed cell volume and hemoglobin status. Maxwell *et al* (1986) suggested that the increase in white blood cell (WBC) count of ascitic birds may be a consequence of stress, since there was an increase in the heterophil count at the expense of lymphocyte numbers. Hernandez (1987) quantified the changes seen the hemoglobin, percent hematocrit, RV/TV ratio, pulmonary arterial pressure, and pulmonary arterial thickness of birds with and without ascites examined at commercial farms maintained at 2600 m above sea level (Table 1-2).

The heart ratio in broilers with the ascites syndrome indicate that normal broiler chickens likely have mean RV/TV ratio of <0.219 and that fast growing broilers develop RVH and dilation that may not cause valvular insufficiency until the ratio approaches or exceeds 0.290. Because chickens in right ventricular failure eat much less and stop growing, they have a much lower oxygen requirement. Because of the valvular insufficiency they are no longer moving as much blood through the lung. The left ventricle becomes hypertrophic which distorts the ratio in chickens which survive for several days or longer with right ventricular failure. The ratio in some broilers with the ascites syndrome was above 0.50 (Julian *et al.*, 1989). An increase in pressure causes an increase in the volume of blood that is pumped throughout the system. The right ventricular area (right ventricle cavity) enlarges with the pressure increase in broilers with the ascites syndrome (Table 1-3).

1.4.2 Electrocardiographic Evaluation

Electrocardiography is routinely used in human and veterinary medicine to assess alterations in cardiac size and function. Electrocardiography is a noninvasive method for detecting cardiac change. When broilers are exposed to hypobaric hypoxia, pulmonary hypertension and cardiac hypertrophy are triggered leading to an increase in wave amplitudes of the electrocardiogram (ECG) lead II RS wave (Wideman and Kirby, 1996).

Rigorous culling criteria designed to minimize confounding sources of variably allow the identification of significant differences in the mean electrical axis (degrees), mean resultant vector amplitudes (millivolts) and lead II RS and S wave amplitudes as early as 5 to 6 d after initiation of pulmonary hypertension, at a time when clinical ascites was not evident. Longer growout periods have been required to detect significant ECG changes, in part because the age of onset of pulmonary hypertension can be highly variable when triggering stressors is less intense. ECG patterns can also be highly variable within broiler populations that have not been specifically culled. Wideman and Kirby (1996) evaluated birds used in ECG studies at day 14 for ECG abnormalities which included those exhibiting erratic or arrhythmic ECG wave forms, missing or inverted wave segments, or ECG lead II RS-wave amplitudes outside a specified range.

For the purposes of the practical application of electrocardiography to identifying susceptible but subclinical individuals in a survey of large numbers of birds, multiple ECG leads and appreciably complicated calculations would be required to obtain standard degree-vector alignments for the frontal plane mean electrical axis (degrees) and mean resultant vector amplitude (millivolts) values. In contrast, the S wave amplitude in lead II S wave amplitude is easily obtained from a simple two-lead (right wing to left leg) ECG. An appropriate initiating stressor (cool temperature, hypobaric hypoxic, pulmonary artery clamp) must be applied to the broilers being surveyed if electrocardiography is to be successfully applied as a noninvasive index for predicting subclinical susceptibility to pulmonary hypertension syndrome (Wideman and Kirby, 1996).

1.4.3 Pulmonary Artery Clamp

Wideman and Kirby (1995) recently developed the pulmonary artery clamp model which was used to synchronize the initiation of pulmonary hypertension. Three interdependent processes related to pulmonary vascular resistance (PVR) theoretically may contribute to the increase in pulmonary arterial pressure (PAP) leading to pulmonary hypertension syndrome (PHS; ascities) in broilers. According to the equation: PAP = cardiac output x PVR, increases in pulmonary arterial pressure must occur:

(1) if the pulmonary vascular capacity is anatomically inadequate to accept the normal cardiac output at a low pulmonary arterial pressure (high anatomical pulmonary vascular resistance;

(2) if an elevated cardiac output must be propelled through a relatively inelastic or noncompliant pulmonary vasculature;

(3) if inappropriate constriction of the pulmonary vasculature directly elevates pulmonary vascular resistance (Julian, 1993).

Chronically clamping one pulmonary artery (Figure 1-10) in clinically healthy 2 to 3-wk-old broiler chicks directly doubles pulmonary vascular resistance and initiates pulmonary hypertension by forcing the right ventricle to develop sufficient pulmonary arterial pressure to force the entire cardiac output through the vasculature of the unobstructed lung. Doubling the pulmonary ventricular resistance by clamping one pulmonary artery in 2 to 3-wk old broiler chicks represents a severe challenge at an early age, thereby exposing ascites susceptibility across most of the population continuum.

In addition to reliably inducing a high incidence of ascites with symptoms paralleling those induced by other commonly used experimental models, the pulmonary artery clamp model also permits effective synchronization of the age at which pulmonary hypertension is initiated. This model initiates a progression of symptoms typical of those observed in broilers developing ascites spontaneously during commercial broiler growout, or during exposure to commonly used experimental models such as cold stress or hypobaric hypoxia (Wideman and Kirby, 1995). The

pathophysiological progression initiated by clamping one pulmonary artery provides direct support for the hypothesis that broilers are susceptible to ascites when their metabolic demand for oxygen is not matched by the capacity of their pulmonary vasculature to accept and oxygenate, at a normal pulmonary arterial pressure, the cardiac output necessary to supply that oxygen (Wideman and Kirby, 1995).

1.5 Introduction to Chapters

Based on the current problems faced in the broiler industry , and the research objectives of the University of Alberta Poultry Research Center research group, three separate experiments were conducted.

Chapter 2: The experiment entitled "Growth performance, carcass characteristics, and the incidence of ascites in broilers in response to feed restriction and litter oiling" was a comparison of feed restricted and *ad libitum* fed birds in combination with the litter oiling and non-oiled litter treatments. The effect of feed restriction and litter oiling on the ascites syndrome in broilers was determined.

Chapter 3: The experiment entitled "Image analysis of right ventricular areas to assess the severity of ascites in broiler chickens" was completed to provide evidence that the image analysis technique used for comparison of the right ventricle area (RVA), left ventricle area (LVA), and heart area (HA) was a repeatable technique.

Chapter 4: The experiment entitled "Growth performance, carcass characteristics, and the incidence of ascites in broiler chickens in response to temperature fluctuations and litter oiling" was a comparison of four pens with 6°C temperature fluctuation and four pens with control temperatures in combination with the litter oiling and non-oiled litter treatments. The effect of temperature fluctuations and litter oiling on the ascites syndrome in broilers was determined.

Chapter 5: This chapter includes the final results and discussion.

TABLE 1-1. Comparison of 35 day-old ascitic and normal broiler chickens

characteristic	normal	ascitic	significance
Body weight (g)	1390	960	**
Heart weight (g)	9.5	9.3	NS
Heart weight (% BWT)	0.7	1.0	*
Hemoglobin (g/100 mL)	9.3	11.6	**
PCV (%)	30	38	**
RBC (10^6/mm^3)	2.6	3.2	**
WBC (10^6/mm^3)	16.1	20.5	*
Heterophils (% WBC)	17.8	32.3	**
Lymphocytes (% WBC)	71.0	52.6	**

Adapted from Maxwell *et al.* (1986)
NS = not significant
* P <0.05
** P<0.01

TABLE 1-2. Cardio-pulmonary changes in ascitic birds held at 2600m altitude

	hemoglobin (g/100ml)	hematocrit (%)	RV/TV (%)	pulmonary arterial pressure (mm)	pulmonary arterial thickness (% diam)
control	10.8	26	15	21	6.6
ascites	12.1	44	41	44	10.1

Adapted from Hernandez (1987)

TABLE 1-3. Heart characteristics quantified using Image Analysis Technology

	RVA (cm^3)	LVA (cm^3)	HA (cm^3)	RVWT (g)	RV/TV (%)
control	0.24	0.06	3.17	1.888	8.413
ascites	1.71	0.15	4.84	3.852	8.719

FIGURE 1-1. Upper diagram. Trachea and lungs, ventral view with the air sacs removed. Lower diagram. Diagrammatic view of the air sacs, lateral view (Sturkie, 1986).

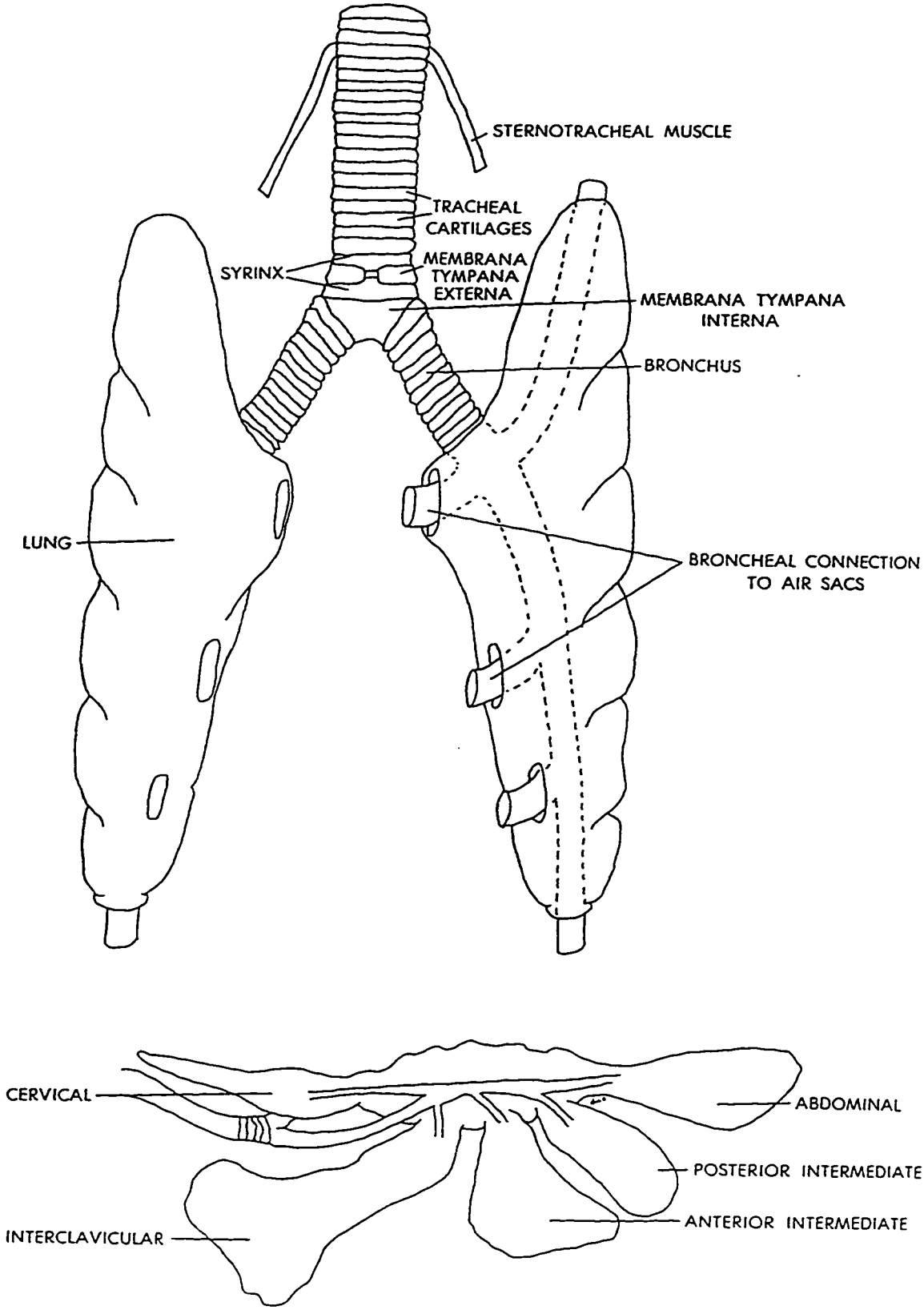


FIGURE 1-2. Possible series of events that lead to the development of the ascites syndrome (Julian, 1993).

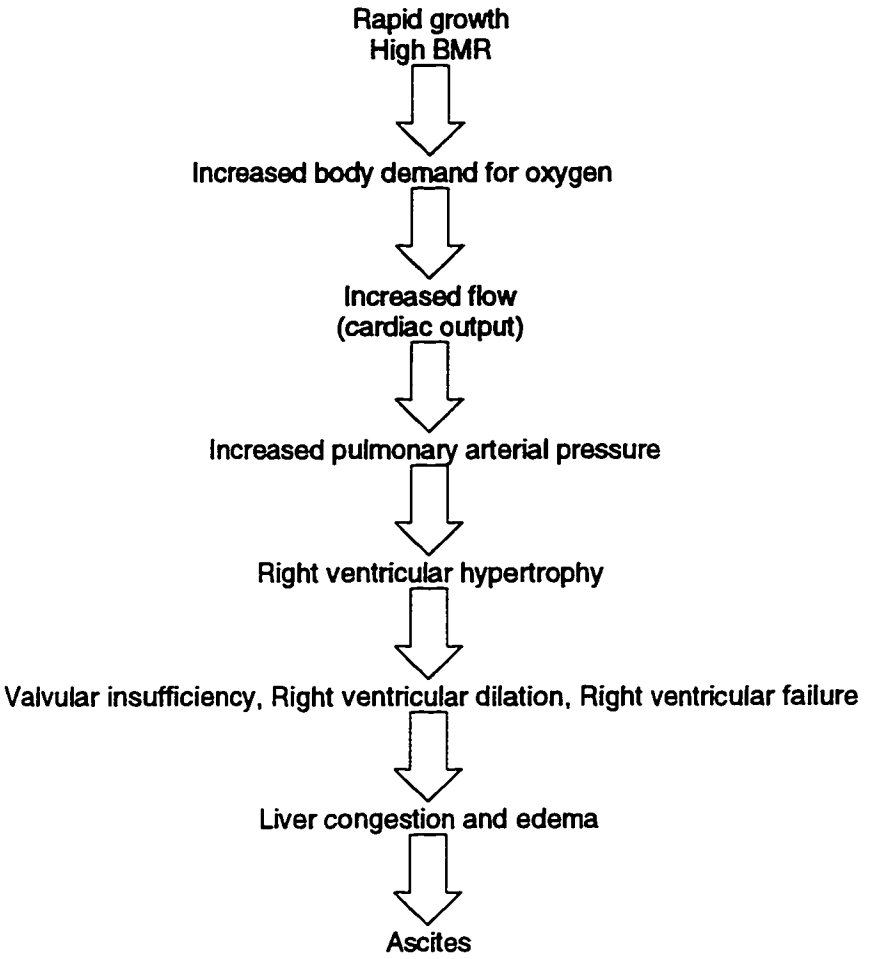


FIGURE 1-3. A generalized diagram of lymphatic drainage. (A). Lymph streams slowly through vessels of increasing caliber (B,C), many of which are provided with valves (D). Lymphocytes, many produced in solitary lymph nodules (E) are added. Lymph is filtered through lymph nodes (F), where more lymphocytes and antibodies are added. Finally it joins the venous circulation (G) (Frandson and Spurgeon, 1992).

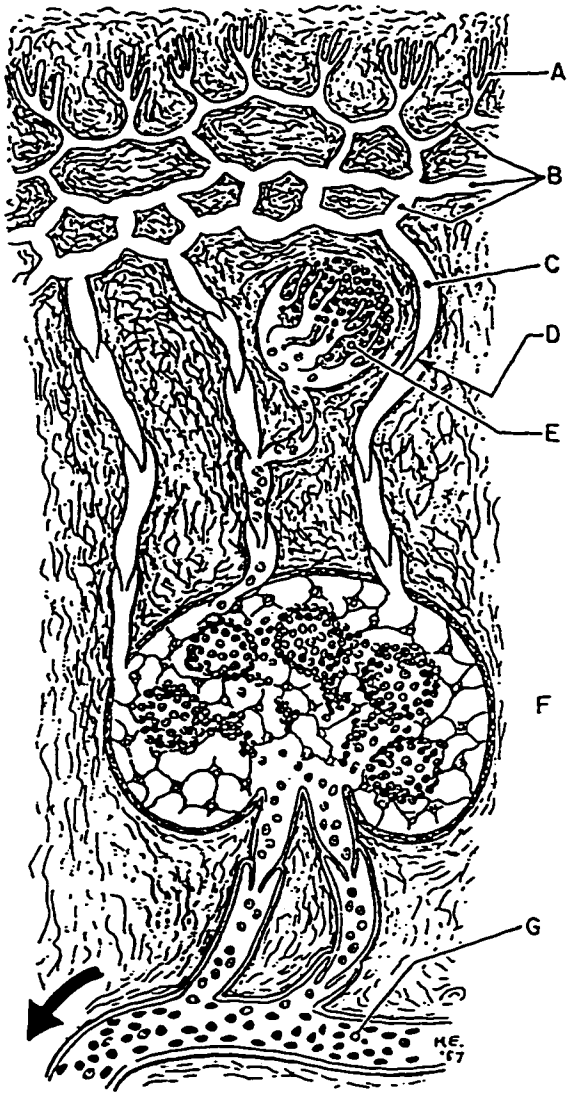


FIGURE 1-4. Blood flow through the heart: pulmonary and systemic circulation's. Relatively higher oxygen content of blood is indicated by stippling (Frandson and Spurgeon, 1992).

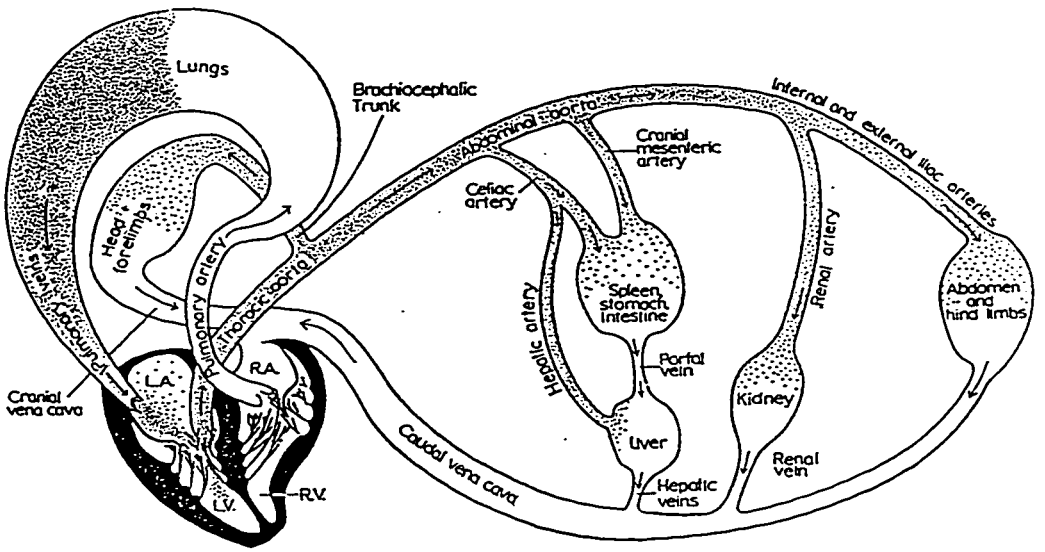


FIGURE 1-5. The relationship of the body cell to blood circulation and lymph drainage. Plasma fluid passes out of the capillary walls. Excess fluid is removed by the lymph vessel (Frandson and Spurgeon, 1992).

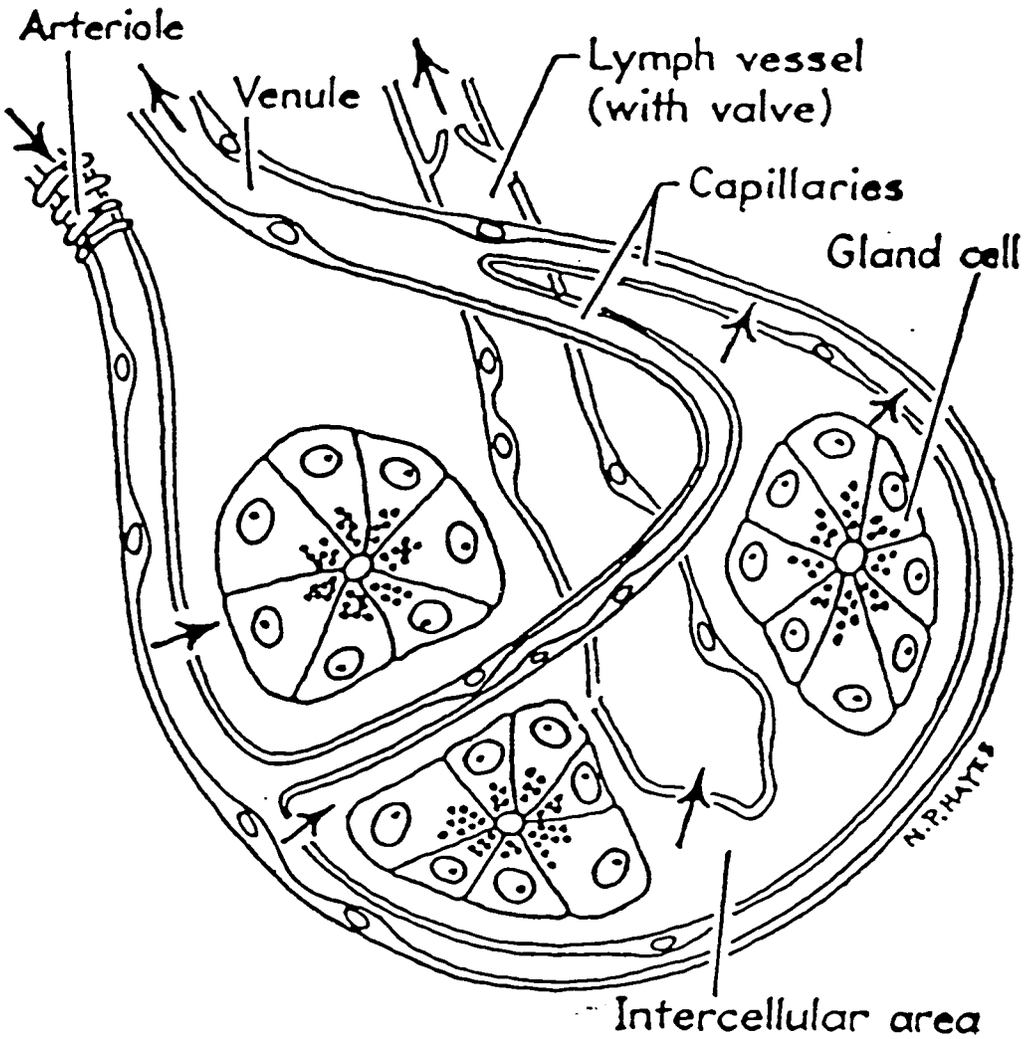


FIGURE 1-6. Possible causes of increased pulmonary arterial pressure in broiler chickens resulting in increased right ventricular work-load (Julian, 1993).

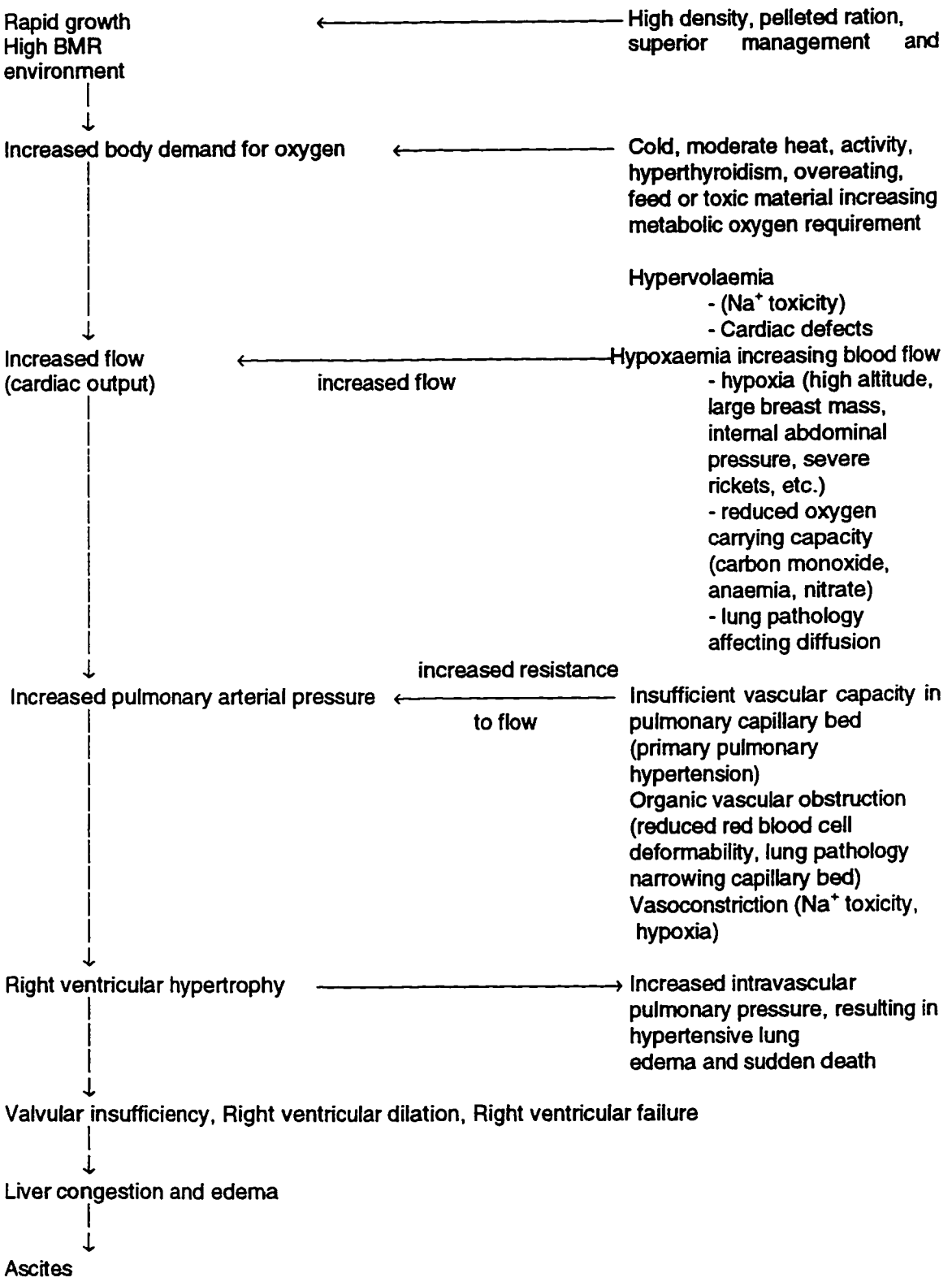


FIGURE 1-7. Possible causes of pulmonary hypertension (Julian, 1993).

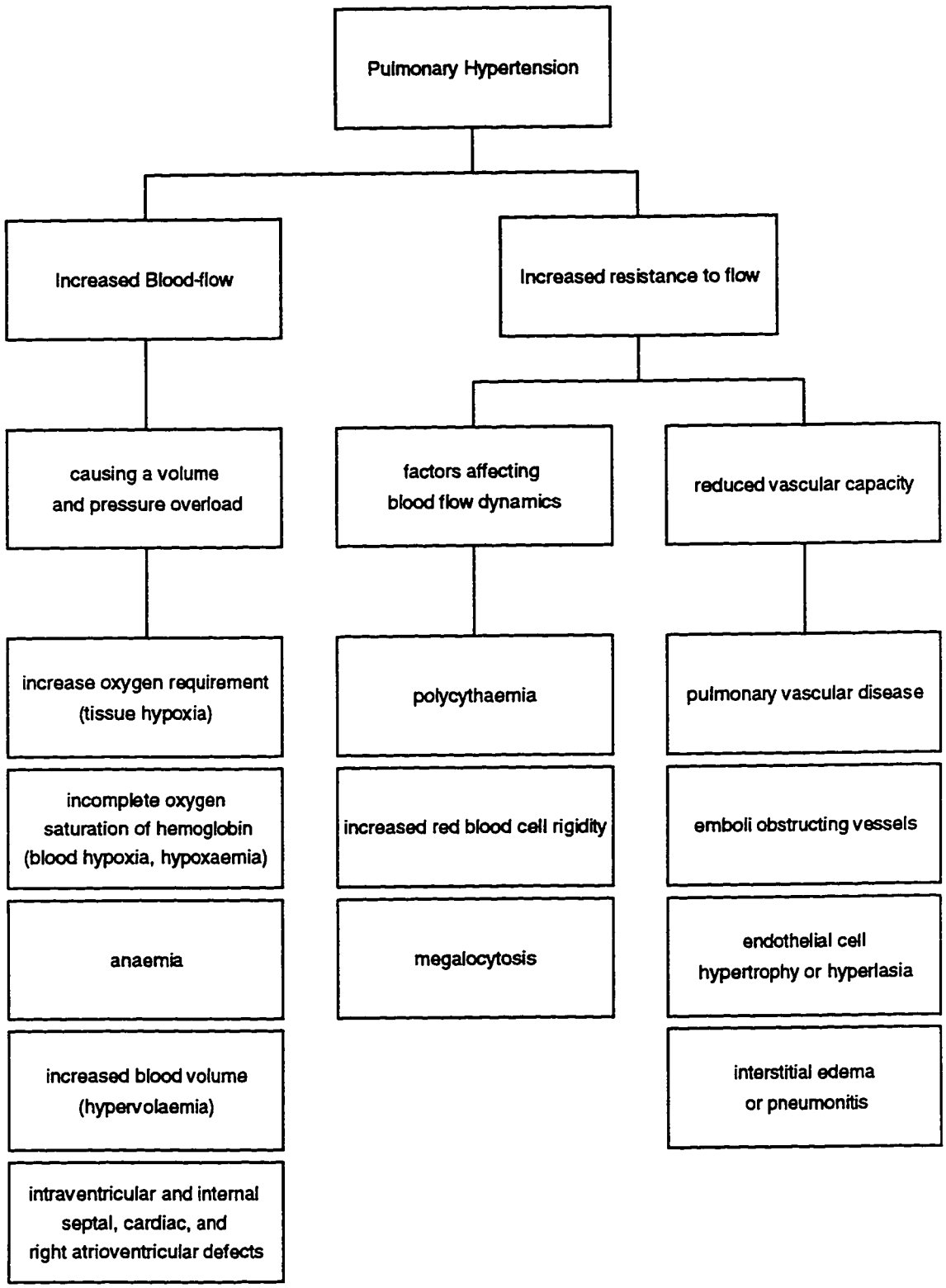


FIGURE 1-8. The avian heart (Sturkie, 1986).

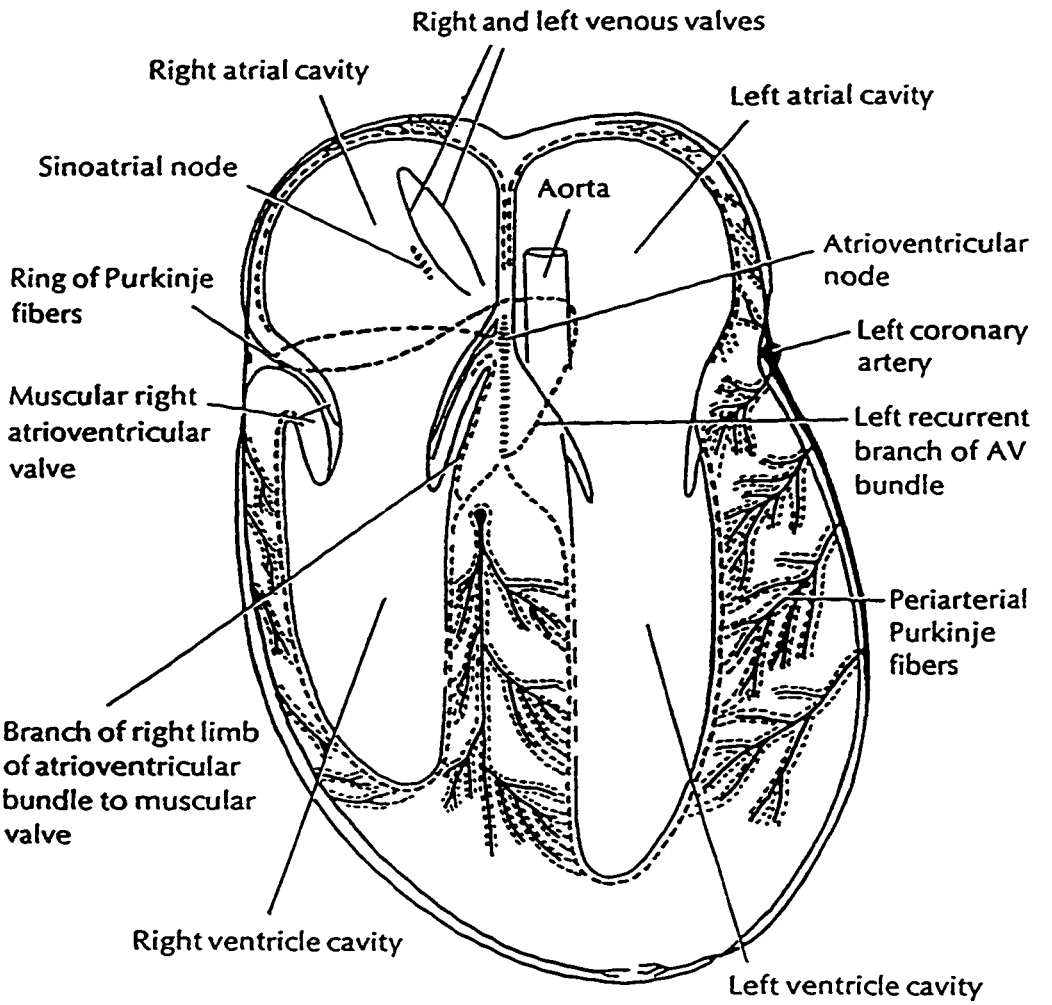


FIGURE 1-9. Increasing photoperiod lighting system (Blair *et al.*, 1993).

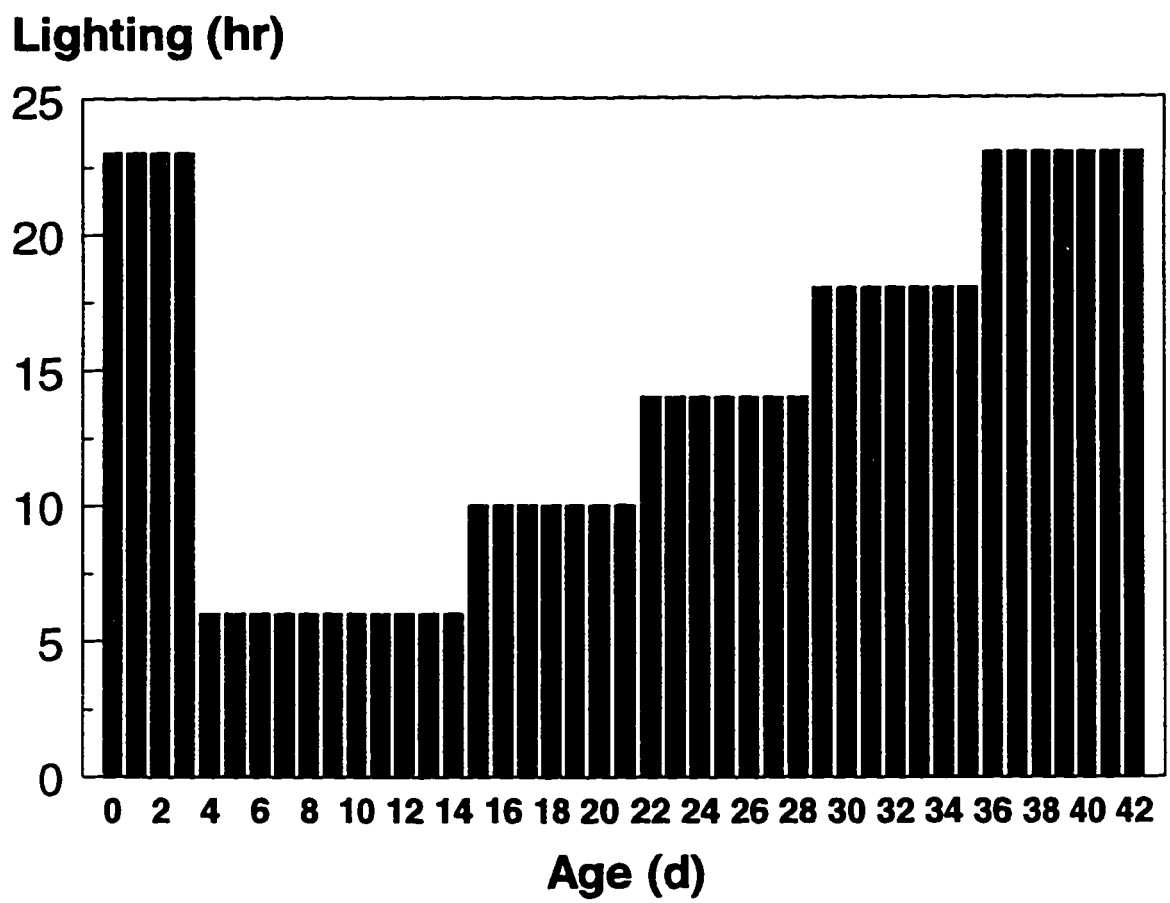
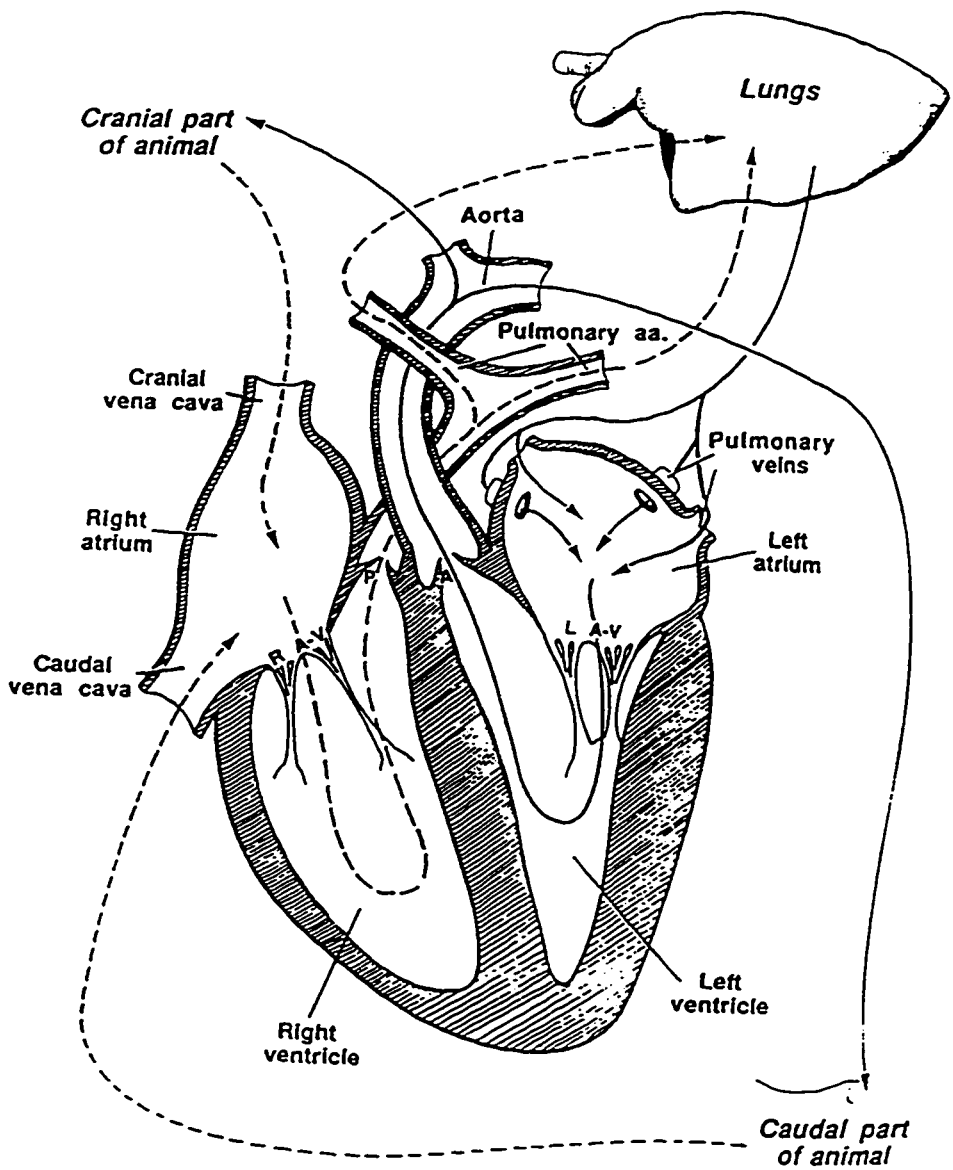


FIGURE 1-10. Clamp positioning for the pulmonary artery clamp (Frandson and Spurgeon, 1992).



Valves of the Heart

R A-V	= Right atrioventricular ("tricuspid") valve
L A-V	= Left atrioventricular ("bicuspid", "mitral") valve
A	= Aortic semilunar valve
P	= Pulmonic semilunar valve

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2. GROWTH PERFORMANCE, CARCASS CHARACTERISTICS, AND THE INCIDENCE OF ASCITES IN BROILERS IN RESPONSE TO FEED RESTRICTION AND LITTER OILING

2.1 INTRODUCTION

Increases in the incidence of ascites in broiler chickens coincide with continuing genetic and nutritional improvements in enhanced feed efficiency and rate of growth. Ascites is a condition in which the body cavity accumulates ascitic fluid, leading to carcass condemnation or death. It is a consequence of cardiopulmonary insufficiency in rapidly growing broiler chickens (Julian, 1993). The housing environment including factors such as temperature (cold, or moderate heat) and air quality (dust concentration, carbon dioxide levels, and oxygen levels) are known to influence the incidence of ascites in broiler chickens. Rapid growth rates in conjunction with high basal metabolic rate create an increased demand for oxygen leading to an oxygen deficit. Increasing the demand for oxygen may exceed the cardiopulmonary capacity to supply sufficient oxygen (Witzel *et al.*, 1990). An increase in hemoglobin concentration, packed cell volume of hematocrit, and erythrocyte count due to the increased demand for oxygen result in higher blood viscosity and may cause pulmonary hypertension (Mirsalimi *et al.*, 1993). Right ventricular hypertrophy and dilation are the result of the increased workload, which in turn leads to right ventricular heart failure, increased portal pressure, edema and ascites. Pulmonary edema increases the resistance to diffusion across the blood gas barrier, thereby pathologically retarding blood oxygenation (Wideman and Kirby, 1995). Hypertrophy of the right ventricular wall can be directly related to the blood pressure (Julian, 1993). The RVA and the ratio of the right ventricle to the total ventricle mass can be used as a measure of the effects of increased blood pressure on the right ventricle (Julian *et al.*, 1986).

The growth rate of broilers has increased markedly in recent years due to improvements in nutrition and genetics. Although this rapid increase in growth rate has been beneficial to the

industry in reducing the time required to bring a bird to market age and in reducing the quantity of feed required, it has had adverse affects on carcass composition and on animal health. There are a wide range of causes for ascites. Situations which impose a higher metabolic oxygen demand on broilers are primarily responsible for the increase in the incidence of ascites. Modification of the growth rate of the broiler by limiting weight gain during the starting period can significantly alleviate the incidence of ascites (Arce *et al.*, 1992). Feed restriction followed by a period of normal feeding can significantly improve feed conversion, reduce carcass fat, reduce the incidence of leg disorders, and mortality (Plavnik and Hurwitz, 1985; Yu and Robinson, 1992).

Feed restriction, when applied at typical industry rates, markedly slows growth in terms of carcass frame size (Yu *et al.*, 1992b), carcass fat accumulation and absolute protein content of the carcass (Yu and Robinson, 1992, Yu *et al.*, 1992a). The broiler is approaching its metabolic limit for growth in relation to oxygen consumption (Scheele *et al.*, 1991). Slower growing birds have reduced oxygen needs (Arce *et al.*, 1992). By slowing the rate of gain during the starting period, through the use of short-term feed restriction for example, the cardiopulmonary systems of the birds may not be taxed as severely. This allows somatic growth to remain in-phase with the functional growth of organs in the cardiopulmonary systems (Arce *et al.*, 1992).

Broilers may not be able to adapt to increased oxygen demands related to environmental stress. High concentrations of respirable dust can cause irritation of the respiratory tract due to its high protein content (Feddes *et al.*, 1992). This affects the health and performance of turkeys (Feddes *et al.*, 1992) and could lead to an increased susceptibility to ascites in broilers. The removal of dust particles may improve oxygen and carbon dioxide transfer in fast growing male broilers since they require large quantities of oxygen and have limited pulmonary diffusion capacity (Julian *et al.*, 1986). If broilers exhibiting lung capacity deficiencies encounter factors that interfere with oxygen transport, breathing ability, or cardiac output, they could develop hypoxia as a primary stimulus of the ascites syndrome (Anthony *et al.*, 1994; Balog *et al.*, 1994).

The prime objective of this study was to discover the effect of feed restriction and litter oiling on the carcass characteristics and heart characteristics of broiler chickens and relate those to differences in the incidence of ascites.

2.2 MATERIALS AND METHODS

Eight hundred male broilers were housed in four pens of 100 birds each over two 6 wk periods. In each replicate, two pens of birds were feed restricted from 7 d to 16 d of age. Two pens of birds received feed *ad libitum* for the 6 wk trial. One restricted and one *ad libitum*-fed pen of broilers received biweekly application

of oil to the litter. The feed restriction was applied at day 7. Each bird received 18 g of feed until day 16, after which normal *ad libitum* feeding was resumed. A total of 0.8 L/m^2 of oil was applied over the 6 wk period. All birds received a standard pelleted broiler starter diet (3250 kcal ME/ kg of diet, 23% CP) from 1 to 3 wk of age. A grower diet (3250 kcal ME/ kg of diet, 21% CP) was fed from 4 to 5 wk of age. The finisher diet (3250 kcal ME/ kg of diet, 19% CP) was fed during wk 6. Concentrations of respirable airborne dust (<10μm and >0.5μm) were measured weekly in each pen using the Climet Air Sampler. A record of the temperature and mortality was also maintained throughout the trial. All dead birds were necropsied to determine cause of death and the heart morphology was examined.

At 3 wk of age, all birds were wing-banded for identification. All broilers were individually weighed weekly from 3 wk of age until 6 wk of age. At 6 wk of age, 30 birds from each pen were killed for determination of weight of the breast, fatpad, and heart. Hearts were collected from the remaining 380 birds at slaughter. All birds were scored for the incidence of ascites (Table 2-1).

All 800 hearts were processed for measurement of RVA using image analysis technology. A transverse cut was made on all hearts just below the tricuspid and bicuspid valves and a second transverse cut was made at the stem of the papillary muscle (Figure 2-1). From the 3 to 5 mm heart slice, a cross sectional image of each heart was digitally recorded and using

image analysis technology (Northern Exposure, 1996[1]) the RVA, left ventricle area (LVA), and total heart area (HA) were determined (Figure 2-2). The right ventricular wall was removed and its weight (RVWT) was compared to the RVA. The RVWT was also expressed as a percentage of total heart weight (HWT). After slaughter, the heart characteristics of all birds were assessed.

Two hundred and forty broilers were selected, from the 800 broilers used, for further analysis of carcass characteristics. The fatpad, *pectoralis minor*, and *pectoralis major* were removed from the 240 broilers for a comparison of the weights. The data were analyzed by analysis of variance and (SAS Institute, 1994) with $P<0.05$. Coefficients of correlation were obtained using SAS with $P<0.05$ (SAS Institute, 1994).

2.3 RESULTS AND DISCUSSION

The objective of this study was to discover the effect of short term feed restriction and litter oiling on the carcass and heart characteristics of broilers and relate those to differences in the incidence of ascites. Initial bird BW were the same for all treatments. After feed restriction at 3 wk of age, the feed restricted birds had a significantly lower BW than the *ad libitum* birds. The *ad libitum* fed broilers were significantly heavier than the feed restricted birds throughout the trial (Table 2-2). Litter oiling also resulted in a significant difference between BW at 3 and 4 wk of age. Broilers in pens with litter oiling weighed 587 g at 3 wk and 1048 g at 4 wk. Pens not receiving the applications of oil weighed 602 g at 3 wk and 1070 g at 4 wk. There was no interaction in BW between feed restriction and litter oiling. Feed conversion was not different between treatments (Table 2-6). Weekly feed consumption was not different in wk 3, 4, 5, and 6 between treatments, however, the overall feed consumption was lower in the feed restricted treatment because feed was withheld to a level of 18 g of feed from day 7 until day 16.

Feed restriction decreased the incidence of ascites syndrome. The feed restricted pens of birds had a significantly lower mortality rate (6.3% compared to 15.9%) (Table 2-7) and a

[1] Empix Imaging Inc., 3065 Ridgeway Drive unit #51, Mississauga, Ontario, Canada, L5L 5M6

lower average ascitic score (0.61 compared to 0.87) than the *ad libitum* fed broilers indicating reduced growth rate which prevents increased blood flow leading to right ventricular enlargement. If growth is slowed by restricting feed or by feeding mash, fewer cases of ascites will develop, probably because of the reduced metabolic rate and lower oxygen requirement (Julian *et al.*, 1986). It is unlikely, however that this degree of feed restriction would be economically viable in current markets. The severe short term feed restriction treatment used for this experiment was necessary to establish the use of the image analysis technique by using a comparison of a group of healthy broilers to a group of broilers with a higher risk of exhibiting ascitic symptoms (*ad libitum* fed treatment).

At an average gain of 50 g/day per pen of broilers, it would take an extra 4.26 days for the feed restricted broilers to reach the equivalent final BW of the *ad libitum* fed broilers. The advantage gained by using feed restriction to reduce mortality does not out weigh the reduction in final bird weights in this case. Extending the cycle length to 48 days would increase production costs (barn heating, maintenance, loss of production (6.6 broiler cycles/yr. rather than 7.4 broiler cycles/yr.), increased feed cost, and increased labor cost), making severe feed restriction regimes undesirable. Shlosberg et al. (1991) showed that mortality could be reduced from 2.32% to 0.9% by subjecting broilers to feed restriction from 6 to 11 d (20 g/bird/day), a 7 d restriction compared to 9 d restriction used in this experiment.

Litter oiling significantly decreased the RVA. Using a total of 0.8 L/m^2 of canola oil over the 6 wk period was an effective amount at a stocking density of eight broilers/m^2. Feed restriction significantly decreased the ascites score, HA, RVA, RVWT, THWT, percent right ventricle area (PRVA), and PRVW (Table 2-3). The THWT as a percentage of total BW was significantly greater in feed restricted broilers than the *ad libitum*-fed broilers.

Ascites score was significantly correlated to the RVA and RVWT (Table 2-4). The RVA and the RVWT were significantly correlated. As shown by the scatter plot (Figure 2-5), there is a large variation in RVA within a score. There is a trend, however, that shows that the increase in the RVA corresponds to an increase in ascitic score. The small RVA that have high ascitic scores

are differences in the onset of the ascites syndrome. Body weight, fatpad weight, *pectoralis minor* muscle weight, and *pectoralis major* muscle weight were significantly correlated to THWT, 0.67, 0.32, 0.60, and 0.60, respectively. Right ventricular weight was significantly correlated to the BW (0.37), *pectoralis minor* muscle weight (0.26), and *pectoralis major* muscle weight (0.33), but was not correlated to fatpad weight. The *pectoralis major* muscle weight, *pectoralis minor* muscle weight were strongly correlated to each other (0.90) and to BW (0.90 and 0.87), respectively. Heart area was correlated to fatpad weight, *pectoralis major* muscle weight, *pectoralis minor* muscle weight, 0.13, 0.44, and 0.41, respectively.

Heart weight as a percent of BW was significantly higher in the feed restricted broilers. Reducing the growth rate by feed restriction allowed heart growth to remain in phase with body growth. Heart characteristics such as RVWT and RVA were also decreased with feed restriction. Large birds with relatively small hearts showing heavy RVWT and large RVA had higher ascitic scores. Limiting weight gain during the starting period significantly alleviated the incidence of ascites. The right ventricle of birds has developed as a volume pump, not as a pressure pump, since it rarely has to respond to changes in pressure (Cartwright *et al.*, 1986), it responds very rapidly to an increased workload (Huchzermeyer *et al.*, 1986).

The interactions between the main effects showed differences in the sensitivity of the RVWT technique of assessing the incidence of ascites compared to the RVA technique of assessing ascites (Figure 2-6 and Figure 2-7). The *ad libitum* - no litter oil treatment birds (1.96 g) and the *ad libitum* - litter oil treatment birds (1.87 g) had significantly higher RVWT than the two feed restriction treatments with litter oiling (1.73 g) and no litter oil (1.64 g) treatment birds. The RVA of the *ad libitum* fed - no litter oil (0.44 cm^2) treatment birds was significantly greater than the RVA of the *ad libitum* fed - litter oil (0.37 cm^2) treatment birds (Table 2-3). The RVA was more sensitive to the interactions between the feed treatment and the litter oiling treatment than the RVWT. The RVA measurements suggest that litter oiling when birds are fed *ad libitum* can improve right ventricle shape by causing a decrease in the RVA.

There were no significant interaction affects between the litter oiling and feed restriction treatments on the fatpad, *minor pectoralis*, and *pectoralis major* weights (Table 2-5). Litter oiling had no significant affect on the measured carcass characteristics. Feed restriction resulted in a significant reduction in fatpad weight (33.14, *ad libitum* fed; 28.44, feed restricted), *pectoralis minor* weight (60.57, *ad libitum* fed; 52.90, feed restricted), and *pectoralis major* weight (255.2, *ad libitum* fed; 220.3, feed restricted). The total breast muscle weight was 42.6 g lower in the feed restricted birds than the *ad libitum* fed birds.

Feed restriction followed by a period of normal feeding reduced carcass fat, *pectoralis minor* weight and *pectoralis major* weight. Energy restriction has been shown to result in a reduction in metabolic energy loss leading to a reduced requirement for maintenance (Plavnik *et al.*, 1985). If during refeeding, this low requirement was maintained, and if growth resumed at a normal or above normal rate (compensatory growth), feed efficiency would be substantially improved. The feed restricted broilers had a significantly lower market weight by 203 g. Although the *pectoralis major* weight was significantly lower in the feed restricted than the *ad libitum* fed treatments, the difference between the weights was only 35 g.

As shown by the growth curves (Figure 2-4), the rate of gain was not significantly different between the two treatments from wk 4 to wk 6. The feed restricted birds were unable to compensate for the limitations on feeding in the starting period. Feed restriction regimens of 7 day or less allow birds time to recover the lost BW gains, but may also result in a higher incidence of ascites syndrome in broilers than the severe feed restriction and an increase in carcass fat compared to the severe feed restriction regimens. Breeding for increased muscle mass has not led to a concomitant increase in the size of the heart and respiratory system, and so the modern broiler has increasing difficulty maintaining oxygen demand for maintenance and growth (Sholsberg *et al.*, 1991).

Oiling the litter did not affect dust levels for the first 2 wk of the experiment. The air quality (respirable dust count in particles/mL) was significantly improved by litter oiling after 2 wk. The dust levels in the oiled pens was significantly reduced after the first 2 wk. Litter oiling

reduced dust levels by 9% in the third wk. Litter oiling continued to reduce dust levels. At wk 4, dust levels, in the pens that received the applications of canola oil to the litter, were reduced by 15%. By wk 5, dust levels were 40% lower in the oiled pens than in the non-oiled pens. At wk 6 litter oiling continued to reduced the dust level by 32% (Figure 2-3).

Although litter oiling did not reduce the average ascites score, litter oiling improved air quality significantly in the pens and also improved heart morphology by reducing the RVA from 0.44 to 0.36 cm^2. The stocking density in this trial was eight broilers/m^2 compared to 15 broilers/m^2 for typical industry stocking density. An increase in stocking density may show a decrease in mortality due to litter oiling. Litter oiling also has a potentially positive effect on humans working in broiler chicken barns.

TABLE 2-1. Description of carcass characteristics related to a specific ascitic score[1]

Score	Identification
0	no lesions or slight hydropericardium and/or slight right hypertrophy or dilation and/or slight pulmonary congestion and edema
2	more pronounced right heart hypertrophy or dilation with more pronounced (moderate) hydropericardium, and congestion of the liver evidenced by darker coloration and rounding of border
3	lesions as in score category 2 with the addition of slight to moderate accumulations of ascitic fluid in one or more ceolomic cavities (other than the pericardium) and irregular raised surfaces on the liver may also have been present
4	marked accumulation of ascitic fluid in one or more ceolomic cavities (other than the pericardium), and also had an extension of the preceding lesions

[1]J. A. Hanson, 1996 (Alberta Agriculture, Food and Rural Development, 6909-116 Street, Edmonton, Alberta, Canada T6H 4P2, personal communication)

TABLE 2-2. BW (g) for feed restricted, ad libitum fed, oiled and non oiled treatments.

treatment		0 week (g)	3 weeks (g)	4 weeks (g)	5 weeks (g)	6 weeks (g)
feed level	ad libitum	39.3 ± 0.16 [a]	702.0 ± 3.84 [a]	1174 ± 6.12 [a]	1725 ± 8.89 [a]	2070 ± 11.75 [a]
	feed restriction	39.1 ± 0.23 [a]	487.8 ± 3.64 [b]	945 ± 5.79 [b]	1498 ±8.42 [b]	1857 ± 11.12 [b]
litter oiling	no oil	39.4 ± 0.21 [a]	602.6 ± 3.75 [a]	1070 ± 5.97 [a]	1620 ± 8.68 [a]	1967 ± 11.46 [a]
	oil	38.9 ± 0.18 [a]	587.2 ± 3.73 [b]	1049 ± 5.94 [b]	1603 ± 8.63 [a]	1960 ± 11.41 [a]
interactions	no oil ad libitum	39.3 ± 0.21 [a]	712.1 ± 5.46 [a]	1190 ± 8.69 [a]	1725 ± 12.63 [a]	2069 ± 16.69 [a]
	no oil feed restriction	39.5 ± 0.38 [a]	493.1 ± 5.14 [b]	950.1 ± 8.17 [b]	1516 ± 11.88 [b]	1865 ± 15.70 [b]
	oil ad libitum	39.2 ± 0.25 [a]	692.0 ± 5.39 [c]	1158 ± 8.58 [c]	1724 ± 12.48 [a]	2071 ± 16.49 [a]
	oil feed restriction	38.6 ± 0.25 [a]	482.5 ± 5.15 [b]	939.2 ± 8.20 [b]	1481 ± 11.92 [c]	1849 ± 15.75 [b]

[a-c] Means in the same column within a main effect or interaction with no common superscript differ significantly ($P < 0.05$). The effect of feed restriction was significant for all BW with the exception of the initial BW.

TABLE 2-3. Heart Characteristics including ascitic score[1], heart area (HA), right ventricle area (RVA), right ventricle weight (RVWT), trimmed heart weight (THWT), percent right ventricle area (PRVA), percent right ventricle weight (PRVW), and heart weight as a percentage of body weight (PHWT) of all birds after slaughter

treatments		score	HA (cm^2)	RVA (cm^2)	RVWT (g)	THWT (g)	PRVA (%)	PRVW (%)	PHWT (%)
feed level	ad libitum	0.87 [a] ± 0.06	3.53 [a] ± 0.03	0.40 [a] ± 0.01	1.92 [a] ± 0.03	7.89 [a] ± 0.06	11.0 [a] ± 0.30	24.0 [a] ± 0.30	0.472 [a] ± 0.004
	feed restriction [2]	0.61 [b] ± 0.06	3.29 [b] ± 0.03	0.35 [b] ± 0.01	1.68 [b] ± 0.03	7.32 [b] ± 0.06	10.0 [b] ± 0.30	23.0 [b] ± 0.30	0.484 [b] ± 0.004
liter oiling	no oil	0.77 [a] ± 0.06	3.44 [a] ± 0.03	0.38 [a] ± 0.01	1.80 [a] ± 0.03	7.59 [a] ± 0.06	11.0 [a] ± 6.0	23.0 [a] ± 0.30	0.477 [a] ± 0.004
	oil [3]	0.72 [a] ± 0.06	3.39 [a] ± 0.03	0.36 [a] ± 0.01	1.80 [a] ± 0.03	7.60 [a] ± 0.06	10.0 [a] ± 6.0	23.0 [a] ± 0.30	0.479 [a] ± 0.004
interactions	no oil ad libitum	0.97 [a]	3.62 [a]	0.44 [a]	1.96 [a]	7.94 [a]	11.6 [a]	2.44 [a]	0.48 [ab]
	no oil feed restriction	0.56 [b]	3.26 [b]	0.33 [b]	1.64 [b]	7.25 [b]	9.90 [b]	2.25 [b]	0.48 [ab]
	oil ad libitum	0.78 [a b]	3.44 [c]	0.37 [b]	1.87 [a]	7.83 [a]	10.2 [b]	23.7 [ac]	0.47 [a]
	oil feed restriction	0.65 [b]	3.34 [b]	0.36 [b]	1.73 [b]	7.38 [b]	10.4 [b]	23.2 [bc]	0.49 [b]
	pooled SEM	0.08	0.04	0.02	0.04	0.08	0.40	0.40	0.50

[1] J. A. Hanson, 1996 (Alberta Agriculture, Food and Rural Development, 6909-116 Street, Edmonton, Alberta, Canada T6H 4P2, personal communication)

[2] Four pens of birds, 400 male broilers, were feed restricted (18g of feed/bird/day from 7 to 15 d of age).

[3] Two restricted and two ad libitum-fed pen received biweekly addition of oil to the litter (totaling 0.8 L/m^2 of oil over 6 wk).

[a-c] Means in the same column within a main effect or interaction with no common superscript differ significantly ($P < 0.05$). The effect of oil was not significant. The effect of feed restriction was significant for all heart characteristics.

TABLE 2-4. Correlation between score, RVA, and RVWT

treatments	n	score[1] and RVA	score and RVWT	RVA and RVWT
all birds	713	0.52	0.50	0.63
no oil & ad libitum	167	0.55	0.46	0.60
no oil & feed restriction[2]	188	0.45	0.47	0.55
oil & ad libitum	171	0.52	0.56	0.66
oil & feed restriction	187	0.53	0.47	0.66

[1] J. A. Hanson, 1996 (Alberta Agriculture, Food and Rural Development, 6909-116 Street, Edmonton, Alberta, Canada T6H 4P2, personal communication)

[2] Two pens of birds for each treatment interaction, a total of four pens, 400 male broilers, were feed restricted (18g of feed/bird/day from 7 to 15 d of age).

[3] Two restricted and two ad libitum-fed pen received biweekly addition of oil to the litter (totaling 0.8 L/m^2 of oil over 6 wk).

TABLE 2-5. Carcass characteristics of selected birds for analysis of fatpad and breast muscle weights with SEM

treatments		BW (g)	fatpad weight (g)	*pectoralis minor* weight (g)	*pectoralis major* weight (g)
feed level	ad libitum	1908 ± 21.3 [a]	33.1 ± 0.9 [a]	60.6 ± 0.9 [a]	255.2 ± 4.2 [a]
	feed restriction [1]	1704 ± 21.2 [b]	28.4 ± 0.9 [b]	52.9 ± 0.9 [b]	220.3 ± 4.2 [b]
litter oiling	no oil	1828 ± 21.6 [a]	31.0 ± 0.9 [a]	57.6 ± 1.0 [a]	240.2 ± 4.2 [a]
	oil [2]	1783 ± 20.9 [a]	30.6 ± 0.9 [a]	55.8 ± 0.9 [a]	235.2 ± 4.1 [a]
interactions	no oil ad libitum	1940 ± 30.2 [a]	32.5 ± 1.3 [ab]	61.5 ± 1.3 [a]	259.0 ± 6.0 [a]
	no oil feed restriction	1717 ± 30.9 [b]	29.6 ± 1.3 [ac]	53.7 ± 1.4 [b]	221.5 ± 6.1 [b]
	oil ad libitum	1876 ± 30.1 [a]	33.8 ± 1.3 [b]	59.6 ± 1.3 [a]	251.4 ± 5.9 [a]
	oil feed restriction	1691 ± 29.1 [b]	27.3 ± 1.3 [c]	52.1 ± 1.3 [b]	219.1 ± 5.7 [b]

[1] Four pens of birds, 400 male broilers, were feed restricted (18g of feed/bird/day from 7 to 15 d of age).

[2] One restricted and one ad libitum-fed pen, over the two periods, received biweekly addition of oil to the litter (totaling 0.8 L/m^2 of oil over 6 wk).

[a-c] Means in the same column within a main effect or interaction with no common superscript differ significantly ($P < 0.05$). The effect of oil was not significant. The effect of feed restriction was significant for all carcass characteristics.

TABLE 2-6. Feed consumption recorded weekly from wk 3 to wk 6 and total feed consumption and feed conversions at 6 wk of age for all treatments.

treatments		feed consumption					Feed Conversion
		wk 3 (kg)	wk 4 (kg)	wk 5 (kg)	wk 6 (kg)	total (kg)	
feed level	ad libitum	59.6 [a]	81.0 [a]	110.6 [a]	82.9 [a]	383.1 [a]	2.36 [a]
	feed restriction [1]	52.4 [a]	73.5 [a]	108.2 [a]	90.8 [a]	351.7 [b]	2.14 [a]
litter oiling	no oil	57.0 [a]	76.0 [a]	108.5 [a]	86.0 [a]	364.0 [a]	2.19 [a]
	oil [2]	55.0 [a]	78.5 [a]	110.4 [a]	87.6 [a]	370.8 [a]	2.32 [a]
	pooled SEM	5.44	5.44	5.44	5.44	6.37	0.095

[1] Two pens of birds, 400 male broilers, were feed restricted (18g of feed/bird/day from 7 to 15 d of age).

[2] One restricted and one ad libitum-fed pen, over the two periods, received biweekly addition of oil to the litter (totaling 0.8 L/m^2 of oil over 6 wk).

[a-c] Means in the same column within a main effect or interaction with no common superscript differ significantly ($P < 0.05$).

TABLE 2-7. The total percentage of mortality for all treatments from day 0 to 42 days of age.

treatments		Mortality (%)
feed level	ad libitum	15.9 [a]
	feed restriction [1]	6.3 [b]
litter oiling	no oil	12.2 [a]
	oil [2]	10.0 [a]
	pooled SEM	3.78
interactions	no oil ad libitum	17.6 [a]
	no oil feed restriction	6.8 [a]
	oil ad libitum	14.2 [a]
	oil feed restriction	5.9 [a]
	pooled SEM	5.35

[1] Two pens of birds, 400 male broilers, were feed restricted (18g of feed/bird/day from 7 to 15 d of age).

[2] One restricted and one ad libitum-fed pen, over the two periods, received biweekly addition of oil to the litter (totaling 0.8 L/m^2 of oil over 6 wk).

[a-c] Means in the same column within a main effect or interaction with no common superscript differ significantly ($P < 0.05$).

FIGURE 2-1. Position of transverse cuts made on the heart.

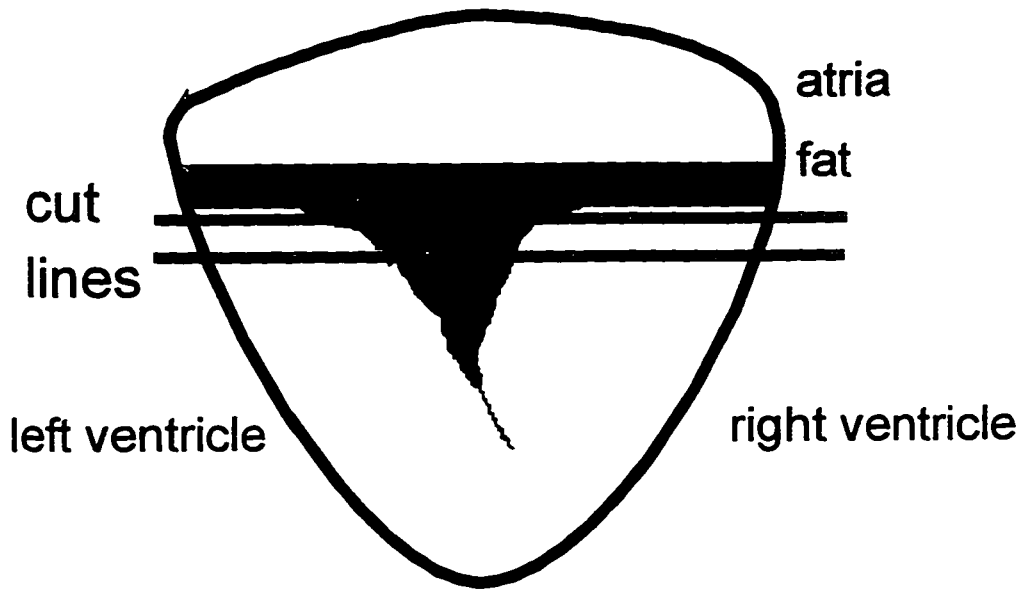


FIGURE 2-2. Cross section of the heart.

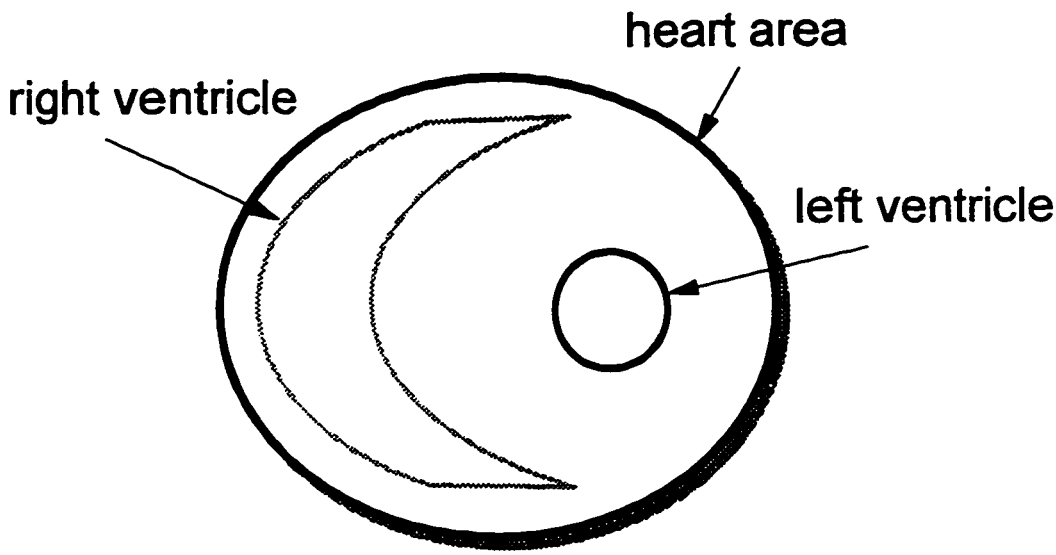


FIGURE 2-3. Dust levels in particles per mL in litter oil treated pens and non-treated pens. Dust levels were significantly less in the oiled pen from wk 3 to wk 6 (P<0.05).

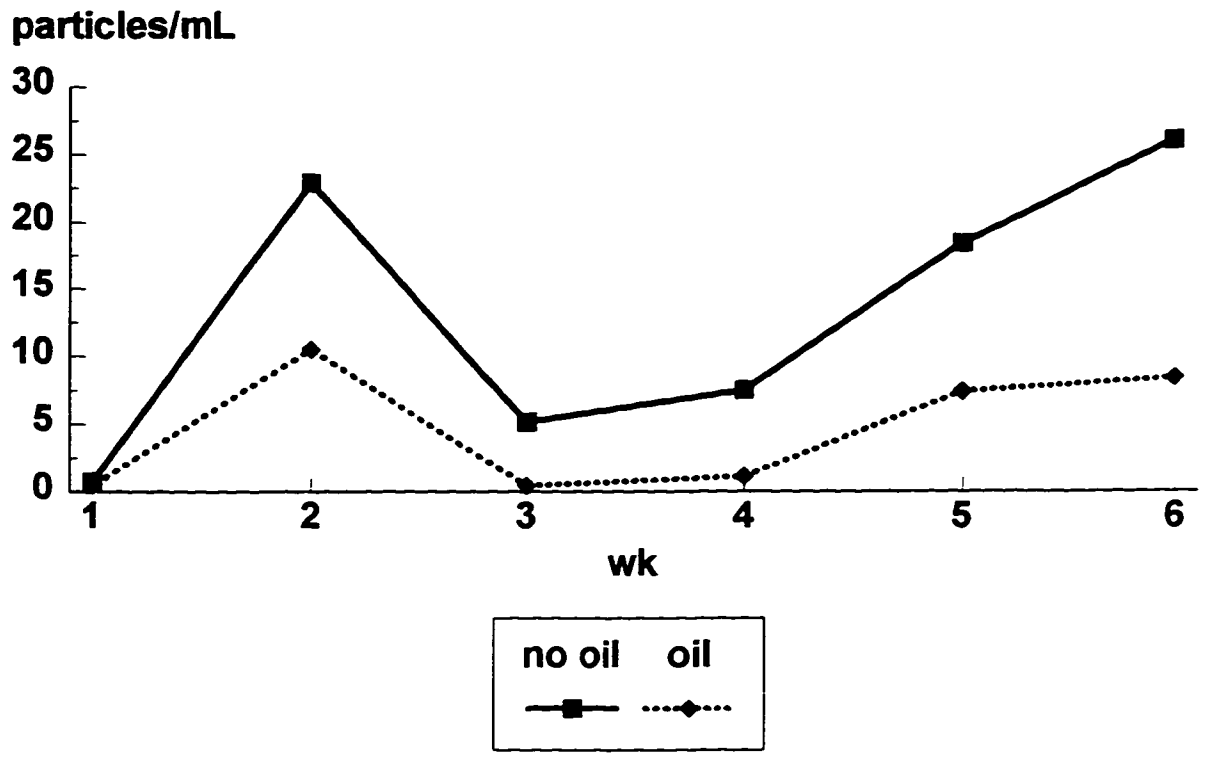


FIGURE 2-4. Growth curves of feed restricted and *ad libitum* fed broilers. Feed restricted broilers had a significantly lower BW than the *ad libitum* fed birds from 3 wk to 6 wk of age (P<0.05).

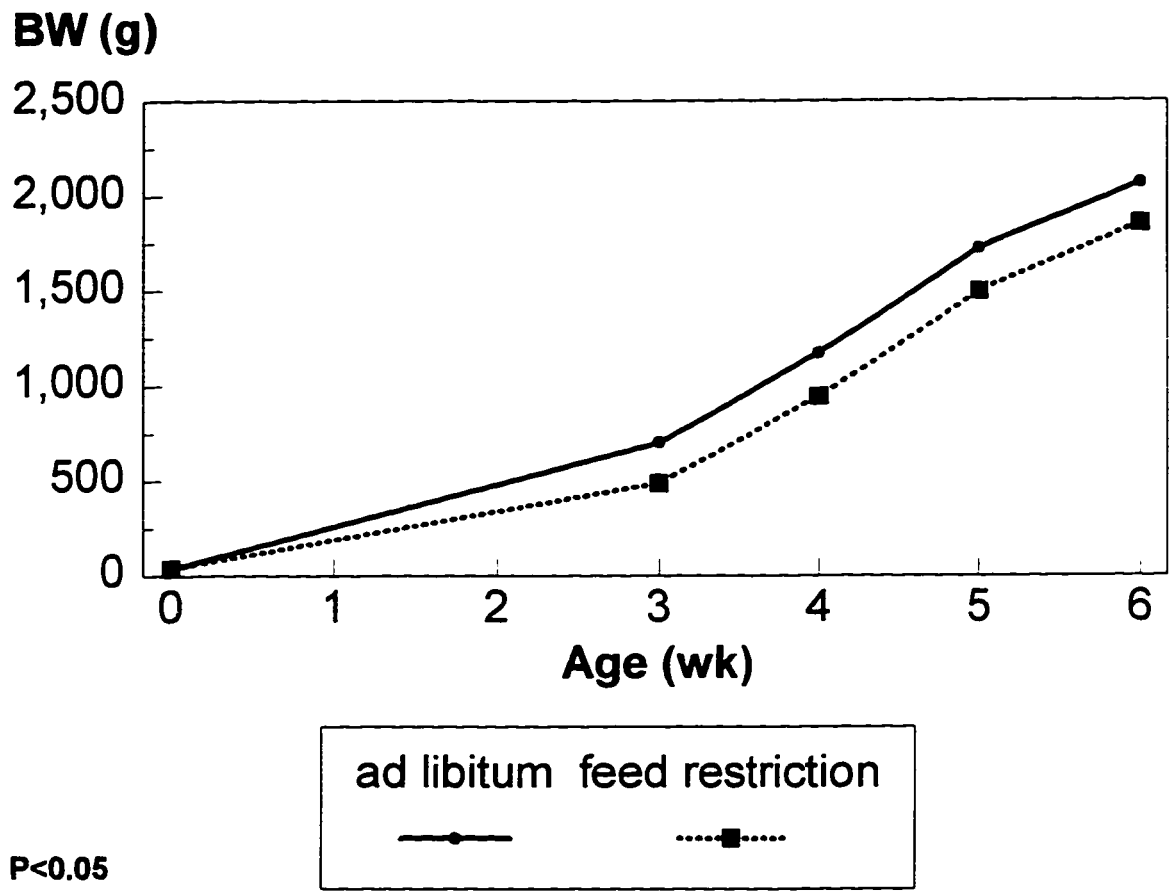


FIGURE 2-5. Scatter plot of the RVA (cm2) against the ascitic score.

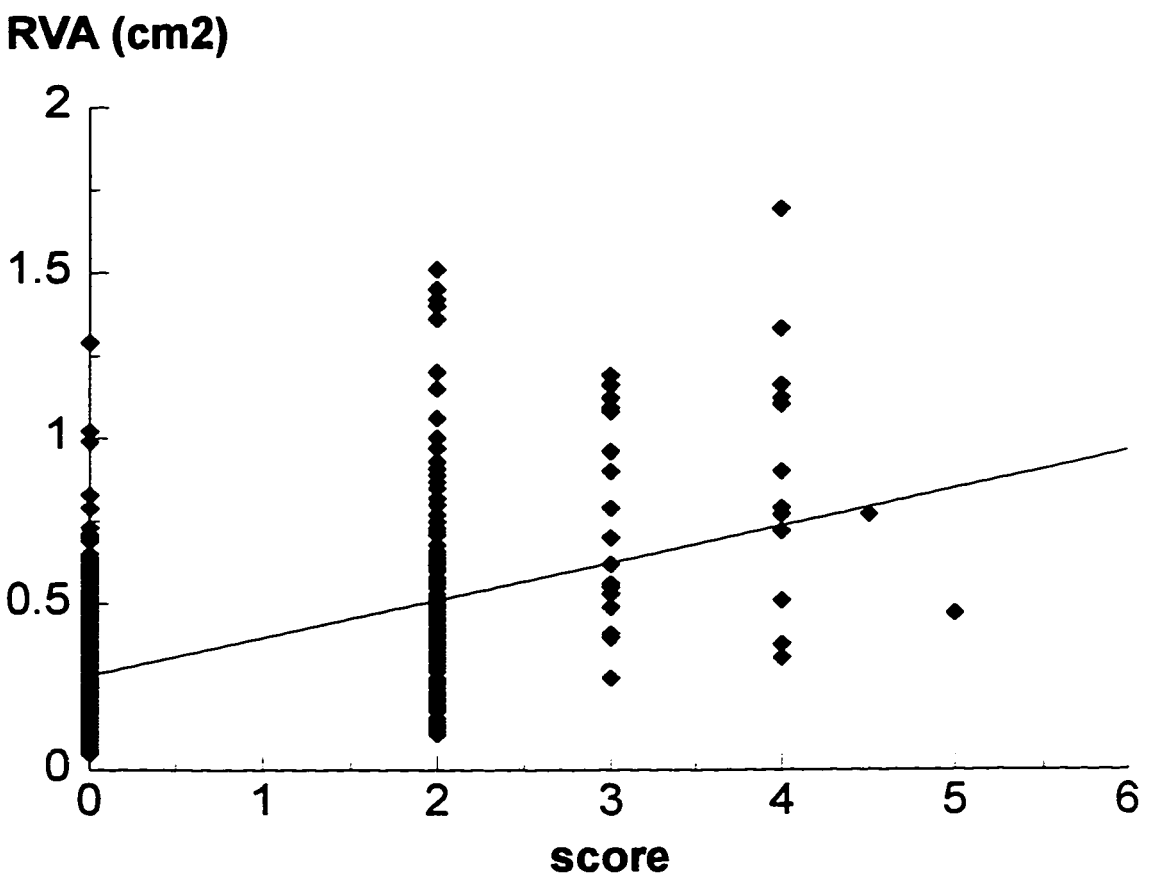


FIGURE 2-6. The RVA (cm2) of the interactions of the main effects including: no oil and *ad libitum* (FF) fed broilers, no oil and feed restricted (FR) broilers, oil and *ad libitum* (FF) fed broilers, oil and feed restricted (FR) broilers.

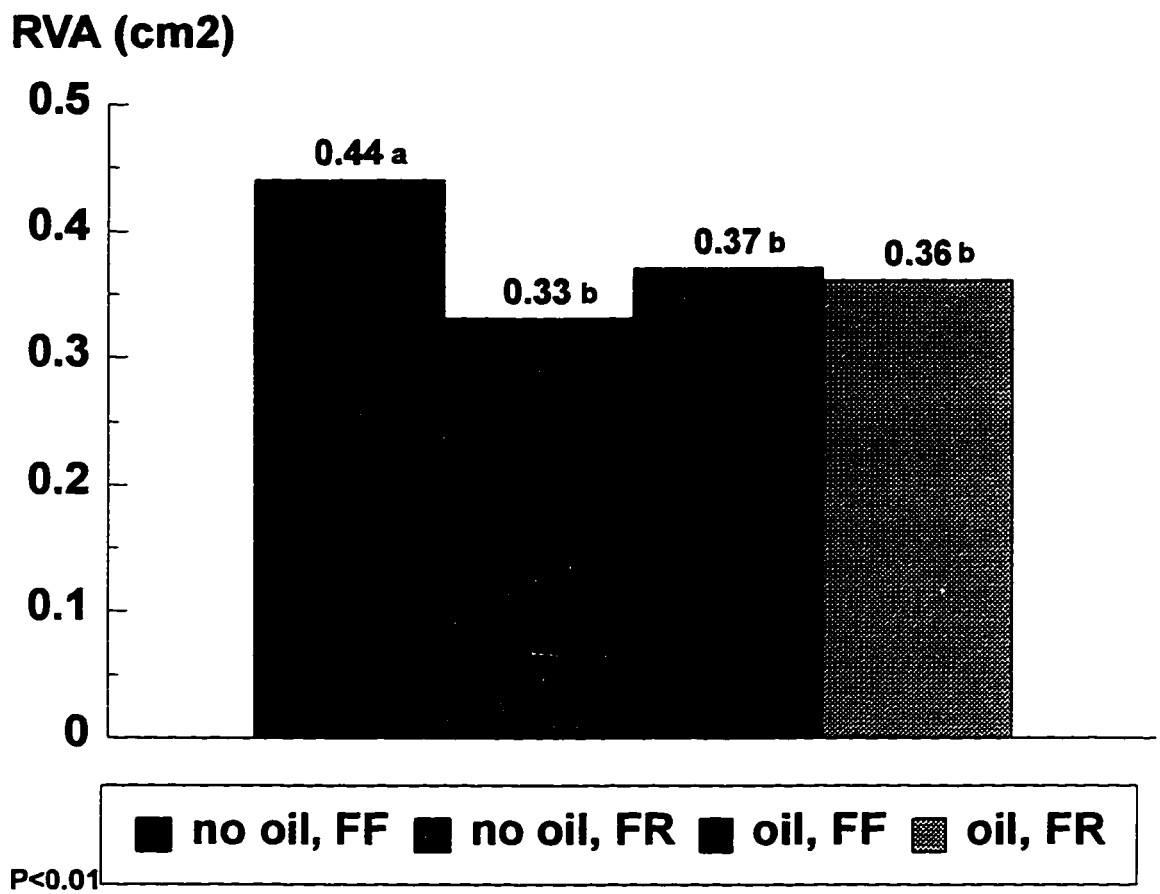
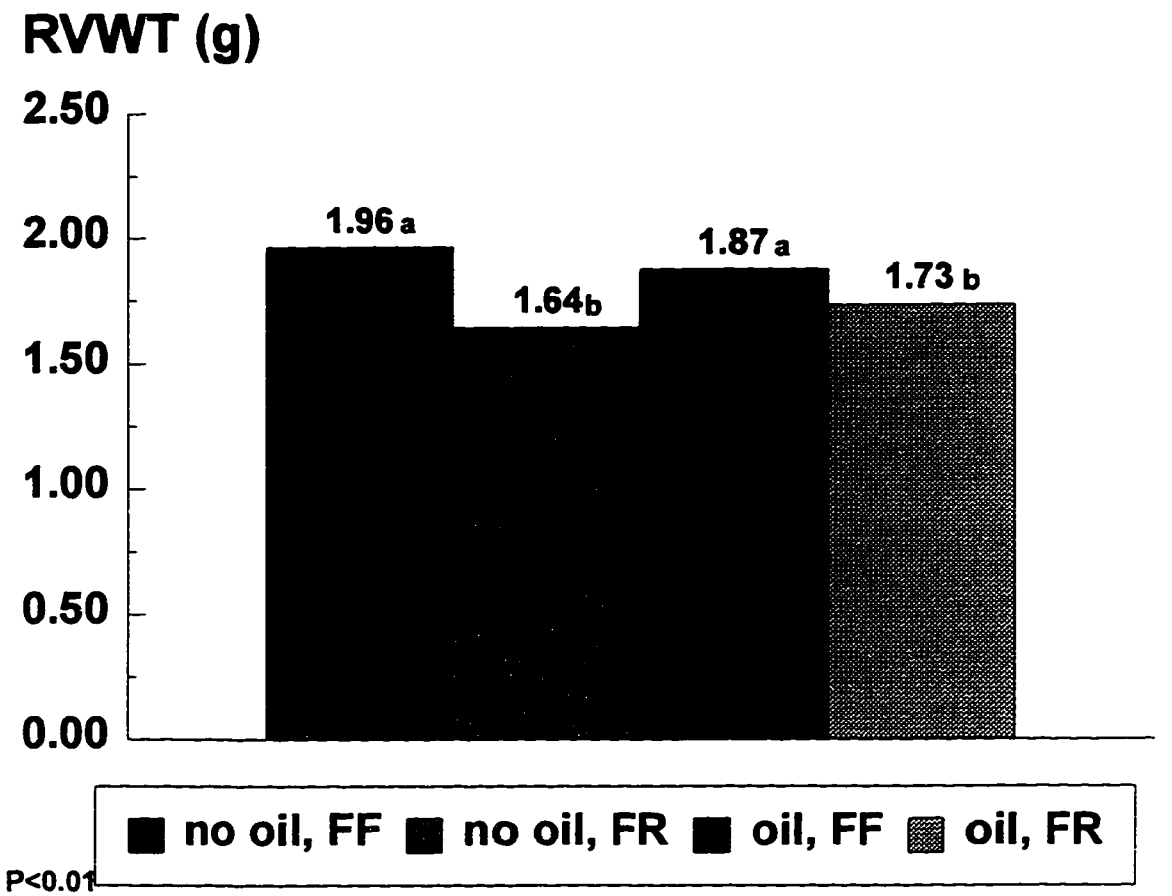


FIGURE 2-7. The RVWT (g) of the interactions of the main effects including: no oil and *ad libitum* (FF) fed broilers, no oil and feed restricted (FR) broilers, oil and *ad libitum* (FF) fed broilers, oil and feed restricted (FR) broilers.



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3. IMAGE ANALYSIS OF THE RIGHT VENTRICULAR AREA TO ASSESS THE SEVERITY OF ASCITES IN BROILER CHICKENS

3.1 INTRODUCTION

Ascites is a condition in which the body cavity is filled with ascitic fluid, leading to death or potential carcass condemnation. It is a symptom of cardiopulmonary insufficiency in rapidly growing broiler chickens (Julian, 1993). Several situations are known to influence the occurrence of ascites in broilers: the housing environment (temperature levels, respirable dust, carbon dioxide and oxygen), rapid growth rates, high BMR, and high energy rations (Scheele *et al.*, 1991; Julian, 1993). Physiologically these conditions can create an oxygen deficit which results in an increased cardiac output. The increase in blood flow results in increased pulmonary arterial pressure. The oxygen deficit causes increased hemoglobin concentration, packed cell volume, and erythrocyte number. These changes result in higher blood viscosity and may lead to pulmonary hypertension (Mirsalimi *et al.*, 1993). Primary pulmonary hypertension triggers a pathophysiological progression leading sequentially to right-sided congestive heart failure, central venous congestion, pressure-induced cirrhosis of the liver, and transudation of fluid into the abdominal cavity (ascites) (Julian, 1993; Wideman *et al.*, 1995).

Historically, the ratio of the right ventricle to the total ventricle mass has been used to assess quantitatively the increased blood pressure on the right ventricle (Julian *et al.*, 1986). As the right ventricle enlarges as a result of increasing blood pressure and increasing blood flow, the volume of blood the right ventricle can hold also increases. The ratio of the right ventricle to the total ventricle mass is a gross indicator of ascites syndrome. The objective of this trial was to test the accuracy and efficiency of using the image analysis technology to quantify the extent of right ventricular expansion in relation to the incidence of ascites syndrome. Although an enlarged ascitic heart has a large right ventricle wall which can be seen with the naked eye, image analysis software can magnify the heart by an additional 40 %, allowing for an accurate

quantification of the RVA. The increased range of RVA values gives a wide distribution that allows the severity of ascites syndrome to be identified.

3.2 MATERIALS AND METHODS

Two flocks of four hundred male broilers were housed in four pens of 100 birds each. All birds received a standard pelleted broiler starter diet (3250 kcal ME/ kg of diet, 23% CP) from 1 to 3 wk of age. A grower diet (3250 kcal ME/ kg of diet, 21% CP) was fed from 4 to 5 wk of age. A finisher diet (3250 kcal ME/ kg of diet, 19% CP) was fed during wk 6. A record of the ambient temperature at bird level and mortality was maintained throughout the trial. All mortality was necropsied to determine cause of death. Hearts of the birds that died during the trial were examined for right ventricular dilation and edema.

Seven hundred and nineteen male broiler hearts were collected from the birds at processing on Day 42. All birds were scored for the incidence of ascites syndrome (Table 2-1). A transverse cut was made on all hearts just below the tricuspid and right atrio-ventricular valves and a second transverse cut was made at the stem of the papillary muscle (Figure 2-1). From the 3 to 5 mm slice, a cross sectional image (Figure 2-2) of each heart was digitally recorded and using image analysis software (Northern Exposure, 1996[1]) the right ventricular area (RVA), left ventricular area (LVA), and total heart area (HA) were determined. The right ventricular wall (RVW) was removed and its weight was determined and expressed as a percentage of total heart weight (PRVW).

To confirm that the shape of the ventricle wall did not significantly affect the RVA, 20 of the heart cross sections were further studied in five RVA categories, ranging from small to large. These were obtained by sorting the heart slices, based on RVA in ascending order and then dividing them into five Groups. Group 1 had a small RVA with an average area of 0.13 cm^2. Groups 2, 3, 4, and 5 had average RVA of 0.20, 0.25, 0.36, and 1.07 cm^2, respectively. To determine the consistency of the right ventricle areas in different placements, each of the four

[1] Empix Imaging Inc., 3065 Ridgeway Drive unit #51, Mississauga, Ontario, Canada, L5L 5M6

hearts was placed beneath the camera and digitally recorded for image analysis four times with four slightly varied right ventricle shapes. Two of the four placements were orientated with the slice atria side up and the other two placements were positioned atria side down (Figure 3-1). Each placement resulted in slight differences in the shape of the extended right and left ventricle walls (Figure 3-2).

The right ventricular wall was removed and its weight (RVW) was compared to the RVA. The RVW was also expressed as a percentage of total heart weight (HWT). A total of 719 male broilers were used for correlation analysis of score to right ventricle area and right ventricle weight.

The data were analyzed by one-way analysis of variance with $P<0.05$ (SAS Institute, 1991). Coefficients of correlation including the score, RVA, and RVW were assessed with $P<0.05$, using SAS (SAS Institute, 1991).

3.3 RESULTS AND DISCUSSION

Heart area varied in size from 2.76 cm^2, in the small RVA Group, to 5.00 cm^2 in the large RVA Group. The LVA was not different between the Groups. The LVA ranged in size from 0.08 to 0.16 cm^2. The RVA, RVW, THW, score, PRVA, PRVW, and PHW were also different between Groups (Table 3-1). These areas and weights were significantly different because RVA Groups were chosen to examine the differences within each Group. Using the data from 719 broilers, the ascites score was found to be significantly correlated to the RVA ($r=0.52$). The ascites score was also significantly correlated to the RVW ($r=0.50$) ($P > 0.0001$). The RVA and the RVW were significantly correlated ($r=0.63$).

All placements produced similar values for LVA. The HA, RVA , or PRVA values within Groups 1, 2, 3, and 4 were not different using the four different placements. The HA, RVA, and PRVA for Group 5 were different with varying placements (Table 3-2). In Group 3, the PRVA were also different using the four placements. Standardizing the placement of the heart cross section eliminated differences in RVA, HA, and PRVA for hearts with large right ventricles. Using

a standard placement of heart cross sections improves the consistency of the areas recorded as an assessment of the severity of ascites syndrome.

The image analysis technology used provided an accurate representation of the heart cross sectional area. The RVA and RVW were correlated to a broilers ascitic score, r=0.52 and r=0.50, respectively). Image analysis was shown to be a valid technique for quantifying the RVA of broiler hearts. Consistent placement is important when heart size or right ventricle size is very enlarged. The image analysis technique shows significant right ventricle enlargements regardless of placement for hearts with large RVA, however, for more accuracy the technique should be standardized. To standardize the technique, the atria side of the heart cross section should be placed upward and the heart shape should be maintained as natural as possible.

Although the image analysis system is relatively expensive and time consuming in comparison to using RVW, the digitally recorded images can be reused and reanalyzed over a long period of time.

TABLE 3-1. Group[1] Averages: Comparison of heart characteristics including the ascitic score, heart area (HA), right ventricle area (RVA), right ventricle weight (RVW), trimmed heart weight (THW), percent right ventricle area (PRVA), and percent right ventricle weight (PRVW) of the 20 selected birds

Group	score [2]	HA (cm^2)	LVA (cm^2)	RVA (cm^2)	RVW (g)	THW (g)	PRVA (%)	PRVW (%)
1	0.0 [a]	2.76 [a]	0.08 [a]	0.10 [a]	1.33 [a]	6.40 [a]	0.04 [a]	0.21 [a]
2	0.0 [a]	2.86 [b]	0.11 [a]	0.25 [b]	1.58 [ab]	7.10 [ab]	0.09 [b]	0.22 [ab]
3	0.5 [a]	3.25 [c]	0.13 [a]	0.35 [c]	1.90 [b]	7.38 [ab]	0.11 [b]	0.25 [b]
4	1.5 [b]	3.53 [d]	0.16 [b]	0.53 [d]	2.05 [b]	8.28 [bc]	0.15 [c]	0.25 [ab]
5	2.0 [b]	5.00 [e]	0.13 [c]	1.42 [e]	2.85 [c]	9.00 [c]	0.29 [d]	0.32 [c]
pooled SEM	0.32	0.23	0.22	0.01	0.17	0.43	0.01	0.01

[1] Group 1, RVA 0.13 cm^2; Group 2, RVA 0.20 cm^2; Group 3, RVA 0.25 cm^2; Group 4, RVA 0.36 cm^2 ; Group 5, RVA 1.07 cm^2 .

[2] J. A. Hanson (1996, Alberta Agriculture, Food and Rural Development, 6909-116 Street, Edmonton, Alberta, T6H 2P4, personal communication)

[a-c] Means in the same column with no common superscript differ significantly ($P<0.05$).

TABLE 3-2. Comparisons heart placement by Group [1] of heart area (HA), right ventricular area (RVA), left ventricular area (LVA), and percent right ventricular area (PRVA)

Group	placement [2]	HA (cm^2)	RVA (cm^2)	LVA (cm^2)	PRVA (%)
1	1	2.51 [a]	0.13 [a]	0.10 [a]	0.05 [a]
	2	2.53 [a]	0.16 [a]	0.13 [a]	0.06 [a]
	3	2.61 [a]	0.19 [a]	0.12 [a]	0.07 [a]
	4	2.64 [a]	0.17 [a]	0.12 [a]	0.06 [a]
2	1	2.71 [a]	0.20 [a]	0.09 [a]	0.08 [a]
	2	2.71 [a]	0.22 [a]	0.10 [a]	0.08 [a]
	3	2.79 [a]	0.25 [a]	0.12 [a]	0.09 [a]
	4	2.79 [a]	0.27 [a]	0.11 [a]	0.10 [a]
3	1	3.16 [a]	0.25 [a]	0.12 [a]	0.08 [a]
	2	3.15 [a]	0.32 [a]	0.11 [ab]	0.10 [ab]
	3	3.16 [a]	0.28 [a]	0.11 [ab]	0.09 [ab]
	4	3.19 [a]	0.37 [a]	0.10 [b]	0.12 [b]
4	1	3.26 [a]	0.35 [a]	0.18 [a]	0.11 [a]
	2	3.23 [a]	0.37 [a]	0.20 [a]	0.11 [a]
	3	3.33 [a]	0.43 [a]	0.18 [a]	0.13 [a]
	4	3.29 [a]	0.43 [a]	0.15 [a]	0.13 [a]
5	1	4.56 [a]	1.07 [a]	0.16 [a]	0.23 [a]
	2	4.39 [b]	1.07 [a]	0.15 [a]	0.24 [a]
	3	4.63 [a]	1.26 [b]	0.17 [b]	0.27 [ab]
	4	4.70 [a]	1.32 [b]	0.15 [b]	0.28 [b]
Pooled SEM		0.05	0.04	0.01	0.01

[1] Group 1, RVA 0.13 cm^2; Group 2, RVA 0.20 cm^2; Group 3, RVA 0.25 cm^2; Group 4, RVA 0.36 cm^2 ; Group 5, RVA 1.07 cm^2 .

[2] Placement is the orientation of the heart for image analysis. The four placements include two orientations with the atria side up, and two orientations with the atria side down, ventricle upward. Each placement had slight differences in the shape of the right and left ventricle wall.

[a-c] Means in the same column within a Group with no common superscript differ significantly ($P<0.05$). All Groups were significantly different from one another.

FIGURE 3-1. Orientation of heart slice for placement. Two of the four heart placements were orientated with the atria side up and two of the four heart placements were orientated with the ventricle side up.

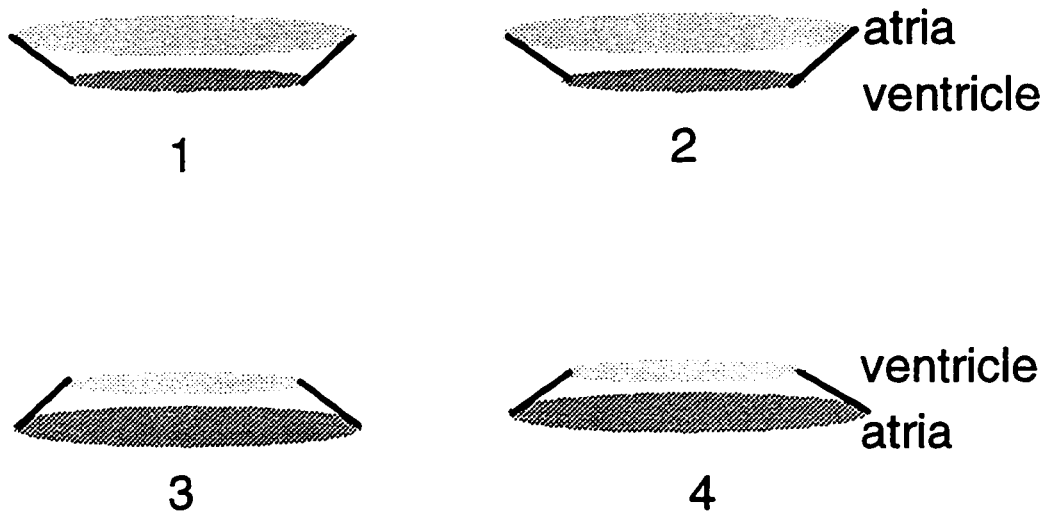
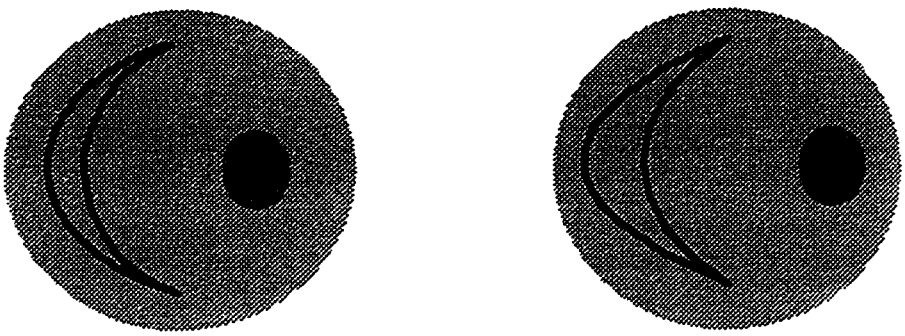


FIGURE 3-2. Extension of the right ventricle walls for placement technique comparison.



1

RVA = 0.10

2

RVA = 0.14

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4. GROWTH PERFORMANCE, CARCASS CHARACTERISTICS, AND THE INCIDENCE OF ASCITES IN BROILER CHICKENS IN RESPONSE TO TEMPERATURE FLUCTUATIONS AND LITTER OILING

4.1 INTRODUCTION

General agreement exists that pulmonary hypertension triggers a pathophysiological progression leading sequentially to right-sided congestive heart failure, central venous congestion, pressure induced cirrhosis of the liver, and transudation of fluid into the abdominal cavity (ascites) (Ploog, 1973; Cueva *et al.*, 1974; Huchzermeyer and DeRuyck, 1986; Hernandez, 1987; Julian, 1988, 1993; Peacock *et al.*, 1989, 1990; Wideman and Bottje, 1993). Right ventricular failure and ascites are a response to increased workload by the right ventricle as a result of pulmonary hypertension. Pulmonary hypertension may develop because of insufficient blood capillary capacity in the lung (Julian *et al.*, 1987). Rapidly growing broiler chickens have a high metabolic requirement for oxygen that require a high volume of blood flow through their lungs (Julian, 1989). Hypertrophy of the right ventricular wall is directly related to pulmonary hypertension and the ratio of the right ventricle to the total ventricle mass which can be used as a measure of the increased pulmonary arterial pressure load on the right ventricle which causes dilation of the chamber.

There are a number of factors which are known to influence the incidence of ascites in broiler chickens such as the housing environment including factors such as temperature levels (cold, or moderate heat), dust, air quality, carbon dioxide levels, and oxygen levels. As well as increasing oxygen consumption, lower outside temperatures can result in reduced ventilation and lower oxygen concentrations in broiler houses. Cold ambient temperatures increase the oxygen requirement, cardiac output, blood flow, and may result in increased pulmonary arterial pressure overload on the right ventricle (Julian *et al.*, 1989). A reduction in environmental temperature from 20 ^{0}C to 2 ^{0}C for example, almost doubles the oxygen requirement in adult female White Leghorn hens (Gleeson, 1986). May and Deaton (1975) showed that exposure to cold

temperatures increased the heart weight of chickens, probably as a result of the increased work-load on both the ventricles.

Environmental stress which accompanies an increased oxygen demand may challenge the broiler beyond its ability to adapt. Respirable dust can cause irritation of the respiratory tract due to its high protein content (Feddes *et al.*, 1992). This affects the health and performance of turkeys (Feddes *et al.*, 1992). Broilers could have an increased susceptibility to ascites because the respirable dust particles affect the oxygen transfer in the lung. Oxygen deficit is the foundation for an increased cardiac output and often leads to ascites in broilers. The removal of dust particles should improve oxygen and carbon dioxide transfer in fast growing male broilers since they require large quantities of oxygen and have limited pulmonary diffusion capacity (Julian *et al.*, 1986). If broilers exhibiting lung capacity deficiencies encounter factors that interfere with oxygen transport, breathing ability, or cardiac output, they could develop hypoxia as a primary stimulus of the ascites syndrome (Anthony *et al.*, 1994; Balog *et al.*, 1994). The primary objective of this study was to discover the effect of diurnal temperature fluctuations and litter oiling at a stocking density of 12 broilers/m^2 (increased from 8 broilers/m^2 in the previous study) on the carcass characteristics and heart characteristics of broiler chickens and relate those to differences in the incidence of ascites.

4.2 MATERIALS AND METHODS

Six hundred male Hubbard High Y (high breast yielding) broilers were housed in four pens of 150 birds each. A total of 1200 broiler chickens were studied within two 6 wk periods. At 1 d of age, all birds were wing-banded for identification. All broilers were individually weighed weekly from 1 d of age until 6 wk of age. Two pens of birds were managed so that they experienced temperature fluctuations. The temperature treatment consisted a fluctuation of 3^{0}C in temperature above the required temperature during the day (6:00 to 18:00 h) and 3^{0}C below the required temperature at night (18:00 to 6:00 h) (a 6^{0}C change in daily temperature) (Figure 4-1, theoretical and actual temperatures recorded in the first period; Figure 4-2, actual

temperatures recorded in the second period). All pens had the same mean temperature. One control temperature pen and one fluctuation temperature pen received biweekly application of canola oil to the litter (1.1 L/m^2 of oil over 6 wk) in one period. All birds received a standard crumble broiler starter diet (3250 kcal ME/ kg of diet, 23% CP) from 1-3 wk of age. A grower diet (3250 kcal ME/ kg of diet, 21% CP) was fed from 4-5 wk of age. The finisher diet (3250 kcal ME/ kg of diet, 19% CP) was fed during wk 6. Concentrations of airborne dust were measured weekly in each pen (Figure 4-3) using the Climet Air Sampler. A record of the mortality was also maintained throughout the trial. All dead birds were necropsied to determine cause of death and heart morphology.

At 6 wk of age, 30 birds from each pen were killed for determination of weight of the breast, fatpad, and heart. Hearts were collected from the remaining surviving birds at slaughter. All birds were scored for the incidence of ascites (Table 2-1). A total of 1045 hearts were processed for measurement of the right ventricular area (RVA) using image analysis technology (12 hearts were not suitable due to processing problems at the processing plant and the remaining 143 birds died during the experiment). A transverse cut was made on all hearts just below the tricuspid and bicuspid valves and a second transverse cut was made at the stem of the papillary muscle (Figure 2-1). From the 4 mm heart slice, a cross sectional image of each heart was digitally recorded and using image analysis software (Northern Exposure, 1996[1]) the RVA, left ventricle area (LVA), and total heart area (HA) were determined (Figure 2-2). The right ventricular wall was removed and its weight (RVWT) was compared to the RVA. The RVWT was also expressed as a percentage of total heart weight (PRVW). After slaughter, the heart characteristics including the ascitic score, HA, RVA, RVWT, THWT, PRVA, PRVW, and PHWT of all birds were assessed.

Two hundred and forty broilers were selected, from the 1200 broilers used, for further analysis of carcass characteristics. The broilers used for further analysis were selected on the basis of BW gain. At 6 wk of age, 30 birds were selected using the broilers BW gain in each pen as the distribution, (10 birds per weight gain category: small weight gain [214 g], medium weight

[1] Empix Imaging Inc., 3065 Ridgeway Drive, Unit #51, Mississauga, Ontario, L5L 5M6

gain [496 g], and large weight gain [564 g]) from each pen. The fatpad, *minor pectorals* muscle, and *major pectorals* muscle were removed from the 240 broilers for a comparison of the weights. The data were analyzed by analysis of variance and (SAS Institute, 1991) with $P<0.05$. Coefficients of correlation were obtained using SAS with $P<0.05$ (SAS Institute, 1991).

4.3 RESULTS AND DISCUSSION

Initial chick body weights was the same for all treatments. At 3 wk of age, the broilers receiving the application of canola oil to the litter had a significantly lower body weight gain than the birds that did not receive litter oiling treatment (Table 4-1). The gain from wk 1 to wk 2 was significantly greater in the non-litter oiling treatment (240 g) than the litter oiled treatment (235 g) (Table 4-2). In wk 3, the non-litter oiling treatment gained 35 g more than the oiled treatment. At wk 4, the non-litter oiling treatment was significantly heavier (1218 g) than the litter oiling treatment (1171 g), this continued in wk 5 (1831 g compared to 1785 g) (Figure 4-4). There were no differences in feed conversions at 6 wk of age and there were no differences in feed consumption between treatments from wk 1 to wk 6 overall and there were no differences on a weekly basis (Table 4-3). The sudden decrease in temperature seen at day 21 (Figure 4-2) was due to a malfunction in equipment and was corrected to that required within 6 h of the lowest recorded temperature.

The final body weights of the broilers were significantly different, the litter oiling treatment (2249 g) had lower weight gain compared to the non-litter oiled treatment (2293 g) (Figure 4-5). Two possibilities for the reduction in the final weights of the litter oiling treatment broilers are litter pecking and heat loss. Broilers reared on a suspended metal floor have a significant increase in growth and there is less foreign material in the gizzard of broilers reared on a suspended metal floor than those reared on litter (Malone *et al.*, 1983). Therefore the reduced weight gain may be due to the broilers eating the straw after application of canola oil to the straw litter. Ground-pecking in poultry is part of the feeding system and is mostly directed onto edible material, although the tendency to peck at inedible objects remains high throughout

life (Blokhuis, 1986). Litter floors have a higher incentive value compared to floors without litter (slats or wire) because of the visual, tactile and gustatory properties of litter, as well as the long-term effects of ingestion of litter (Blokhuis and Van Der Haar, 1989). It is unlikely that the broilers in this case were eating a significant amount of litter because the feed was supplied *ad libitum,* the straw was very course at 3 wk of age, and there were no differences in feed consumption (Table 4-3).

It is more likely, however, that the broilers subjected to the litter oiling treatment may have had a higher heat loss due to the covering of canola oil. Although the canola oil is applied directly to the litter, the broilers attain a low level of coverage due to daily activity. Plumage contributes considerably to temperature regulation. Oily feathers cause a decrease in thermal resistance because the oil reduces the ability of the feathers to reduce convective heat loss by trapping air. Using the required temperatures for optimal broiler growth can result in poor feathering and cause an increase in the convective heat loss of broilers. The higher heat losses experienced by the broilers in the litter oiling treatment may have occurred within the starting period and by wk 3 the differences in body weight were unrecoverable with the continued application of the canola oil to the litter. It may be beneficial to the broiler to refrain from using canola oil on the litter until 3 wks of age. Further research is required to assess the differences in the initiation time of litter oiling and the benefits in litter oiling from wk 3 to wk 6.

In the previous experiment "Feed Restriction and Litter Oiling", the stocking density was 8 broilers/m^2 and the canola oil was applied at a rate of 0.8 L/m^2 (0.1 L/broiler over the 6 wk period), because the typical industrial stocking density is much greater than eight broilers/m^2 for this experiment the stocking density was increased to 12 broilers/m^2. The increased stocking density produced a need for an increase in the rate of application of canola oil to the litter to 1.1 L/m^2 over the 6 wk period (0.09 L/broiler over the 6 wk period). This level of litter oiling reduced the respirable dust in the pens to a maximum of 2.5 particles/mL. Although the application rate was decreased from 0.1 L/broiler to 0.09 L/ broiler, the overall increase in the rate of application of canola oil to the litter was detrimental to the growth rate of the broilers. To prevent heat loss

leading to lower body weight gains the maximum rate of canola oil application should not exceed 0.8 L/m^2.

Litter oiling did not result in a decrease in the RVA of the broilers (Table 4-4), as it did in a previous study (Feed Restriction and Litter Oiling). This could be due to the increased cold stress due to the lower thermal resistance of the feathers due to oil coverage on the bird. The total heart weight (THWT) as a percentage of total BW (PHWT) was significantly lower in broilers in the non-oiled treatment. The application of canola oil to the litter did not affect the RVWT or the PRVA as it did in the previous experiment. The score, HA, RVA, THWT, and PRVA were not significantly affected by either application of canola oil to the litter or temperature fluctuations.

As shown by the scatter plot (Figure 4-8), there is a large variation in RVA within an ascities score, assessed at the time of processing. There is a trend, however, that shows that the increase in the RVA corresponds to an increase in ascitic score. The small RVA that have high ascitic scores are due the variation seen in biological systems. The ascitic score is a gross estimate of the degree of ascites in a flock based on heart and liver characteristics. A greater range of ascites is gained by using the ascitic score, however, there are a number hearts with enlarged RVA or increased RVWT which may in turn deserve to be classified with a higher degree of ascites. Some hearts that were classified with an ascitic score of 2 may not yet be showing outward signs (body weight reduction, Figure 4-7) of edema or fluid accumulation and are on the verge of right ventricular failure due to the degree of right ventricular dilation. The comparison of the technique of using the percent RVWT as an assessment of the degree of ascites to the RVA produces a strong correlation (Figure 4-9). The RVA increases as the RVWT increases.

Although dust can negatively affect rate of gain, thereby decreasing revenues of turkey production (Feddes *et al.*, 1992), results of the present study showed that litter oiling, which significantly reduced dust had a negative affect on the growth of male broilers and did not affect the RVA or RVWT. Dust particle concentrations can reach a maximum of 44 particles/mL in the respirable range late in the growth cycle of turkeys (Feddes *et al.*, 1992). For this trial, the

maximum dust particle concentrations reached were 24.2 particles/mL. Air quality is recognized as having a significant affect on animal health, performance, and product quality in intensive animal production (Glennon *et al.*, 1989). The application of canola oil to the litter significantly improved air quality in the barn as evidenced by the reduction in respirable dust particles in the air, but any improvement in animal health, performance, or product quality was not seen due to the apparently excessive rate of canola oil application.

The broilers selected for analysis of carcass characteristics exhibited no difference in BW within a main effect or interaction. The fatpad, lung, *pectoralis minor*, and *pectoralis major* weight were not significantly different between the litter oiled pens and non-litter oiled pens (Table 4-5). The shank length of the litter oiled treatment was significantly shorter than the non-litter oiled treatment. Temperature fluctuations did not produce significant differences in the fatpad weight, shank length, or lung weight. The *pectoralis minor* and *pectoralis major* weight were significantly heavier in the temperature fluctuation treatment than the control temperature treatment by 3.0 g and 12.0 g respectively. The increase in *pectoralis minor* and *pectoralis major* weight could be accounted for by the slight increase in BW, although the difference was not significant. The temperature fluctuation treatment birds had slightly heavier body weights (12 g). The broilers in the temperature fluctuation treatment also had a slightly higher feed consumption 15.8 g, though it was not significant because the level of feed consumption was high, it may account for the increase in breast muscle weight.

Carcass composition is affected by environmental temperature (Leenstra and Cahaner, 1991). Selection and management in broiler production is increasingly aimed at high meat yield (protein deposition) and low fat deposition. At increasing temperatures, fat and energy deposition in the carcass are increased. Temperatures below the lower critical temperature stimulate feed intake and weight gain, especially with high-energy diets. Scheele *et al.* (1987) reported that a significantly higher fraction of the energy was retained as protein at lower temperatures than at moderate temperatures. Low temperatures act as stimulators in protein accretion, however, at higher energy costs (Leenstra and Cahaner, 1991).

In pens with no oil and temperature fluctuations the *pectoralis minor* and *pectoralis major* muscle weights were heavier than those in pens that were oiled and were subjected to the control temperature by 4.0 g (P = 0.0214) and 20.9 g (P = 0.0483), respectively. The non-litter oiled and control temperature treatment and the litter oiled and temperature fluctuation treatment showed no differences from any other treatment combination. Temperature fluctuations caused an increase in *pectoralis minor* and *pectoralis major* muscle weight and have a greater final weight when the litter was not treated with canola oil most likely because of the convective heat losses associated with the litter oiling treatment.

The birds subjected to the control temperature treatment had a lower RVWT than the birds subjected to the fluctuating temperature treatment. The birds subjected to the control temperature treatment also had a decreased PRVW and PHWT. The ascites score was significantly correlated to the RVA (r=0.41) and RVWT (r=0.43). The RVA and the RVWT were also significantly correlated (r=0.54).

Due to environmental changes in outside temperature between the day temperatures and night temperatures, regulating barn temperatures can be difficult. Barn temperatures can drop at night with the ambient temperature. Under-sized heating equipment combined with heat loss from the ventilation system, when minimums are set too high, can cause a 6 °C shift in temperature between day and night temperature in the barn. Cool environmental temperatures ranging from 15 to 10 °C from 3 wk of age have been shown to induce ascites (Wideman *et al.*, 1995). Factors such as cold, which increase oxygen requirement, also increase cardiac output and blood flow and may result in increased pulmonary arterial pressure and cause both a volume and a pressure overload in on the right ventricle (Julian *et al.*, 1989). A nightly decrease of 6°C caused an increase in RVWT, PRVW, and PHWT (Table 4-4).

Chicks have a very narrow thermoneutral zone and any temperature below an optimum increases the oxygen requirement (Julian *et al.*, 1989). Even a 6°C temperature fluctuation may cause stress to the heart. Fluctuation temperatures did not negatively affect the RVA possibly because the mean temperatures were the same for both temperature treatments. Severe

temperature fluctuations did not affect performance, however, temperature fluctuations cause an increase in mortality of 1.4 %.

The increase in oxygen requirement caused by the additional heat loss may have a marked affect on the incidence of ascites in meat-type chickens. This could be a significant factor at high altitude where less oxygen is available for respiration and where broilers may be housed in unheated, open-sided buildings even though the temperature drops markedly at night (Julian *et al.*, 1989). The thermal insulation afforded by the feathers is an important factor in determining the zone of thermoneutrality. In a group of birds, social factors also affect the zone of thermal neutrality. By huddling, the bird is better able to withstand low environmental temperatures. Fowl show pronounced diurnal rhythm in its metabolic rate, accompanied by a corresponding rhythm in the deep body temperature. The metabolic rate increases proportionally to the reduction in environmental temperature until it reaches a maximum (Freeman, 1971).

Growth rates of chicks are adversely affected by cold drafts (air circulation in the barn), probably because heat loss is accelerated under these conditions and energy otherwise available for growth is used to maintain the body temperature. A decrease in temperature causes an increase in metabolism encouraging the broilers to consume food slightly more feed (15.8 g) though it was not significant. The feed consumed may have be partitioned differently due to the temperature fluctuations causing an increase in *pectoralis major* and *pectoralis minor* muscle mass.

The composition of the gained body weight during growth is influenced by the environmental temperature. For example, the amount of fat stored per gram body weight increase was maximal at an environmental temperature of 32 ^{0}C and was less at temperatures of 21 ^{0}C or 40 ^{0}C (Sturkie, 1986). There was no significant difference between the control temperature treatment fatpad weight and fluctuating temperature treatment fatpad weight because the environmental temperatures were not as severe as in Sturkie (1986).

The majority of mortality overall was due to ascites (Figure 4-6). The majority of mortality for the temperature fluctuation treatment and the litter oiling treatment was due to ascites. There was significantly more deaths of broilers in the temperature fluctuation treatment

pens, 12.3 %, than in the control temperature pens, 10.9 % (Table 4-6). Although fluctuating temperatures were expected to produce an increase the mortality due to ascites, the increase was not significant (6.3 % temperature fluctuations and 4.6 % control temperature). The mortality for the birds subjected to the litter oiling treatment was greater from ascites than all other categories of mortality including other causes of mortality such as peritonitis, omphilities, liver hemorrhage, dehydration, cull (runt), hepatitis, anemia, or intestine rupture. The non-litter oiling treatment birds had similar rates of mortality due to ascites and sudden death syndrome (4.7 % death due to ascites compared to 4.4 % deaths due to sudden death syndrome). Litter oiling did produce a decrease in mortality due to sudden death syndrome suggesting that the left ventricle may be more reactive to respirable dust than the right ventricle of the broiler heart.

TABLE 4-1. BW (g) for temperature fluctuation, control temperature, oiled and non oiled treatments

treatments		0 wk (g)	1 wk (g)	2 wk (g)	3 wk (g)	4 wk (g)	5 wk (g)	6 wk (g)
temperature	control temperature	41.6 ± 0.14 [a]	143.0 ± 0.9 [a]	381.4 ± 2.0 [a]	719.4 ± 3.7 [a]	1192 ± 6.2 [a]	1808 ± 9.6 [a]	2265 ± 13.3 [a]
	temperature fluctuations	41.8 ± 0.14 [a]	144.2 ± 0.8 [a]	380.7 ± 2.0 [a]	729.5 ± 3.7 [a]	1196 ± 6.1 [a]	1808 ± 9.5 [a]	2277 ± 13.1 [a]
litter oiling	no oil	41.7 ± 0.14 [a]	143.3 ± 0.9 [a]	383.3 ± 2.0 [a]	744.1 ± 3.7 [a]	1218 ± 6.2 [a]	1831 ± 9.6 [a]	2293 ± 13.3 [a]
	oil	41.7 ± 0.14 [a]	143.9 ± 0.8 [a]	378.8 ± 2.0 [a]	704.9 ± 3.7 [b]	1171 ± 6.1 [b]	1785 ± 8.6 [b]	2249 ± 13.1 [b]
interactions	no oil control temperature	41.3 ± 0.20 [a]	140.7 ± 1.2 [ac]	379.3 ± 2.8 [ac]	739.3 ± 5.2 [a]	1223 ± 8.8 [a]	1828 ± 13.6 [a]	2279 ± 18.8 [ab]
	no oil temperature fluctuations	42.1 ± 0.20 [b]	146.0 ± 1.2 [b]	387.2 ± 2.8 [b]	749.0 ± 5.3 [a]	1212 ± 8.8 [a]	1835 ± 13.7 [a]	2307 ± 18.9 [a]
	oil control temperature	41.8 ± 0.20 [ab]	145.3 ± 1.2 [bd]	383.5 ± 2.8 [ab]	699.6 ± 5.3 [b]	1161 ± 8.9 [b]	1789 ± 13.7 [b]	2252 ± 18.9 [b]
	oil temperature fluctuations	41.6 ± 0.19 [ab]	142.5 ± 1.2 [cd]	374.2 ± 2.7 [c]	710.1 ± 5.1 [b]	1181 ± 8.5 [b]	1782 ± 13.2 [b]	2246 ± 18.2 [b]

[a-c] Means in the same column within a main effect or interaction with no common superscript differ significantly ($P < 0.05$). The effect of feed restriction was significant for all BW with the exception of the initial BW.

TABLE 4-2. BW gain (g) for temperature fluctuation, control temperature, oiled and non oiled treatments

treatment		1 wk (g)	2 wk (g)	3 wk (g)	4 wk (g)	5 wk (g)	6 wk (g)
temperature	control temperature	101.4 ± 0.83 [a]	238.5 ± 1.38 [a]	338.0 ± 2.32 [a]	472.7 ± 3.27 [a]	616.3 ± 4.53 [a]	456.7 ± 6.30 [a]
	temperature fluctuations	102.4 ± 0.82 [a]	236.5 ± 1.36 [a]	348.8 ± 2.28 [b]	466.9 ± 3.22 [a]	611.8 ± 4.46 [a]	468.3 ± 6.20 [a]
litter oiling	no oil	101.6 ± 0.83 [a]	240.0 ± 1.38 [a]	360.8 ± 2.32 [a]	473.6 ± 3.27 [a]	613.7 ± 4.53 [a]	461.3 ±6.29 [a]
	oil	102.2 ± 0.82 [a]	235.0 ± 1.36 [b]	326.0 ± 2.29 [b]	466.0 ± 3.23 [b]	614.3 ± 4.46 [a]	463.7 ± 6.21 [a]
interactions	no oil control temperature	99.4 ± 1.17 [a]	238.7 ± 1.94 [a]	359.9 ± 3.27 [a]	484.1 ± 4.61 [a]	604.7 ± 6.38 [a]	450.5 ± 8.87 [a]
	no oil temperature fluctuations	103.9 ± 1.17 [b]	241.3 ± 1.95 [a]	361.2 ± 3.29 [a]	463.2 ± 4.64 [b]	622.7 ± 6.42 [b]	472.0 ± 8.93 [b]
	oil control temperature	103.4 ± 1.17 [b]	238.3 ± 1.96 [a]	316.1 ± 3.29 [b]	461.4 ± 4.65 [b]	627.8 ± 6.43 [b]	462.9 ± 8.94 [ab]
	oil temperature fluctuations	101.0 ± 1.14 [ab]	231.7 ± 1.88 [b]	336.0 ± 3.17 [c]	470.7 ± 4.48 [b]	600.8 ± 6.19 [a]	464.6 ± 8.61 [ab]

[a-c] Means in the same column within a main effect or interaction with no common superscript differ significantly ($P < 0.05$). The effect of feed restriction was significant for all BW with the exception of the initial BW.

TABLE 4-3. Total feed consumption and conversion at 6 wk of age

treatments		wk 1	wk 2	wk 3	wk 4	wk 5	wk 6	feed consumption	total feed conversion
temperature	control temperature	17.6 [a]	48.9 [a]	87.0 [a]	131.3 [a]	171.1 [a]	160.4 [a]	616.3 [a]	2.03 [a]
	temperature fluctuations [1]	18.7 [a]	50.8 [a]	90.4 [a]	136.3 [a]	173.3 [a]	162.7 [a]	632.1 [a]	2.00 [a]
liter oiling	no oil	17.9 [a]	49.1 [a]	88.3 [a]	133.1 [a]	172.7 [a]	158.3 [a]	619.4 [a]	2.01 [a]
	oil [2]	18.4 [a]	50.6 [a]	89.1 [a]	134.5 [a]	171.8 [a]	164.8 [a]	629.0 [a]	2.03 [a]
	pooled SEM	2.42	2.42	2.42	2.42	2.42	2.42	6.94	0.015

[1] Two pens of birds, 600 male broilers, had temperature fluctuations (3^0C above the required temperature during the day and 3^0C below the required temperature at night) over two 6 wk periods.

[2] One control temperature and one temperature fluctuation pen received biweekly addition of oil to the litter (totaling 1.1 L/m^2 of oil over 6 wk) over two 6 wk periods.

[3] Sudden Death Syndrome

[4] Category includes peritonitis, omphilities, liver hemorrhage, dehydration, cull (runt), hepatitis, anemia, intestine rupture

There were no differences in percent mortality between treatments.

TABLE 4-4. Heart Characteristics including ascitic score, heart area (HA), right ventricle area (RVA), right ventricle weight (RVWT), trimmed heart weight (THWT), percent right ventricle area (PRVA), percent right ventricle weight (PRVW), and heart weight as a percentage of body weight (PHWT) of all birds after slaughter

treatments		score[1]	HA (cm^2)	RVA (cm^2)	RVWT (cm^2)	THWT (g)	PRVA (%)	PRVW (%)	PHWT (%)
Temperature	control temperature	1.32 [a] ± 0.03	3.46 [a] ± 0.02	0.59 [a] ± 0.01	2.34 [a] ± 0.03	9.22 [a] ± 0.07	0.17 [a] ± 0.003	0.25 [a] ± 0.003	0.00409 [a] ± 0.00003
	temperature fluctuation [2]	1.37 [a] ± 0.03	3.52 [a] ± 0.02	0.61 [a] ± 0.01	2.45 [b] ± 0.03	9.34 [a] ± 0.07	0.17 [a] ± 0.002	0.26 [b] ± 0.003	0.00413 [a] ± 0.00003
Litter oiling	no oil	1.33 [a] ± 0.03	3.46 [a] ± 0.02	0.59 [a] ± 0.01	2.37 [a] ± 0.03	9.20 [a] ± 0.07	0.17 [a] ± 0.003	0.26 [a] ± 0.003	0.00403 [a] ± 0.00003
	oil [3]	1.35 [a] ± 0.03	3.52 [a] ± 0.02	0.61 [a] ± 0.01	2.42 [a] ± 0.03	9.37 [a] ± 0.07	0.17 [a] ± 0.002	0.26 [a] ± 0.003	0.00419 [b] ± 0.00003
Interaction	*no oil* control temperature	1.33 [a]	3.44 [a]	0.59 [a]	2.33 [a]	9.18 [a]	0.168 [a]	0.253 [a]	0.0041 [ab]
	no oil temperature fluctuation	1.33 [a]	3.48 [ab]	0.60 [a]	2.41 [ab]	9.21 [ab]	0.166 [a]	0.266 [b]	0.0040 [a]
	oil control temperature	1.31 [a]	3.49 [ab]	0.59 [a]	2.36 [a]	9.26 [ab]	0.165 [a]	0.253 [ab]	0.0041 [b]
	oil temperature fluctuation	1.41 [a]	3.56 [b]	0.62 [a]	2.48 [b]	9.49 [b]	0.171 [a]	0.261 [ab]	0.0042 [c]
	pooled SEM	0.04	0.03	0.02	0.04	0.10	0.004	0.004	0.00004

[1] J. A. Hanson, 1996 (Alberta Agriculture, Food and Rural Development, 6909-116 Street, Edmonton, Alberta, Canada T6H 4P2, personal communication)

[2] Two pens of birds, 600 male broilers, had temperature fluctuations (3°C above the required temperature during the day and 3°C below the required temperature at night) over two 6 wk periods.

[3] One temperature fluctuation and one control temperature pen received biweekly addition of oil to the litter (totaling 1.1 L/m^2 of oil over 6 wk) over two 6 wk periods.

[a-c] Means in the same column within a main effect or interaction with no common superscript differ significantly ($P < 0.05$). The effect of oil was not significant. The effect of feed restriction was significant for all heart characteristics.

TABLE 4-5. Carcass characteristics of selected birds for analysis of fatpad and breast muscle weights with SEM

treatments		BW (g)	fatpad (g)	shank length (mm)	lung (g)	minor pectorals (g)	major pectorals (g)	percent breast [3] (%)
temperature	control temperature	2151 [a]	40.2 [a]	103.3 [a]	11.6 [a]	64.4 [a]	273.6 [a]	16.8 [a]
	temperature fluctuations [1]	2197 [a]	42.4 [a]	103.9 [a]	12.3 [a]	67.4 [b]	285.6 [b]	16.9 [a]
liter oiling	no oil	2186 [a]	42.1 [a]	104.3 [a]	13.1 [a]	66.4 [a]	284.1 [a]	16.8 [a]
	oil [2]	2162 [a]	40.5 [a]	102.9 [b]	10.8 [a]	66.5 [a]	275.1 [a]	16.8 [a]
	pooled SEM	23	1.2	0.5	1.1	0.9	4.2	0.12
interactions	no oil control temperature	2153 [a]	33.0 [a]	104.2 [ab]	12.4 [a]	64.7 [ab]	277.9 [ab]	16.8 [a]
	no oil temperature fluctuations	2219 [a]	44.3 [a]	104.4 [b]	13.8 [a]	68.0 [a]	290.3 [a]	16.9 [a]
	oil control temperature	2149 [a]	40.5 [a]	102.4 [a]	10.8 [a]	64.0 [b]	269.4 [b]	16.7 [a]
	oil temperature fluctuations	2175 [a]	40.6 [a]	103.5 [ab]	10.9 [a]	66.9 [ab]	280.9 [ab]	16.9 [a]
	pooled SEM	33	1.6	0.7	1.5	1.3	6.0	0.17

[1] Two pens of birds, 600 male broilers, had temperature fluctuations (3^0C above the required temperature during the day and 3^0C below the required temperature at night) over two 6 wk periods.

[2] One control temperature and one temperature fluctuation pen received biweekly addition of oil to the litter (totaling 1.1 L/m^2 of oil over 6 wk) over two 6 wk periods.

[3] The addition of the pectoralis major and the pectoralis minor divided by the total live weight at time of processing.

[a-c] Means in the same column within a main effect or interaction with no common superscript differ significantly ($P < 0.05$). The effect of oil was not significant. The effect of feed restriction was significant for all carcass characteristics.

TABLE 4-6. The total percentage of mortality from day 0 to 42 day of age and the percent of mortality due to ascites compared to leg problems, Sudden Death Syndrome (SDS), or other causes

treatments		total mortality (%)	mortality due to: ascites (%)	leg problems (%)	SDS [3] (%)	other [4] (%)
temperature	control temperature	10.9 [a]	4.6 [a x]	2.3 [a y]	2.9 [a xy]	2.3 [a y]
	temperature fluctuations [1]	12.3 [b]	6.3 [a x]	2.0 [a yz]	3.6 [a z]	1.1 [a y]
liter oiling	no oil	11.7 [a]	4.7 [a x]	1.8 [a y]	4.4 [ax]	2.1 [ay]
	oil [2]	11.6 [a]	6.2 [a x]	2.4 [a y]	2.1 [ay]	1.3 [ay]
	pooled SEM	0.31	0.78	0.18	0.55	0.39

[1] Two pens of birds, 600 male broilers, had temperature fluctuations (3^0C above the required temperature during the day and 3^0C below the required temperature at night) over two 6 wk periods.
[2] One control temperature and one temperature fluctuation pen received biweekly addition of oil to the litter (totaling 1.1 L/m^2 of oil over 6 wk) over two 6 wk periods.
[3] Sudden Death Syndrome
[4] Category includes peritonitis, omphilities, liver hemorrhage, dehydration, cull (runt), hepatitis, anemia, intestine rupture
There were no differences in percent mortality between treatments.
[a-b] Means in the same column within a main effect with no common superscript differ significantly ($P< 0.05$).
[x-y] Means in the same row within a main effect with no common superscript differ significantly ($P< 0.05$). The effect of oil was not significant. The effect of feed restriction was significant for all carcass characteristics.

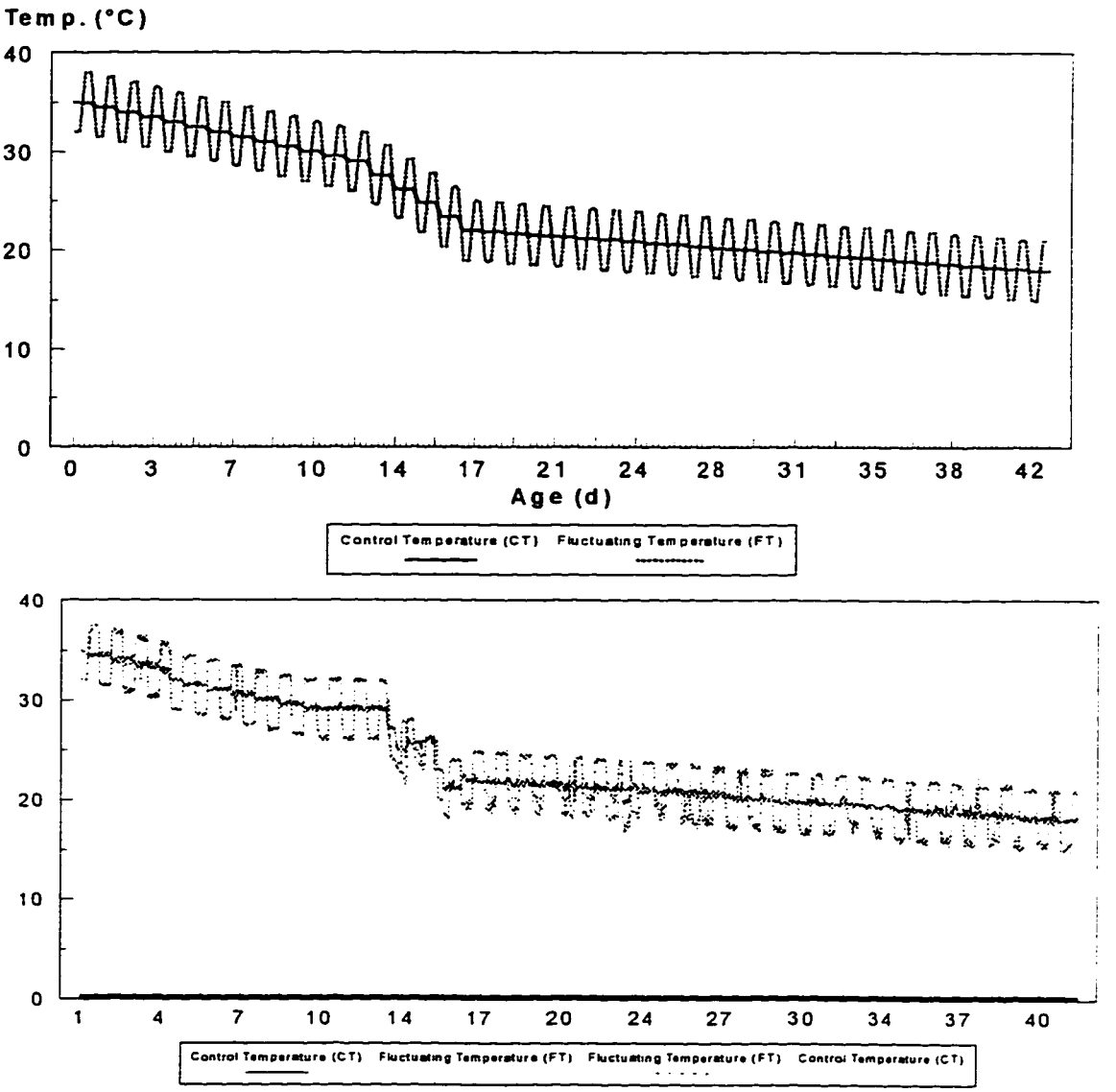

FIGURE 4-1. Upper figure: Target temperature recordings (^{0}C). Two pens in each of the two 6 wk periods had temperature fluctuations (TF) 3^{0}C above the required temperature during the day and 3^{0}C below the required temperature at night (a 6^{0}C change in temperature daily). The control temperature (CT) pens followed the required temperature. All pens had the same mean temperature. Lower figure: Actual temperature recordings (^{0}C) taken every 30 minutes in the first period in all four pens.

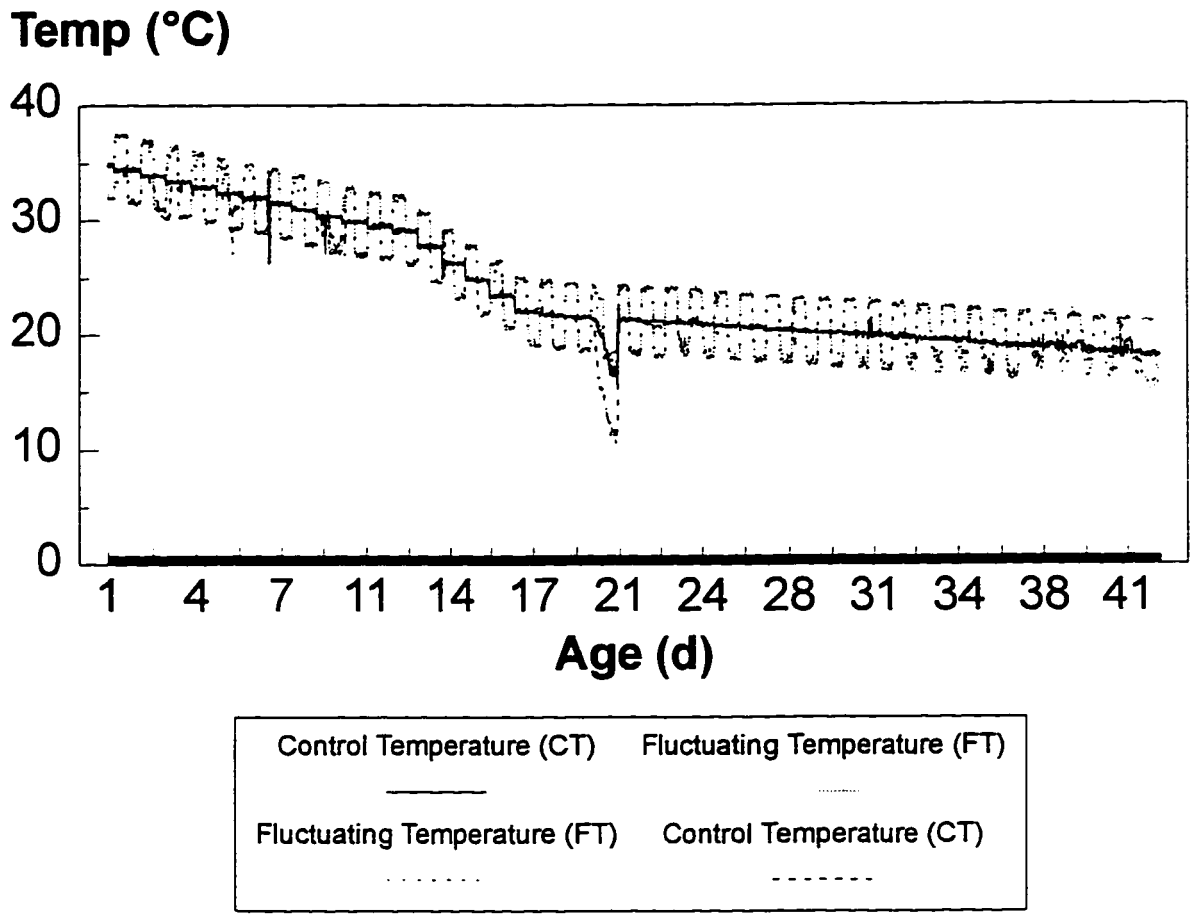

FIGURE 4-2. Actual temperature recordings (^{0}C) taken every 30 minutes in the second period in all four pens. Two pens in each of the two 6 wk periods had temperature fluctuations (TF) 3^{0}C above the required temperature during the day and 3^{0}C below the required temperature at night (a 6^{0}C change in temperature daily). The control temperature (CT) pens followed the required temperature. All pens had the same mean temperature.

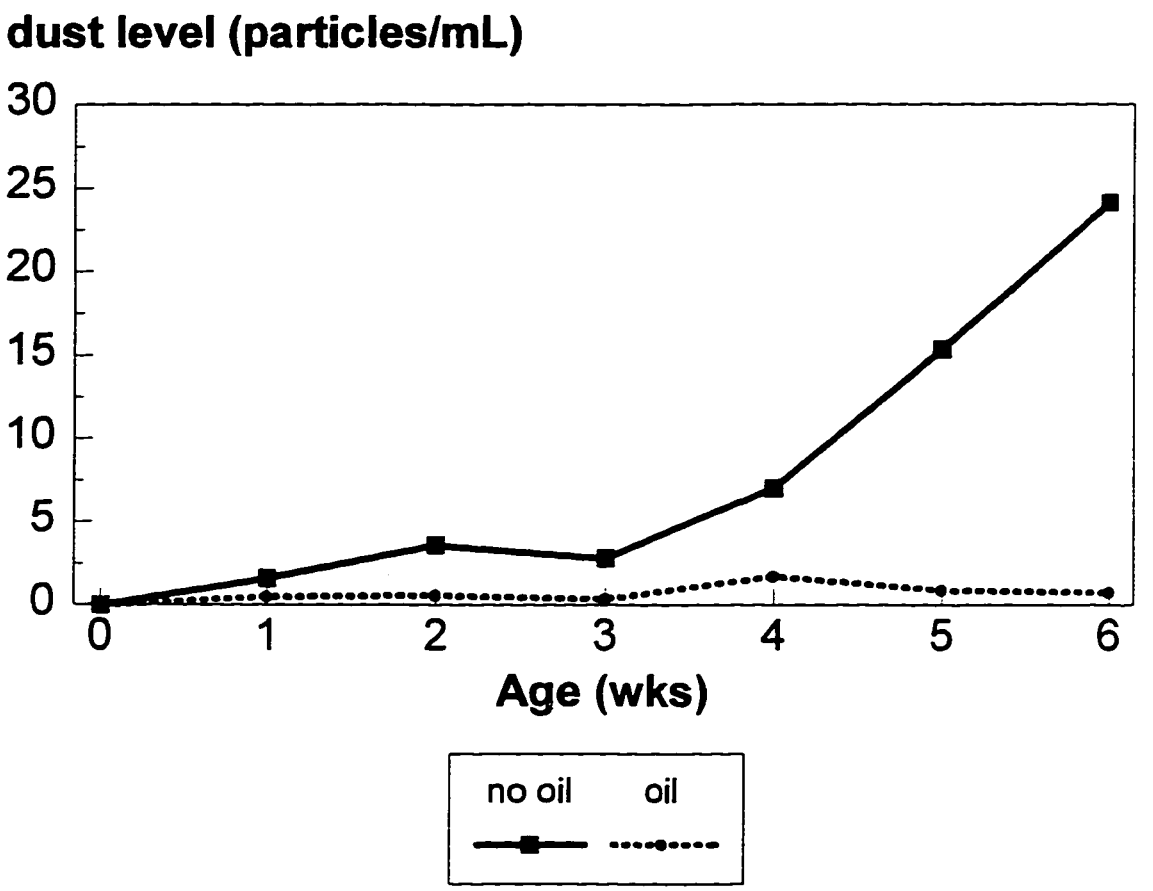

FIGURE 4-3. Dust levels in particles per mL in litter oil treated pens and non-treated pens. Dust levels were significantly less in the oiled pen from wk 1 to wk 6 ($P<0.05$).

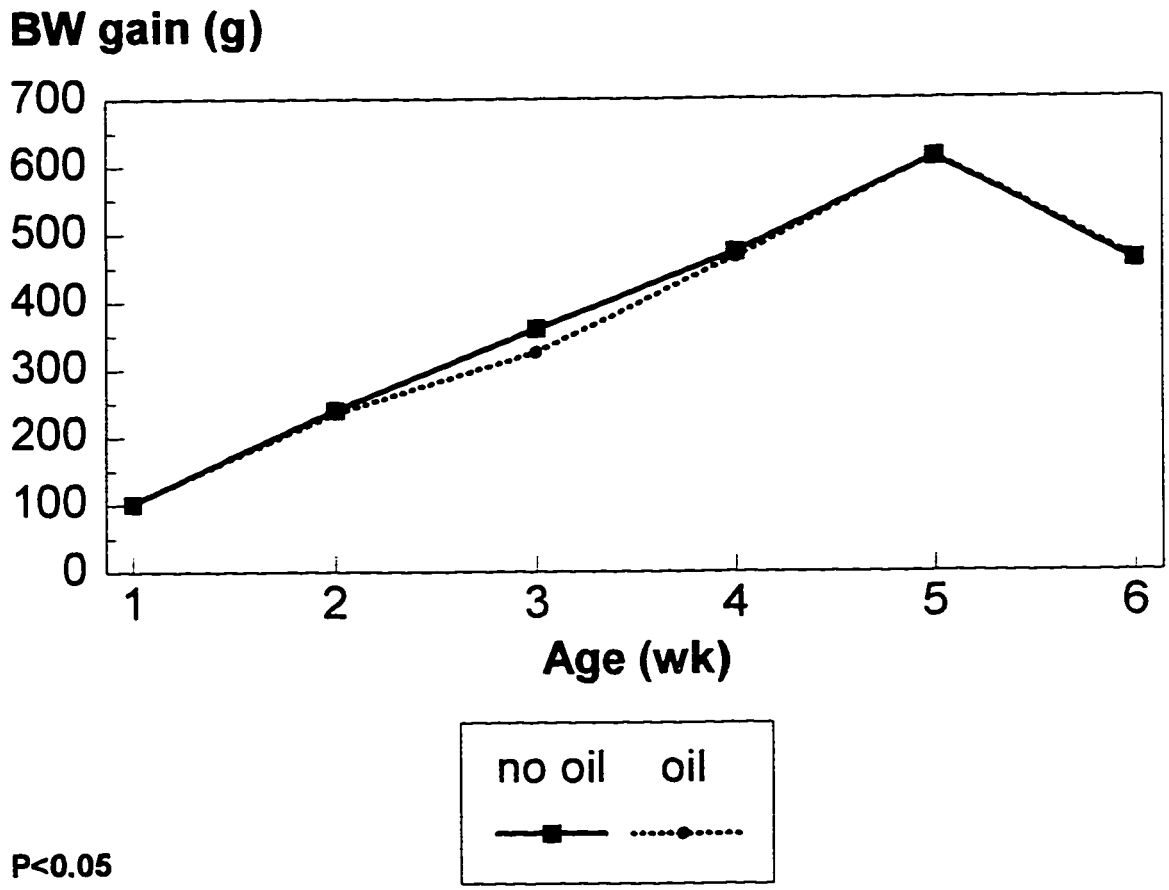

FIGURE 4-4. BW gain of birds in litter oiled treatment were lower at wk 3 and 4 than the non-litter oiled treatment.

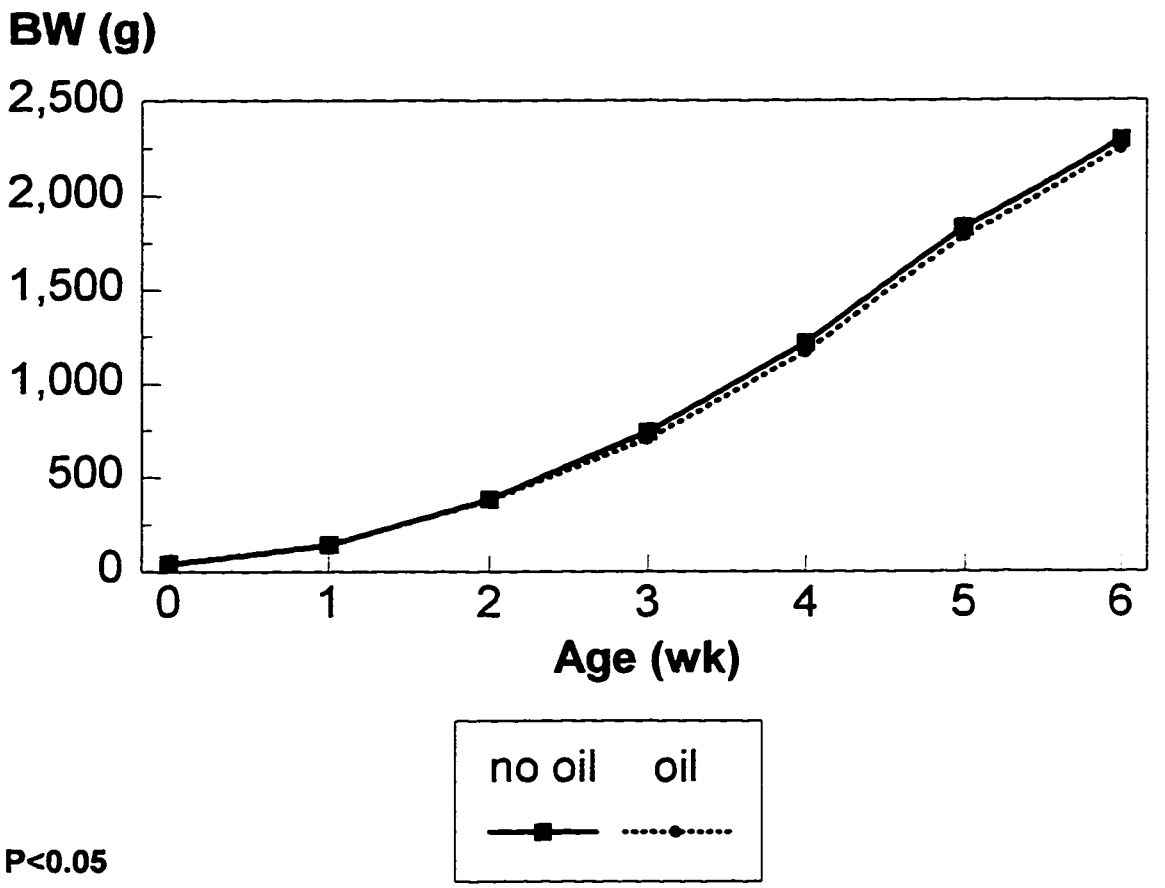

FIGURE 4-5. BW of birds in litter oiled treatment were lower at from wk 3 of age to processing at 6 wks than the non-litter oiled treatment.

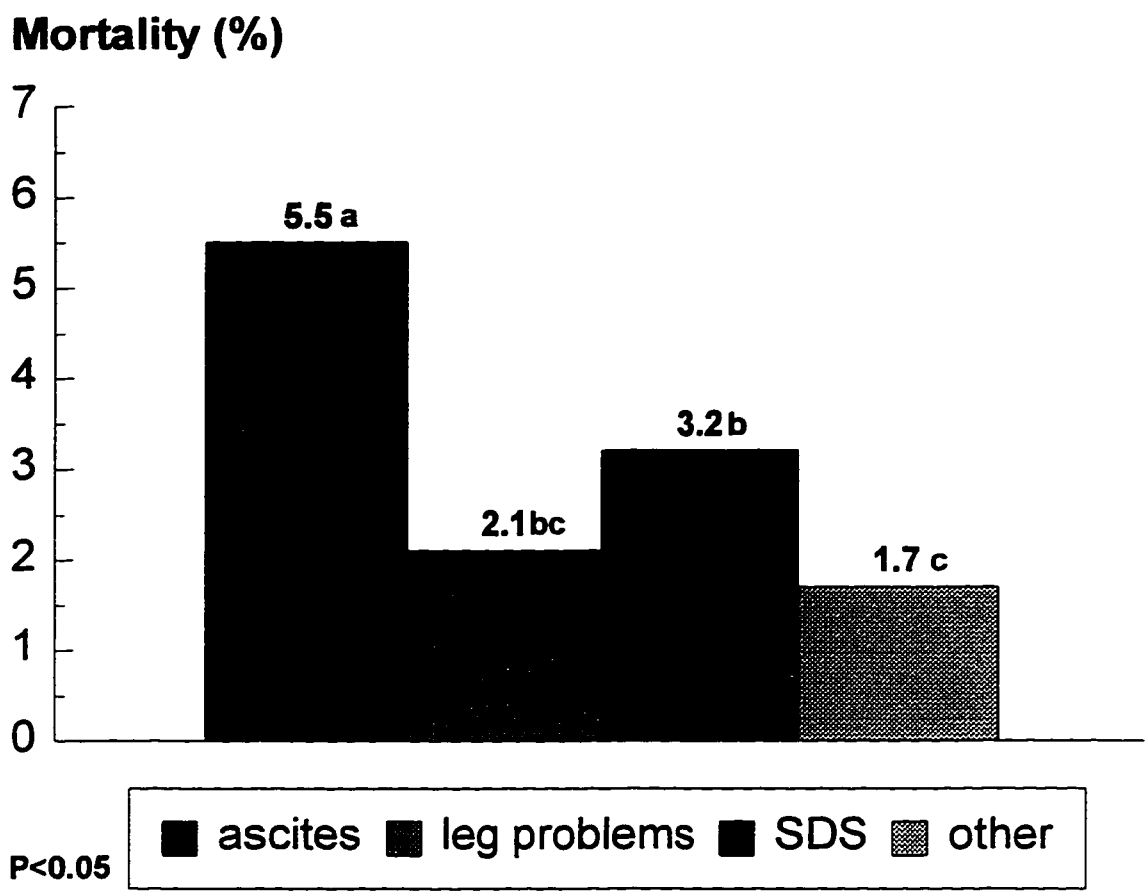

FIGURE 4-6. The majority of mortality was due to ascites.

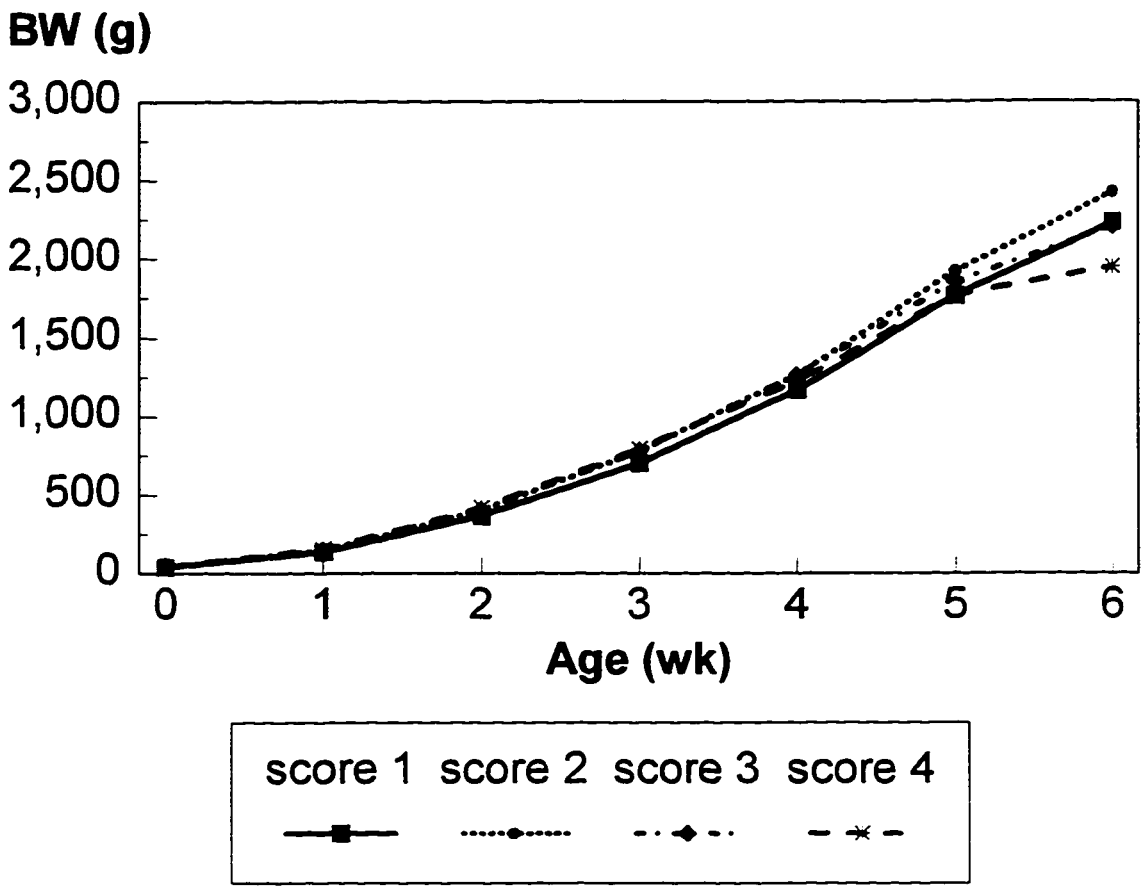

FIGURE 4-7. Growth curve of broilers with varying degrees of ascites.

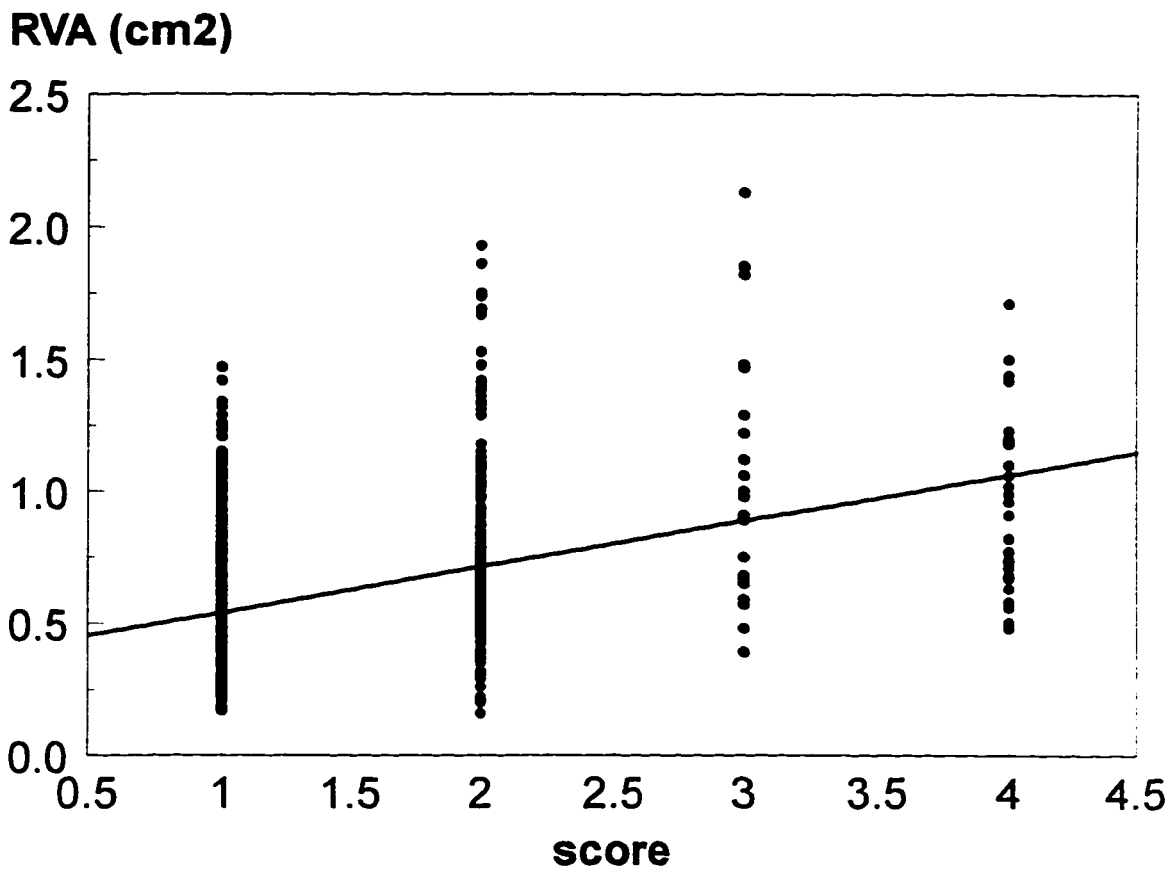

FIGURE 4-8. A scatter plot of the RVA against the ascitic score.

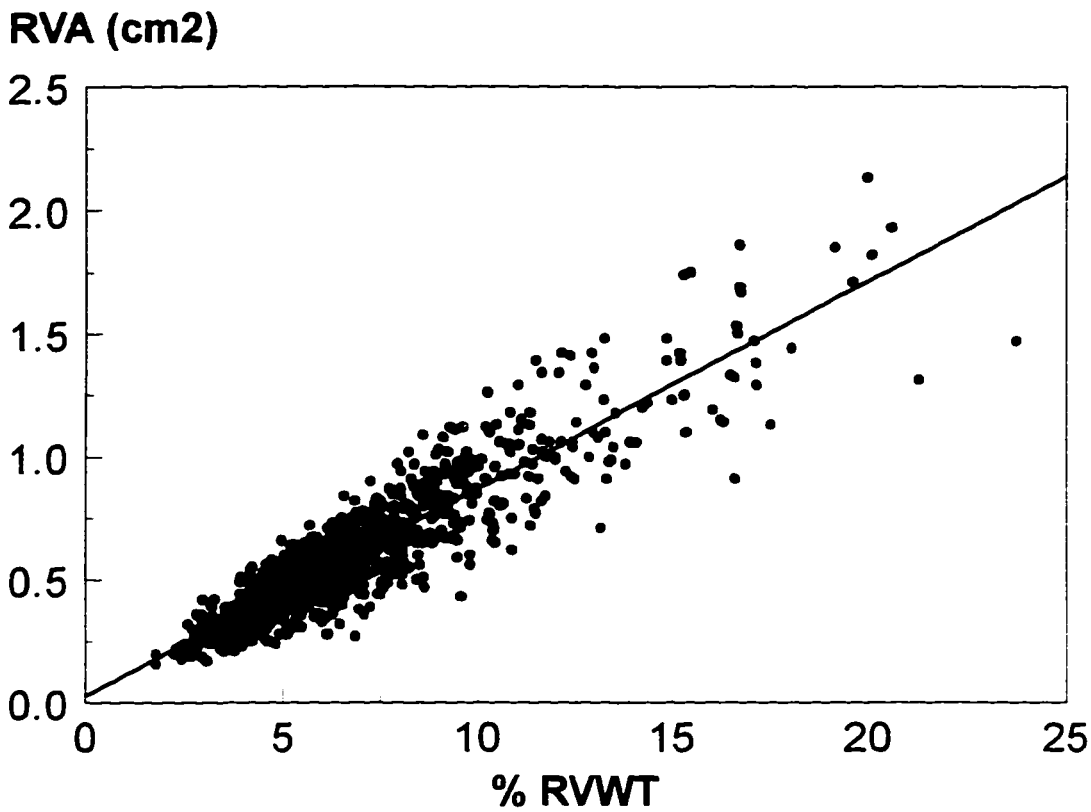

FIGURE 4-9. A scatter plot of the RVA against the RVWT (R^2=0.83).

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5. GENERAL DISCUSSION AND CONCLUSIONS

5.1 Introduction

Broiler chicken genetics have been pushed to the extreme for the purpose of increasing body weight. Broiler chickens reach market age in a shorter period of time with a higher breast muscle mass than ever before. Although genetic selection has successfully increased growth rates for faster production of broilers, genetic selection has also led to increases in mortality due to ascites. Rapid growth rates and genetic improvements have resulted in the average incidence of ascites increasing to 4.2% in 21 countries worldwide (Maxwell and Robertson, 1997).

There are two solutions to ascites. Firstly, the ascites syndrome can be minimized with good management techniques. For example, the most effective preventative methods that are favored by broiler companies include: different feeding strategies which reduce body weight gain, remove of excessive salt or sodium from the diet, and supplement diets and/or water with vitamin C; environmental control strategies which ensure the provision of adequate ventilation, aide in the prevention of any unnecessary respiratory challenges, and reduce ammonia contamination, and provide relief from cold stress. Management (to reduce growth rates primarily) should be recognized as a short-term solution to ascites (Leeson et al., 1995). Changes in management minimize losses without directly addressing the problem of ascites.

The second solution is genetic. Broiler breeders must be selected on the basis of heart growth. Broilerizing broiler breeders and selecting them based on their ability to keep heart growth in phase with body growth and the increase in muscle mass is of significant importance to the broiler industry. Ascites causes an average of 4.2% condemnations worldwide. Selection for non-ascites-susceptible broiler breeders that produce progeny that are also 'resistant' to ascites is a more cooperative solution to ascites than a continual struggle between the birds genetic predisposition and current broiler management.

The broiler breeder species is currently moving in the genetic direction of the White Broad Breasted Turkey. The continual drive for an increase in muscle mass is causing continued selection for growth characteristics with a relative lack of vision for reproductive characteristics. The genetic strategies of the poultry industry need to be drastically altered before artificial insemination is the only method of reproduction for broiler breeders and broilers are incapable of responding to any physiological challenge without causing increased mortality.

5.2 General Discussion

The objectives of these experiments were to assess:

1. the management technique of feed restriction and litter oiling to reduce ascites,
2. the image analysis technique as a method of assessing the degree of ascites and to ensure that the technique was consistent and repeatable, and
3. the common phenomenon of diurnal temperature fluctuations and its effect on broilers and the incidence of ascites, as well as, to assess the management technique of litter oiling for a higher stocking density in an effort to reduce the incidence of ascites.

5.2.1 Feed restriction

Feed restriction is currently the most effective means of reducing mortality due to ascites in broiler flocks (Shlosberg et al., 1991). The feed restriction treatment used in the first experiment produced lower mortality rates, as well as, lowering the average ascitic score. Reduced growth rate prevents increased blood flow leading to right ventricular enlargement and ascites in broilers.

Current broiler markets are very competitive. The advantage gained by using feed restriction to reduce mortality does not out weigh the reduction in final bird weights in this case. Even short term feed restricted broilers had a significantly lower market weight, *pectoralis major*

weight, and *pectoralis minor* weight than *ad libitum* fed broilers. A lower percentage of abdominal fat at market age was desirable. Early feed restriction contributes to carcass yield and quality. Extending the cycle length to allow the broilers to compensate for feed restriction regimens causes an increase in production costs, making severe feed restriction regimes undesirable.

The feed restriction levels used in the first experiment were severe. They would not be used in commercial broiler production. The severe short term feed restriction treatment used for this experiment was intended as a method of establishing the use of the image analysis technique by using a comparison of a group of healthy broilers to a group of broilers with a higher risk of exhibiting ascitic symptoms (*ad libitum* fed treatment). The feed restriction treatment was used as a reference point for image analysis of the right ventricle. The reference point was needed for determining any changes that may occur to the *ad libitum* fed treatment when raised on oil treated litter. Milder feed restriction regimens allow for a quicker recovery of body weight and reduced mortality due to the ascites syndrome, as well as, reduced leg problems. The most effective treatment reported for the control of ascites was a skip-a-day feed restriction program used in Kenya. The skip-a-day program was applied during the starter period (no feed on days 7, 9, 11, and 13) in association with a low protein starter capon mash. This program has been used successfully for the past two years and consequently has dramatically reduced the annual incidence at altitude (Nairobi - 5 600m) from >30% to 5% (Maxwell and Robertson, 1997).

The degree of feed restriction used to control growth rates and prevent ascites is very variable. A compromise between final body weights and reduced mortality and ascites can be circumvented using diet formulation and a short term feed restriction. Breeding for increased muscle mass has not led to a concomitant increase in the size of the heart and respiratory system, and so the modern broiler has increasing difficulty maintaining oxygen demand for maintenance and growth (Shlosberg *et al.*, 1991). Although feed restriction has been successful in controlling the incidence of mortality due to ascites, it is not an adequate solution to the problem.

5.2.2 Temperature Fluctuations

Factors such as cold, which increase oxygen requirement, also increase cardiac output and blood flow and may result in increased pulmonary arterial pressure and cause both a volume and a pressure overload in on the right ventricle (Wideman and Kirby, 1995a). Chicks have a very narrow thermoneutral zone and any temperature below an optimum increases the oxygen requirement (Julian et al., 1989). The 6°C temperature fluctuation treatment caused stress to the heart. These data suggest that the birds subjected to the control temperature treatment were less physiologically stressed than the birds subjected to the fluctuating temperature treatment because they had a lower average RVWT. The birds subjected to the control temperature treatment also had a decreased PHWT. Fluctuating temperatures did not increase the RVA possibly because the mean temperatures were the same for both temperature treatments. The temperature fluctuation treatment had a higher rate of mortality. The *pectoralis minor* and *pectoralis major* muscle weights were significantly heavier in the temperature fluctuation treatment than the control temperature treatment. This was not due to the increase in feed intake because the feed consumption of the birds in the temperature fluctuation treatment and the control temperature treatment were not different. The differences in *pectoralis minor* and *pectoralis major* muscle weight could be due to the effect of diurnal temperature changes on feeding habits causing changes in protein metabolism and muscle deposition. Further research is required to identify the role of temperature fluctuations on protein metabolism and muscle deposition.

Cyclic environmental temperatures may cause broilers to change consumption patterns and consume more feed or less water during the part of the day with the lowest temperature. Increasing temperatures (9 °C) stimulate water consumption which in turn may cause broilers to continue consuming feed (May and Lott, 1992). Diurnal temperature changes (6 °C) may be taken as a signal for broilers to commence feed consumption.

Broilers respond to specific events, such as a workers passing through the barn or the sound of the auger, with renewed interest in feed consumption. Temperature fluctuations could

be a stimulus causing a renewed interest in feed consumption. Although the birds subjected to the temperature fluctuation treatment had a greater *pectoralis major* and *pectoralis minor* than the control temperature birds, there was no difference in final body weight between the treatments. Carcass composition is dependent on environmental temperature (Leenstra and Cahaner, 1991). Scheele *et al.* (1987) reported that a significantly higher fraction of the energy was retained as protein at lower temperatures than at moderate temperatures. Low temperatures act as stimulators in protein accretion, however, at higher energy costs (Leenstra and Cahaner, 1991). More experiments are required to assess the increase in performance due to temperature stimulus of protein partitioning in broilers.

The increase in oxygen requirement caused by the additional heat loss may have a marked affect on the incidence of ascites in meat-type chickens. This could be a significant factor at high altitude where less oxygen is available for respiration and where broilers may be housed in unheated, open-sided buildings even though the temperature drops markedly at night (Julian et al., 1989).

The importance of careful management control of day-old chicks, not only following placement but even before their arrival on the farm must also be emphasized (Maxwell and Robertson, 1997). Broilers are susceptible to ascites. Temperature fluctuations may not directly induce ascites, but fluctuating temperatures cause an increase in mortality and are an added stress that would not be recommended to producers even though it may lead to an increase in muscle mass.

5.2.3 Litter Oiling

The application of canola oil to the straw litter did not affect the amount of mortality. Pens that received the litter oiling treatment had a significantly higher mortality when there were temperature fluctuations than when the pen received a constant temperature due to the heat loss caused by the coating of oil on the broiler. When the straw litter was not oiled there was no difference in mortality between the temperature treatment.

Body weight gain was negatively affected by litter oiling. In both experiments, the broilers in the pens that received the application of canola oil to the litter had lower body weights. In experiment one (Feed Restriction and Litter Oiling) at 3 wk of age, the broilers receiving the application of canola oil to the litter had a significantly lower weight gain than the birds that did not receive litter oiling treatment. This trend continued into wk 4. At processing, however, there were no significant differences in body weight. However, in experiment two (Temperature Fluctuations and Litter Oiling), there were differences between the treatments from 2 wk of age and the final body weight of the broilers receiving litter oiling treatment was significantly lower to the broilers in the pens that were not oiled.

In the first experiment (Feed Restriction and Litter Oiling), the stocking density was 8 broilers/m^2 and the canola oil was applied at a rate of 0.8 L/m^2 (0.1 L/broiler over the 6 wk period). Typical industrial stocking densities are much greater than 8 broilers/m^2 therefore for at the conclusion of the first experiment, it was hypothesized that an increase in stocking density may show an decrease in the average ascites score and a decrease in mortality when applying canola oil to the litter. The increased stocking density produced a need for an increase in the rate of application of canola oil to the litter to 1.1 L/m^2 over the 6 wk period (0.09 L/broiler over the 6 wk period). This level of litter oiling reduced the respirable dust in the pens to a maximum of 2.5 particles/mL. The second experiment did not show reduction in the ascites score, but a decrease in body weight gain resulted from the increase in the amount of canola oil used to significantly reduce the respirable dust and stocking density. The reduction in respirable dust was improved in the second experiment with the increase in rate of canola oil application. Although the application rate was decreased from 0.1 L/broiler to 0.09 L/broiler, the overall increase in the rate of application of canola oil to 1.1 L/m^2 was excessive and consequently detrimental to the growth rate of the broilers. To prevent increased heat loss leading to lower body weight gains the maximum rate of canola oil application should not exceed 0.8 L/m^2. It may also be more beneficial to the producer to refrain from using canola oil on the litter until the broiler reaches 3 wks of age. Further studies on the time of initiation of litter oiling and duration are required.

For a comparison the litter oiling treatment with the non-litter oiling treatment the results of both experiments were amalgamated. The birds in the *ad libitum* fed treatment from the first experiment and the birds in the control temperature treatment from the second experiment were combined into an new data set and reanalyzed. When the litter oiling treatments were compared using this data set, there was no significant difference between the mean 6 wk body weight of broilers subjected to either the litter oiled or non-oiled treatment.

Litter oiling did not show a decrease in the RVA of the broilers in the second experiment, as it did in the first experiment. However, when the experiments were reanalyzed using the combine data set which included the control pens in both experiments (Feed Restriction and Litter Oiling and Temperature Fluctuations and Litter Oiling) that received the oiling treatments, there was a significant decrease in RVA overall (0.48 compared to 0.52 cm^2). The percentage RVA was also significantly decreased by the application of canola oil to the litter.

The average stocking density used by the majority of countries is 14-15 birds/m^2 with a slight reduction in warmer countries (Maxwell and Robertson, 1997). In the United Kingdom's broiler ascites survey, the incidence of ascites was significantly greater ($P<0.001$) when birds were stocked 15-18/m^2 compared with 18-24/m^2. This increase may be associated with the better growth rate as a result of the reduced stocking density (Maxwell and Robertson, 1997). This effect was not evident in these experiments most likely because the experimental stocking densities used did not approach the bottom range of industrial stocking density.

The broilers subjected to the litter oiling treatment may have had a higher heat loss due to a decrease in thermal resistance because of a coating of canola oil on the feathers. Although the canola oil is applied directly to the litter, the broilers attain a low level of coverage due to lying on the litter. Using the required temperatures for optimal broiler growth can result in poor feathering and cause an increase in the convective heat loss of broilers.

Litter oiling also produced differences in the type of mortality seen. The no oil treatment had similar percent mortality due ascites and sudden death syndrome (SDS) (which affects the left ventricle of the heart), however, when the canola oil was applied to the litter there was a reduction in mortality due to SDS (from 4.4 % no oil treatment to 2.1 % oil treatment $P >0.06$).

Within the litter oiling treatment, there was a significantly greater amount of mortality due to ascites than leg problems or SDS or other causes of mortality which could include peritonitis, omphilites, liver hemorrhage, dehydration, cull (runt), hepatitis, anemia, or intestine rupture. Within the non oiled treatment, there was more mortality due to ascites and SDS than leg problems and other.

Although litter oiling was unable to reduce the mortality due to the ascites syndrome, litter oiling improved the RVA and the air quality in the pens and reduced dust accumulation on the drinkers. Litter oiling also has a potentially positive affect on workers in broiler chicken barns. Litter oiling in small amounts (0.8 L of oil/m^2 of floor area over 6 wks) would be beneficial in reducing respirable dust.

5.2.4 Image Analysis

As with all new techniques, standardization is the first step to producing consistency. The placement (shape and orientation) of the heart cross section was standardized to provide an accurate assessment of the severity of ascites. The image analysis technology used to capture heart images was effective in producing RVA that correlated to the ascitic score proportional to the RVWT correlation to the ascitic score, though the correlation's did differ slightly between trials. The RVA and the RVWT were also highly correlated. The RVA seemed more sensitive than the RVWT in the first trial because differences in the RVA were apparent between treatment interactions which were not exhibited by the RVWT.

The pulmonary arterial clamp model is useful for inducing ascites (Wideman and Kirby, 1995b). Elevated RV:TV ratios indicate that pulmonary arterial hypertension did develop in broilers with a clamped pulmonary artery. Due to the survival rate, the postsurgical survival rate was 55% in their first experiment and 66% in their second experiment, and the cost performing the surgery, the continued application of the pulmonary arterial clamp model seems limited (Wideman and Kirby, 1995b).

Image analysis has more possibilities for applications in ascites research than simply the analysis of the RVA. The lung nodules or nodules on the atrio-ventricular valve could also be quantified using the image analysis technology. Nodules on the atrio-ventricular valve could be a factor in the blood flow back into the lungs due to an inadequate valvular seal. The incidence of Sudden Death Syndrome can also be characterized by the LVA. Image analysis exceeds the right ventricle/total ventricle weight ratio technique not only because the long term storage of RVA files is possible, but also because the analysis of these files can be reviewed, reassessed, and new data such as wall thickness can be quantified. Although image analysis is labor intensive, image analysis techniques will continue to be used in various applications such as ascites research projects, other broiler research projects, and will likely expand into other areas of poultry science.

5.3 Conclusions

The following conclusions were drawn from this study:

1. Feed restriction is a viable and effective method of reducing the incidence of ascites. The level of feed restriction is dependent upon the flock size, final required market weights, and the strain of broiler used.
2. Temperature fluctuations do not cause an increase in mortality due to ascites, however, they cause an increase in the overall mortality and RVWT. Temperature fluctuations cause an increase in stress on the broilers, as seen in the increase in RVWT, and should therefore be avoided until possible the advantages of these temperature fluctuations on *pectoralis major* and *pectoralis minor* muscle weight can be more thoroughly determined.
3. Litter oiling is beneficial in reducing respirable dust and the ascites syndrome as shown by the RVA, even though it does not yet result in a decrease in mortality or condemnations due to ascites. As the broiler becomes increasingly selected for muscle growth, litter oiling will gain importance in reducing the RVA until the breeder companies begin selection for heart growth which can sustain the current rapid muscle growth.

4. Image analysis technology is an important tool for studying ascites. Once standardized, the technique of placement of the heart slice is repeatable and provides consistent results. Image analysis has benefits in establishing a long term data base of RVA, as well as, providing short term results on the incidence of ascites. The RVA has shown differences in treatments that were not indicated with RVWT. Because the RVA can provide long term discriminating evidence on heart growth and right ventricular development, it could be an effective tool for selection of strains and more in-depth studies into management solutions for the reduction of the incidence of ascites.

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