BODY WEIGHTS AND SELECTED ORGAN MEASUREMENTS FROM PIGS STILLBORN OR DYING NEONATALLY

by

CHARLES EDWARD HERREN

B. S., Kansas State University, 1954

D. V. M., Kansas State University, 1954

A MASTER'S THESIS

submitted in partial fulfillment of the

requirements for the degree

MASTER OF SCIENCE

Department of Surgery and Medicine

KANSAS STATE UNIVERSITY
Manhattan, Kansas

1977

Approved by:

Major Professor

TABLE OF CONTENTS

INTRODUCTION

A seven to ten percent neonatal mortality and a five percent rate of stillbirth continue to be a problem in the swine industry. Little detailed information pertaining to stillborn pigs and those dying in the immediate neonatal period has been recorded.

Stillbirth is defined as those pigs which are delivered at term dead but not mummified. Immediate neonatal mortality concerns those pigs born alive but which die within 48 hours.

Stillbirths can be divided in two major categories: epidemic and the category in which one or two pigs are born dead in approximately one third of the litters.

A high proportion of the stillbirths occurring over a short time span constitute the epidemic type. Generally epidemic stillbirths result from an infectious cause, occur with abortions and are likely to result in a positive diagnosis. Infectious causes of the epidemic type of Fungi, Leptospirosis, SMEDI (Entero viruses) or the viruses of Hog Cholera and Aujeszky's disease. Deficiencies in the gestation ration of certain nutrients (Iron, Vitamin A and Iodine) have been incriminated in the epidemic type.

The most common and difficult to explain type of stillbirth is the loss of a small number of pigs in part of the litters. This category overlaps neonatal mortality and exhibits several unique characteristics. The rate increases in large litters and with a sow's increased parity. From the first to the fourth litter the increase in stillbirths is

correlated with a concurrent increase in litter size, however, after a sow's fifth litter the sharp increase is unrelated to the litter size. The extended farrowing interval in old, large sows may explain this increase.

Differing from those in the epidemic death category, these stillbirths probably die of asphyxia. Their lungs are full of meconium and some show evidence of post partum circulatory activity. Asphyxiation due to premature umbilical rupture leads to depression, weakness, chilling and crushing.

Most stillbirths occur in the later stages of delivery of litters born during a normal time interval (under 4 hours). A prolonged farrowing interval may increase stillbirths or conversely an increased number of dead pigs may slow the birth rate.

This study was initiated to obtain information concerning organ weights and gross pathology occurring in stillborn pigs or in those dying in the immediate neonatal period.

LITERATURE REVIEW

Edwards[20] (1972) reviewed the causes of prenatal and perinatal mortality in pigs and concluded that a hard core of everyday losses remained even after subtracting losses from specific diseases. This core consisted of the 5% stillbirths and the 7-10% neonates dying within the first week from physical or developmental inadequacies. Stillbirth and weakness at birth reflect impaired intrauterine fetal development and/or pathology associated with parturition.

Bille, et al, [8] (1974) surveyed 14,390 pigs and reported that 13.2% died during the period which extended from the last few days of gestation to the third postpartum day. This period accounted for 58.2% of the total preweaning losses. Perinatal mortality rate increased from 11.8% with a parturition time of less than 6 hours to 21.3% where the parturition time exceeded 6 hours.

The marked influence of litter number and litter size on perinatal mortality found by Bille, et al,[8] (1974) confirmed previous survey and field studies on the incidence of baby pig survival.[1,2]

Since increased litter size at birth and birth weight seem to be associated with lower viability of undersized pigs, this may partly explain the high mortality rate in large litters.[65,70] Other factors to be considered are the lack of milk and more pigs than teats.

England, et al,[21] (1976) divided pigs into weight groups of less than 455 gm. and proceeding by 227 gm. increments through the range of greater than 1590 gm. and found the percent survival by weight groups at

72 hours was 0, 56, 92, 99, and 100 respectively. Mortality occurred at younger ages for pigs of lower birth weight and continued to be more prevalent to 35 days of age as well as in the first 72 hours. Little effect of farrowing intervals were noted on survival of pigs in litters where farrowing was of long duration but otherwise normal.

Baker, et al,[3] (1958) found fetal weights associated positively albeit insignificantly with both number of fetuses in the litter and number in the uterine horn at 25 days gestation. By 20 days a marked change had occurred and the regression of the number in the horn was significant and differed (p .01) from the regression of litter size. There was a trend between 25 and 70 days wherein the effect of number of fetuses in the uterine horn was noted on fetal weight, and this effect was not related to placental membrane weight.

Bille, et al,[8] (1974) found the perinatal mortality rate to be 20% greater in winter than summer and noted that the second quarter of the year produced the lowest rate despite the fact that during this period the percent of larger litters was the highest. This seasonal variation for perinatal mortality follows a pattern similar to that reported by Nielsen, et al,[60] (1974).

Bille, et al,[8] (1974) found herd size and protein level in the gestation ration to have no significant effect on perinatal mortality. Similar results were obtained from mode of housing -- whether crates or pens. Attended farrowing showed a 2.8% lesser rate of perinatal mortality than ones receiving no attention.

In Bille's, et al,[8] (1974) study 58% of all the litters contained affected pigs and 54.7% of these pigs were male, while the distribution

of normal liveborn males is 52%. Earlier work by Nielsen, et al,[60] (1974) Bereskin, et al,[7] (1973) demonstrated that while male pigs weighed an average of 30 grams more at birth than females, the female survival rate was 5 to 9% higher. Survival rate of both sexes rose with higher birth weight.

The stillborn antepartum group was differentiated by the reddish brown discoloration of the liver, lung and spleen due to hemolysis, autolysis and partial resportion. A larger portion of the stillborn antepartum group were born preterminally than the stillborn intrapartum and early neonatal death groups, and this fact was not related to the litter size, seasonal variation or sexual influence. Bille, et al,[8] (1974) recorded a prolonged farrowing time for litters with antepartum dead pigs and concluded that this confirms the experience that a dead pig is more troublesome for a sow to deliver than a live one.

Dunne[15] (1975) showed that the incidence of stillborn antepartum pigs may increase significantly in a herd with clinical or subclinical infections. Previously reported high incidences[59,60] of stillborn antepartum pigs were thought to be caused by viral infections in nonimmune dams.

Although births occur with relative ease in the sow, stillbirths are nevertheless associated with prolonged farrowing, umbilical cord damage and birth during the last third of parturition.[20,21,68] This suggests hypoxia as a major hazard of birth in swine as in other species.[10]

Randall[67] (1971) showed that asphyxia is an important factor in the etiology of intrapartum stillbirths and may influence the viability of live born pigs for several hours. He also noted asphyxia associated with

losses due to crushing by the dam. Strict confinement had an adverse effect and attended farrowing had beneficial effects.

Randall[68] (1972) demonstrated premature rupture of the umbilical cord in 93.6% of stillborn pigs. The frequency of the premature rupture of the cord was higher at later stages of parturition and pigs born with a ruptured cord showed marked acidosis and hypercapnia.

Jackson[37] (1975) found the overall stillbirth rate of 19.0% in 195 cases of dystocia was at least three times the figure for normal births. Stillbirth was noted in 63.1% of the dystocia litters. Where dystocia occurs the stillbirth rate has been clearly shown to be minimally influenced by a duration of labor under 5 hours but will increase to nearly 50% where labor extends beyond 20 hours. When labor is prolonged placental separation with fetal anoxia can give rise to a high stillbirth rate. High stillbirth rates (55.2%) were reported in association with primary uterine inertia and in toxemic sows with primary uterine inertia (88.2%). The stillbirth rate in cases of secondary utiner inertia was 31.9%.

Dawkins[10] (1966) suggested that intrapartum anoxia interferes with lung expansion and gas exchange by causing intrauterine gasping and inhalation of amniotic fluid. Anoxia resulted in constricted pulmonary vasculature with reduced pulmonary blood flow. Both preceding factors, in infants, leads to a loss of pulmonary surface active phospholipid and complicates lung expansion.

Randall[67] (1971) noted some of the newborn pigs had a detectable heart beat but inspiration was never observed, probably because anoxia depressed the respiratory centers.

Milosavljevic, et al,[53] (1971) found that 30% of the pigs with a detectable heart beat but not breathing could be revived with artificial respiration.

The interval between birth of pigs in a litter affects the prevalence of stillbirth. Even though the interval is greater for stillborn pigs, dead fetuses might be the cause rather than the result of delayed parturition. Approximately 80% of intrapartum deaths occur in the last third of the litter.[68]

Sprecher, et al,[72] (1974) concluded that perhaps intrapartum death removes part of the fetal endocrine components that control parturition. He felt that this could delay the emergence of pigs in the ovarian end of the horn causing even more fetal death from anoxia. When dichlorvos was fed to sows during the last 21 to 30 days of gestation the birth interval decreased from 16 minutes in control sows to 11 minuted in treated sows.[72] The rate of stillbirths were reduced from 6% to 3% and there was a dose related increase in the mean birth rate of treated litters.

Leman & Sprecher[41] (1976) in a later study found carbacholine was inconsistant in reducing stillbirths and its side effects made the drug unsuitable for routine usage. Neostigmine was variable in its effects on stillbirth rate and birth interval. Dichlorvos reduced the stillbirth rate but not the perinatal death rate, and had no effect on weaned weight.

The third major category demonstrated by Bille, et al,[8] (1974) involved the undersize live pigs dying within 3 days and showed that 50.7% weighed less than 800 grams. The postmortem findings in this group resembled starvation, the carcass was emaciated and dehydrated with mahogany brown

masculature. The stomach and intestines were distended with air and scratches were often present from fighting with litter mates.

Pomeroy[65] (1960) observed that early deaths occur most frequently in pigs of subnormal size that resembled animals on a sub-maintenance diet.

Several authors have studied the problem of undersized live pigs. Perry and Rowell[62] (1969) showed the position of the fetus in the horn had no effect on fetal weight when there were less than 5 fetuses, but as the number increased those in the ends of the horn have an increased advantage over those in the middle. This can be explained by differences in vascular architecture.

Widdowson[82] (1971) observed that growth retardation in the uterus and delivery of undersized pigs or runts is interpreted as delivery of immature, not fully developed pigs. The organs and muscles of "runt" pigs were smaller and contained less protein and less DNA, the brain, heart, spleen and stomach were larger, but the brains were less highly developed chemically than their larger litter mates. Analyses of liver weights were inconclusive.

Goodwin[28] (1955) and Hakkarainen[31] (1972) showed that the newborn easily develops hypoglycemia as a result of low body fat and underdeveloped gluconeogenesis. Other investigators[56,58] have desmonstrated a correlation between the weight of pigs and their ability to adapt to environment. Undersized pigs are especially sensitive to chilling due to greater surface area in proportion to body weight. Wallach, et al,[80] (1947) proved the evaporation of amoniotic fluid was a critical factor and the pig must expend a

considerable amount of heat to dry. Leech[39] (1976) concluded the main hazards of a new born pig are not disease, but its mother or its litter mates with whom it has to compete for milk.

Disturbances in the maternal environment, including psychosocial stress, alter the development and performance of offspring in other species.[5,29,42] Stress susceptibility (Porcine Stress Syndrome) seems to be a heritable trait in some growing swine.[77]

Howe, et al,[34] (1969) found the large lipid masses in the zona reticularis of pig adrenals (known to be stress susceptible) were induced by fluctuating environmental temperatures or by low environmental humidity.

Judge, et al,[38] (1965) suggested that pigs with pale, soft exudative musculature may have some degree of adrenocortical insufficiency.

Some sows may be highly reactive to stress through the activation of the sympathoadrenal system[84] which may affect their reproductive performance. The resulting reduction in uterine blood flow would have more effect on the fetuses which are located in the middle of the uterine horn.

Stave[75] (1970) feels that survival of the neonate is a reflection of maturation, adaptation and tolerance. Maturation being morphological and functional changes, genetically determined, which occur prenatally and perinatally. Adaption allows the neonate to adjust to the demands of harsh extra uterine environment. Tolerance is the ability of the fetal or newborn animals physiological system to tolerate stresses which in the adult would exhaust energey reserves.

Stanton, et al,[75] (1973) used the rate of decrease of rectal temperature of 5^{o}C to measure viability of newborn piglets and examined the inter

relation of litter size, birth weight, farrowing intervals, birth order, umbilical cord glucose and blood lactate. The loss of temperature was more rapid for the smallest piglets, however, first born lost heat faster than last born, when the size was held constant. Piglets with high blood lactate levels and low hemoglobin levels lost temperature more rapidly than larger litter mates.

Stanton & Carroll[74] (1974) postulated that maturation and adaptation of the fetus could be significantly modified by its own endocrine systems which are activated by maternal releasing hormones liberated during stress. This has not been demonstrated in swine.

Many infectious agents have been linked with perinatal death in swine. Ferguson & Powers[24] (1956) incriminated Leptospira pomona in abortion of sows two to three weeks prior to term. Weak pigs born at term but dying shortly thereafter was common. In one survey twenty-five sows aborted or farrowed 257 of which 161 were stillborn and 17 were weak pigs that died following parturition.

Sows infected at mid-pregnancy or later with Leptospira canicola did not abort but farrowed weak pigs.[49,50,51] These sows showed anorexia, dullness and a copious vaginal discharge within 3 to 6 days after inoculation.

Viruses more commonly cause embryonic and fetal death than abortion. Hog cholera virus infections at 90 days gestation or later caused the greatest number of stillborn pigs.[16] Infection within one week of term did not affect viability but the virus was isolated from the newborn pigs by fluorescent antibody culture.

Hog cholera vaccination of susceptible sows with an attenuated vaccine

killed half of the pigs in utero, and the majority of the pigs born alive died within 8 days.[35]

SMEDI viruses A, B, C and D (picorna viruses) isolated from stillborn pigs or fetuses are capable of causing death of embryos with absorption, death of fetuses with mummification or stillbirth, and death of the newborn within a few days after birth. Transmission appears to be other than venereal. Immunity appears to protect against recurrence.[35]

Dunne, et al,[18,19] (1967-1971) affirmed increasing evidence that other viruses including parvoviruses and reovirus are also associated with the SMEDI syndrome. SMEDI (entero) viruses were isolated from stillborn pigs, 3 day dead pigs and fetuses found dead in utero following hysterectomy at mid-pregnancy.[17] Infections were associated with small litter sizes, lower survival rates, and immunity of sows following infection. Most pigs dying in the perinatal period had no discernable lesions attributable to virus infection. Dunne postulated that death of the newborn was of bacterial causes because of lower resistance from the in utero virus infection.

McAdaragh and Robl[45] (1976) produced stillbirths, mummified fetuses and small weak pigs, delivered at term, by inoculating sows between 40 and 85 days of gestation with porcine reovirus.

Mengeling and Cutlip[48] (1976) diagnosed porcine parvovirus (PPV) infection in cases of reproductive failure in swine using immunofluorescent microscopy. These were naturally occurring cases of stillbirths and dead fetuses delivered at term.

large vaccination programs have been used in Japan with the aim of controlling Japanese encephalitis epidemics in man by reducing the number

of non-immune swine. Hsu, et al,[32] (1972) found the total incidence of stillbirth in the vaccinated group (1/74) was significantly lower than that in the unvaccinated controls (21/68).

Avitaminosis A is a common non-infectious cause of multiple stillbirths in swine.[61] Edema in many tissues and ascites was seen in pigs from Vitamin A deficient sows by Goodwin and Jennings[27] (1958). Pigs are usually stillborn at term or are weak and die shortly thereafter.

Moore[54] (1965) found some sow herds with stillbirth problems to have as much as a 50% reduction in hemoglobin. These stillbirths were reduced following addition of 100 to 200 ppm of ferrous sulfate to the gestating ration with a subsequent return to normal hemoglobin levels.

A genetic cause of stillbirths was alluded to in several articles, but no evidence could be found to prove this has any effect other than early embryonic death. McFeely[46] (1967) found 10% of pig blastocysts with aberrant chromosomal complements. Lubs and Ruddle[43] (1970) found between 0.5% and 1.0% incidence of chromosomal abnormalities in newborn babies. Similar surveys have not been done on animals, but a few chromosomal abnormalities have been uncovered in some malformed or stillborn young. This approach will not detect chromosomally aberrant but phenotypically normal young animals.[23]

Organ measurements and body weights were taken on 254 normal newborn porcine fetuses at various known stages of gestation and on 35 normal newborn pigs.[79] The data for males and females were combined except for gonad weight. Skeletal measurements generally exhibited a rectilinear relationship with age. Soft tissue growth rate increased with age and was curvilinear,

with the exception of the lungs and spleen which changed rate and direction of growth between 93 days and birth. The following table is reproduced in part from the Ullrey, et al,[79] (1965) publication.

MEASUREMENTS (MEANS AND S.E.) OF FETUSES AND NEWBORN PIGS FROM GILTS

Measurement	Birth No.	Birth Mean	Birth S.E.
Weight of:			
Body, gm.	35	1040.9	42.7
Brain, gm.	10	35.0	0.65
Liver, gm.	10	24.9	2.9
Spleen, mg.	10	781.0	66.0
Length of:			
Femoral diaphysis, mm.	10	38.8	1.4

Morrill[55] (1952) postulated that the liver loses considerably more weight than the average for body tissues of newborn pigs during fasting. A small part of this is due to exhaustion of the hepatic glycogen store but most of the difference must be dehydration.

Itoh, et al,[36] (1965) found that of 90 newborn pigs, the body weight averaged 1 kg. and the fresh liver weight averaged 29 gm. The fetal to dam ration for liver glycogen averaged 1:2.

In their study of protein deprivation of the dam on wet weight of newborn piglet livers Frape, et al,[26] (1967) found the following:

Feed -- Gestation	Pig Wt.	Liver Wt.
Low protein, low food intake	939	34.5
High protein, high food intake	992	41.0
Low protein, high food intake	935	33.0
High protein, low food intake	985	39.8

Despite a low ratio of liver to feed Vitamin A the dams dietary protein and food intake both influenced the quantity of liver Vitamin A in the unborn when energy intake is minimal. Increased dietary protein is a more potent factor than increased food intake in raising Vitamin A storage.

Frape, et al,[25] (1969) in later research demonstrated that liver size and its N content are very sensitive to variations in dietary protein and energy in the growing adult rat. However, it cannot be assumed that the fetal liver will respond similarly to these dietary changes, although the size and N content of the fetal liver may reflect the concentration of the maternal plasma protein. Plasma protein synthesis is influenced by liver size in adult rats and, indeed the addition of carbohydrates to a low protein diet can reduce the plasma albumin concentration in young pigs through its effect on the liver.[33,44]

Bengtsson[6] (1974) proved that a very effective barrier existed between the sow and the fetus and suggested that fetal and neonatal serum proteins are of fetal origin and not transferred from the mother. An increase of Beta globulin and albumin after the first few hours of nursing has to be due to colostrum intake. The total protein in newborn pre-colostral piglets was less than one-half that in sows, and the level of albumin, Beta and Gamma globulin were higher in the sow. Alpha globulin was lower in the sow than in the piglets.

Platt, et al,[63] (1967) noted that the most impressive post mortem finding in the protein calorie deficient pig was the reduction in size or virtual absence of the thymus.

Wenham, et al,[81] (1969) showed the within litter differences in bone measurements were closely associated with fetal weight differences. The

relationshps of litter mean log. length of bones upon age were best described by quadratic regression while the mean log. fetal length of bones upon age was best fitted by linear function regression.

Schain, et al,[71] (1967) in studies with ^{14}C labeled phenylalanine found that some factor present in the uterine environment promotes incorporation of amino acid into brain protein. Protein synthesis in tissues may be maintained at a higher rate by placental delivery of amino acids and energy supplying substrates. Newborn animals may be unable to duplicate entirely this placental function. Maternal hormones may play a role. Nucleic acid levels may not be maintained following birth thereby affecting tissue protein metabolism. In humans a premature decrease in brain protein synthesis during a critical period of growth of the CNS may be responsible for cerebral syndromes manifesting themselves at a later time.

Done[14] (1968) demonstrated the absolute and relative growth curves for the cerebellum and whole brain of the pig fetus from 45 days to term to be related to conceptual age and newly calculated body length curve. He also devised the equations relating conceptual age to crown rump length, and cerebellum to brain ratio from age or crown rump length. Naeye[57] (1965) found that the mean weight of the liver, spleen, thymus and adrenals, of small-for-dates infants, were less than half those of normal infants, while the mean brain weights were 82% of the normals. Platt and Stewart[64] (1968) have seen that bitches raised from weaning age on a protein restricted diet gave birth to pups of small weight with neurological disorders.

In a review article Milner[52] (1973) observed that placental pathology in the human causes intrauterine under-nutrition resulting in impaired transport of nutrients to the fetus. The thymus, liver, spleen and adrenal glands were

much smaller than controls in cases of maternal under-nutrition. This reduction in organ size is mainly due to smaller cell size not to reduced cell number.

MATERIALS AND METHODS

All the full term stillborn baby pigs and those dying within 48 hours post partum were taken continuously from each of two farrowing units until 200 were received. Both farms handled the gestating sows in essentially the same manner.

Herd 1 was a commercial herd which used domestic crossbred sows hand mated to Duroc and Yorkshire boars. Gestation confinement was in dry lot on a 14% commercial ration. Farrowing was accomplished in crates, on steel slatted floors in a centralized farrowing house. Manure was handled by allowing an overflow pit to drain into two lagoons. Minimal attention was given, because of labor shortage, to the sows during parturition.

Herd 2 was a university herd and was composed of purebred, highly inbred Duroc swine, which were eating a trial gestating ration containing 97% dehydrated alfalfa meal and 3% mineral salt combination. Farrowing was also accomplished in crates, with cement slatted floors, to which a carpet pad was added during parturition. A great deal of attention was given to the farrowing sow with the exception of night time attendance.

The dead piglets were refrigerated to $4^{\circ}C$ on the farm and transported within 48 hours to the necropsy laboratory.

Each pig was weighed and catalogued according to source farm, date of death and sex. Necropsy was conducted in a prescribed procedure. All weighing was done wet and to the nearest .1 gm on a (Dial-O-Gram) dial and counter weight balance. The thymus, spleen, liver and brain were weighed immediately following excision.

A wide band of tissue along the ventral midline including the cervical and ventral thoracic skin, the sternum and the ventral abdominal wall was removed, exposing the pericardial space, pleural cavity and abdominal viscera. A sample of fluid was aspirated from the pericardial sac into a sterile, numbered Monoject syringe and refrigerated at 40° for future examination.

The thoracic inlet was then opened by retracting the first pair of ribs and both the cervical and cardiac portions of the thymus was dissected, removed and weighed.

The degree of aeration of the lung was determined by removing a portion of the diaphragmatic lobe to see if it would sink or float in a beaker containing fresh water.

The heart was examined internally and externally for texture, size, presence of hemorrhage, and congenital defects but was not weighed.

The liver was removed by dissecting away the mesenteric attachments, and weighed with the gall bladder and cystic duct in place. Presence of sanguinous engorgement, fatty infiltration plus the shape of the borders of the liver was noted.

The spleen was dissected away from the stomach, closely trimmed at the hilus, and weighed.

The stomach was opened along the greater curvature and the presence or bsence of food was noted.

The small and large intestine was examined as a unit for the presence of enteritis. The presence of hemorrhage or swelling in the kidney was noted prior to removal of these organs.

The left femur was disarticulated through the coxofemoral and stifle

joint, trimmed of muscle, numbered and frozen for storage. Upon accumulation of 50 femurs they were radiographed on a 14" x 17" film using a 1000 m., 3 phase Profexray. The settings were 200 ma., at .006 sec., with 56 Kvp., and an aluminum stepwedge was placed on the plate, to be used as a comparison for bone density. The aluminum stepwedge was also used as a calibration, to correct for parallax, when measuring the femurs. The length of calcified diaphyses or main centers of ossification (measured parallel to the axis) was determined for each femur, and reduced by 2% to correct for parallax.

The cranial cap was removed with shears, the brain stem severed just caudal to the cerebellum. The brain was removed using a small plastic spoon, the cranial vault was carefully scraped, and the brain tissue weighed.

The day following necropsy the pericardial fluid was examined under a Leitz microscope, using a darkfield condenser, with wet mounts at 400X, for the presence of spirochaetes. All positives were confirmed by a qualified microbiologist.

Ten normal live born pigs (6 male and 4 female) were purchased from the commercial herd, euthanatized and subjected to the same necropsy procedures as the stillborn specimens. These pigs were selected at 36 hours of age from 5 litters. Euthanasia was accomplished with 10 mgm Sucostrin (succinylcholine Squibb) injected intramuscularly and subsequent exposure to carbon monoxide. The pigs were stored over night at $4^{o}C$ to fix tissue and blood in the organs.

The data was programmed into the university computer where means, standard errors and least squares difference of the body and organ measurements were obtained for several classes and subclasses. Parameters for class divisions were: 1. stillborn pigs with ateletic lungs; 2. normal active pigs euthanatized

before 48 hours of age; 3. neonatal death (NND) as evidenced by aerated lungs and/or food in the stomach. The source farm and sex were also entered into the program.

Means, standard errors and least squares difference of body and organ weights were also recovered for all possible subclass combinations. Linear regressions were performed on the subclasses showing a high probability of difference.

RESULTS

Of the 200 stillborn and neonatal death pigs surveyed 132 (66%) were received from the commercial source (Herd 1) and 68 (34%) from the university herd (Herd 2). The male to female ratio for the entire group was 108 (54%) males and 92 (46%) females. Aerated lungs were found in 67 (33.5%) and these specimens were placed in the neonatal death category in this report, while atelectic lungs were found in the remaining 122 (66.5%) and consequently were classified as stillborn. A majority of the stillborn pigs were surrounded by the fetal membranes.

Of the normal controls received from Herd 1, six (60%) were males and four (40%) were females. Only five of the stillborn pigs showed evidence of prepartum mummification, and these were all from Herd 1.

Milk curd was found in the stomach of 37 of the 67 neonatal death pigs of which 24 (65%) were from Herd 1 and 13 (35%) were from Herd 2. These specimens all had aerated lungs and were classified neonatal death.

Seventeen of the pigs showed confirmed spirochetes in the pericardial fluid and of those, six were from Herd 1 and eleven were from Herd 2. Of the eleven from Herd 2, three were marked strongly positive by the microbiologist. Serologic examination for Leptospira antibodies were performed on ten sows from Herd 2 that had delivered stillborn pigs. These ten sows showed no antibody titer to any of five species of Leptospira.

No gross cardiac pathology was noted either in any of the pigs. No sanguinous engorgement or fatty infiltration of the livers was noted. Enteritis was noted in two pigs, both of which had a milk curd in their stomachs.

An unequal subclass analysis of variance using the body and organ weight data was programmed into the university computer. The measurement means and their standard errors for these classes and subclasses are presented in Table I.

The mean body weights were higher for normals (1434.08 g.) when compared to the stillborn neonatal death mean (1094.40 g.). The mean body weights for the two source farms differed with Herd 2 having a 44.88 g. higher mean. Females in the stillborn neonatal death category were 41.38 g. heavier than males in the same category regardless of source farm. Stillborn pigs weighed 31.94 g. heavier than the liveborn pigs dying within 48 hours (NND).

Between stillborn and neonatal death groups on each source farm the mean body weights maintained the relationship of the total of these two categories. In both herds the females with aerated lungs (NND) weighed 150 g. more than males in the same class, while stillborn males weighed 66 g. more than stillborn females.

The mean body weight of each sex maintained the same relationship in both herds and in the combined herd figures, however, the males from each herd were more nearly alike in weight than females. Stillborn males weighed 140 g. more than males dying within 48 hours (NND) while females dying neonatally outweighed their stillborn counterparts by 75 g.

When mean body weights were compared in all eight possible combinations certain significant differences are apparent. Liveborn (NND) female Herd 2 pigs dying within 48 hours outweighed males in the same category by 236 g. Similarly the mean body weight of stillborn Herd 2 males was 100 g. larger than stillborn Herd 2 females. Neonatal death Herd 1 females weighed 140 g.

less than their Herd 2 female counterparts. When the mean body weight of stillborn Herd 1 males and females are compared to stillborn Herd 2 males and females very little significance was noted.

The least significance difference test was run at the .05 level on organ measurements and body weights. Body weights of the normal subclass were more nearly equal to the stillborn (LSD 205.56) and the neonatal death (NND) (LSD 213.69) subclasses than either the stillborns or the neonatal death (NND) subclasses were to each other (LSD 111.19). Thymus and spleen weights and femoral lengths were more similar when compared to body weights in all three subclasses. However the mean log. of the thymus weight in the normal subclass was smaller (.70) than the mean log. thymus weight in the neonatal death (NND) subclass (1.09). With the least significant difference test the liver weight showed more similarity in the stillborn and neonatal death (NND) subclass (LSD 3.79) than the normals did when compared to either the stillborn (LSD 7.02) or the neonatal death (NND) (LSD 7.29) subclass. Brain weights also were found to be more nearly equal in the stillborn (LSD 3.02) or the neonatal death (NND) LSD 3.17) subclasses were equal to the brain weights of the normals. Both brain and liver weights were found to depend not on sex or source farm as much as on the subclassification.

When regression lines were plotted on the organ weights compared as a function of body weights, the slope was found to be not linear. All organ weight regressions followed the formula: Organ weight (g) = $K \times BW^{x}$. When K is the constant, BW is the body weight in grams and x is the expotential function but not 1. To demonstrate the wide dispersion of data and the low the low confidence interval, scatterplots were printed on the weights of brain, liver, and these lines are presented in Tables 5 and 6. Radiographs of femurs

for the stillborn neonatal death subclass are shown in Figures 1, 2, 3 and 4 and the normal pigs are shown in Figure 5.

DISCUSSION

The main thrust of the study was to obtain data from pigs lost due to the nonepidemic type of stillbirth and neonatal death. Efforts were made to select pigs from a source farm (Herd 1) on which controlled nutrition, sanitation and immunization practices helped minimize the influence of deficiency and infectious causes of stillbirth and neonatal death. Specimens were received from the commercial herd in order to obtain data from a commercial standard. Normal pigs were also taken from this herd to satisfy the same criteria. Pigs were also selected from Herd 2 to explore possible differences caused by the experimental management practices taking place in this unit; e.g., the high content of alfalfa meal in the gestating ration plus increased inbreeding coefficient.

Clinical observation, plus the absence of mummification, on both farms suggested that neither was infected with any of the SMEDI viruses at the time of sampling. Three samples which were strongly positive for spirochetes aroused suspicion that Leptospirosis might be influencing the rate of stillbirth and neonatal deaths in Herd 2. Blood was collected for serologic examinations. While sows were negative, suggesting that Leptospirosis was not a factor, it would have been useful to conduct a second serologic examination 30 days later.

Organ weights of pigs from the 2 herds were similar when plotted as a function of body weights. That the goal to receive specimens from the nonepidemic type of stillbirths was achieved, is suggested by the lack of evidence of gross pathology or necropsy.

The male to female ratio among stillbirth and neonatal death pigs were

.very similar to that found by other workers with males exceeding in numbers whereas the females were larger in size.

The results of analysis demonstrated a smaller mean thymus weight in pigs of the normal subclass when compared to pigs in the neonatal death (NND) subclass, although both of these classes were approximately the same age. This difference was more marked when the body weights of the two groups were compared. Dunne, et al,[15] (1975) states that the thymus continues to increase in size for 4 to 6 weeks postpartum.

Liver weights may have been decreased in the normals because of increased glycogen utilization following birth of these active pigs.[10] The decrease when compared to body weight was too small to be significant. Liver weights within the stillborn neonatal death class, while not demonstrating a linear regression, were generally correlated with body size. The low energy gestation ration in Herd 2 makes it difficult to account for the larger livers in these pigs. Possibly the large increase of Vitamin A or of protein in the alfalfa meal contributes to this finding.

Of the entire study the most promising finding was the 6 gm. decrease in the mean brain weight when compared to the mean body weight in the stillborn group versus normals and those dying neonatally. The comparison of brain weights of stillborn with the full term normals reported in Ullrey, et al,[79] was even more suggestive (10 gm. decrease). Anoxia has been shown by several workers to be the immediate cause of stillbirth; however, the findings of this study did permit the assumption that the etiology of the anoxia might be the decreased brain size.

Intrapartum anoxia activates the sympathoadrenal system of the fetus, liberating catecholamines which are deleterious to the neonate. When

catecholamines were experimentally infused blood pH feel and plasma CO_2 increased indicating acidosis due to mobilization of glycogen reserves in the heart, skeletal muscle and liver. Reduction of carbohydrate reserves reduces the newborn pigs ability to withstand prolonged stress, since the newborn pig has no adipose tissue and shivers vigorously when exposed to cold.[10] Intrapartum anoxia was postulated to lead to CNS depression and even brain damage. This may in turn injure the thermoregulatory centers causing chilling.

Stillborns may be the immediate result and neonatal deaths the later result of anoxia but smaller brains may predispose some pigs to membership in both categories. Parasympathomimetic drugs which decrease the birth interval, lowers the stillbirth rate, but apparently keeps the pigs alive only to die in the neonatal period.[41] Altered brain development rather than body size may be the cause for placing pigs in the stillborn or neonatal death classes.

Fetal nutritional disturbances during gestation may lead to a reversible decrease in cell size or an irreversible decrease in cell number. Milner[52] observed that investigators have found human placental growth occurred by increased cell numbers until 36 weeks but increased cell size was responsible for any subsequent placental growth. The hemodynamic theory of uterine vascular architecture proposed for rats if modified for swine cannot be ruled out. This theory proposed that the uterine blood supply forms an arterial vascular arcade, arising from the aorta, and traverses the uterine horn giving off branches. This artery then joins the ovarian artery near its aortic origin. Blood perfusion pressure would be the greatest at the ovarian and cervical ends of the horn but would decrease toward the middle of the

horn. Alterations to blood flow would be most evident to the fetuses in the middle of the horn. The work of Perry and Rowell[62] showing larger pigs at each end of the uterine horn may substantiate this.

Two periods of cell multiplication were observed in human fetal brains, one at 15-20 weeks gestation and another beginning at 25 weeks and ending postnatally. It was further demonstrated that neurones multiplied before glia and that the early cell division was neuroblastic multiplication and the later period consisted of glial proliferation.[52] Milner[52] (1973) and others have found that before 30 weeks in the human embryo there was some increase in the protein/DNA ratio in the kidney, heart and muscle but none in the brain. Arbitrarily organ/DNA is a measure of cell number and protein/DNA ratio is a measure of cell size.

Cellular growth patterns are important because impaired growth during hyperplastic periods is irreversible while catch up growth can replace losses during periods of hypertrophy. Milner[52] (1973) feels there are vulnerable periods in the developing brain. In the pig this period extends from 6 weeks before birth to 5 weeks after, and embraces an early phase of rapidly increasing cellularity, and a later one of rapid deposition of lipid. Placental pathology causing intrauterine undernutrition can result in impaired transport of nutrients to the fetus. If this pathology coincides with the rapid growth interval of the fetal brain changes may take place that are irreversible. The complex pattern of changing relationship throughout the period of growth help to explain the impact of undernutrition on the chemical structure.

SUMMARY

The results of this study may be summarized as follows:

1. The normal sexual grouping of pigs of 52% males and 48% females was found to be altered to 54% males and 46% females in the stillborn, neonatal death (NND) group. The males in this group were 41.38 g. lighter than females. Stillborn pigs were 32 g. heavier than live-born pigs dying within 48 hours (NND).
2. Stillborn males weighed more than stillborn females and females dying neonatally (NND) weighed more than males in the same class.
3. Herd differences in body weights and organ measurements were insignificant.
4. The mean thymus weights in the normal subclass were smaller than the mean thymus weights in the stillborn neonatal death subclass.
5. Least significant differences of the mean body, liver and brain weights for the 3 important subclasses are as follows:

	LEAST SIGNIFICANT DIFFERENCES		
Comparison	Body Weight	Brain Weight	Liver Weight
Neonatal Death to Stillborn	111.19	1.70	3.79
Neonatal Death to Normals	213.68	3.17	7.29
Stillborn to Normals	205.56	3.02	7.02

6. Regression lines of organ weights as a function of body weights were not linear and demonstrated poor confidence limits because of the extreme dispersion of the data. A formula was devised to fit this function.
7. When plotted, liver weights compared to body weights for the stillborn pigs were insignificantly greater than the liver weights of the neonatal death (NND) and normal groups.

8. Brain weights of the stillborn group, when plotted with the body weights were significantly smaller than brain weights of either the normals or the live born pigs dying within 48 hours.

CONCLUSIONS

Results of the study suggest that while intrapartum anoxia may be the immediate cause of nonepidemic stillbirth and neonatal death an underlying cause of diminished brain size may predispose at least stillbirth. We might hypothesize that the decreased brain size found in the stillborn pigs may be due to reduced uterine vaculature causing fetal and placental undernutrition to certain occupants of the uterine horn.

This avenue of investigation needs to be explored further.

ACKNOWLEDGEMENTS

The author wishes to express his sincere appreciation to Dr. J. E. Mosier and Dr. J. Richard Olson of the Department of Surgery and Medicine for the consultation and advice throughout the course of study and preparation of this thesis.

Appreciation is also given to Dr. S. M. Kruckenberg of the Department of Pathology, Mr. David Culp of the Statistical Laboratory and Ms. Barbara Kelly of the Diagnostic Laboratory for their advice and help, freely given, in collecting and analyzing the data in this study.

Further the author wishes to commend his wife and daughter for their patience and help conferred during the course of preparation of this paper.

BIBLIOGRAPHY

1. Anonymous: A Survey of the Incident and Causes of Mortality in Pigs. I. Sow Survey. Vet. Rec. 71 (1959) 777-786.

2. Backstrom, L.: Environment and Animal Health in Piglet Production, A Field Study of Incident and Correlation. Thesis Acta. Vet. Scand. Suppl. 41 (1973) 1-140.

3. Baker, L. M., Chapman, A. B., Grummer, R. H., and Casida, L. E.: Some Factors Affecting Litter Size and Fetal Weight in Purebred and Reciprocal Cross Matings of Chester White and Poland China Swine. J. Anim. Sci. 17 (1958) 612-621.

4. Bell, C.: Dual Vasoconstrictor and Vasodilator Innervation of the Uterine Arterial Supply in the Guinea Pig. Cir. Res. 23 (1968) 279-289.

5. Bell, C.: Autonomic Nervous Control of Reproduction. Pharmacol. Rev. 24 (1972) 657-736.

6. Bengtsson, S. G.: Identification and Characterization of Soluble Blood Serum Proteins in Newborn Piglets. J. Anim. Sci. 38 (1974) 95-99.

7. Bereskin, B., Shelby, C. E., and Cox, D. F.: Some Factors Affecting Pig Survival. J. Anim. Sci. 36 (1973) 821-827.

8. Bille, N., Nielsen, N. C., Larsen, J. L., and Svendsen, J.: Preweaning Mortality in Pigs. 2. The Perinatal Period. Nord. Vet. Med. 26 (1974) 294-313.

9. Davison, A. N., Dobbing, J.: Myelination as a Vulnerable Period in Brain Development. Brit. Med. Bull. 22 (1966) 40-44.

10. Dawkins, M. J. R.: The Hazards of Birth. Adv. Reprod. Physiol. 1 (1966) 217-264.

11. Dickerson, J. W. T., and Dobbing, J.: Prenatal and Postnatal Growth and Development of the Central Nervous System of the Pig. Proc. Roy. Soc. Lond. 166B (1967) 384-395.

12. Dickerson, J. W. T., Dobbing, J., and McCane, R. A.: The Effect of Undernutrition on the Postnatal Development of the Brain and Cord in Pigs. Proc. Roy. Soc. Lond. 166B (1967) 396-407.

13. Dickerson, J. W. T., Merat, A., and Widdowson, E. M.: Intra-Uterine Growth Retardation in the Pig. III The Chemical Structure of the Brain. Biol. Neonate. 19 (1971) 354-362.

14. Done, J. T., Herbert, C. N.: The Growth of the Cerebellum in the Foetal Pig. Res. Vet. Sci. 9 (1968) 143-148.

15. Dunne, H. W.: Abortion, Stillbirth, Fetal Death and Infectious Infertility. In Diseases of Swine, 4th edition. Edited by H. W. Dunne. The Iowa State University Press, Ames, Iowa, USA, 91970. (1975) 918-952.

16. Dunne, H. W., and Clark, C. D.: Embryonic Death, Fetal Mummification, Stillbirth, and Neonatal Death in Pigs of Gilts Vaccinated with Attenuated Live-Virus Hog Cholera Vaccine. Am. J. Vet. Res. 29 (1968) 786-796.

17. Dunne, H. W., Gobble, J. L., Hokanson, J. F., Kradel, D. C., and Bubash, G. R.: Porcine Reproductive Failure Associated with a Newly Identified SMEDI Group of Picorna Viruses. Am. J. Vet. Res. 26 (1965) 1284-1297.

18. Dunne, H. W., Kradel, D. C., Clark, C. D., Bubash, G. R., and Ammerman, E.: Porcine Enteroviruses. A Serological Comparison of Thirty-Eight Pennsylvania Isolates with Other Reporting North American Strains, Teschen, Talfan, and T80 Serums - A Progress Report. Am. J. Vet. Res. 28 (1967) 557-568.

19. Dunne, H. W., Wang, J. T., and Ammerman, E.: Classification of North American Porcine Enteroviruses: A Comparison with European and Japanese Strains. Infect. Immune. 4 (1971) 619-631.

20. Edwards, B. L.: Causes of Death in Newborn Pigs. Vet. Bull. 42 (1972) 249-258.

21. England, D. C., Day, P. E., and Fogg, T. J.: Association of Birthweight, Birth Interval, and Milk Availability in Early Postnatal Life with Survival and Growth Patterns of Pigs to 72 Hours and 35 Days Post-farrowing. In 1976 Proceedings of the International Pig Veterinary Society, Ames, Iowa, D-7.

22. English, P. R.: A Study of the Apparent Relative Contribution of Factors to Pig Mortality. Preliminary Report to PIDA. Sch. Agri. Univ. Aberdeen. (1968).

23. Fechheimer, N. S.: Chromosome Abnormalities and Embryo Death in Farm Animals. Vet. Rec. 90 (1972) 241-243.

24. Ferguson, L. C. and Powers, T. E.: Experimental Leptospirosis in Pregnant Swine. Am. J. Vet. Res. 17 (1956) 471-477.

25. Frape, D. L., Wolf, K. L., Wilkinson, J., and Chubb, L. G.: Liver Weight and Its N and Vitamin A Contents in Piglets From Sows Fed Two Levels of Protein and Food. J. Agric. Sci. Camb. 73 (1969) 33-40.

26. Frape, D. L., Hocken, R. W. Wolf, K., and Chubb, L. G.: Interrelationships of the Protein, Energy and Food Intake of Sows with the Vitamin A, Nitrogen and Wet Weight of Newborn Piglet Livers. Proc. Nutr. Soc. 26 (1967) XXXVI-XXXVII.

27. Goodwin, R. F. W., and Jennings, A. R.: Mortality of Newborn Pigs Associated with a Material Deficiency of Vitamin A. J. Comp. Path. 68 (1958) 82-95 4 plates.

28. Goodwin, R. F. W.: Some Common Factors in the Pathology of the Newborn Pig. Br. Vet. J. 111 (1955) 361-372.

29. Greber, W. F.: Maternal Influences on Fetal Cardiovascular System in the Sheep, Dog and Rabbit. Amer. J. Physiol. 202 (1962) 653-660.

30. Gruenwald, P.: Chronic Fetal Distress and Placental Insufficiency. Biol. Neonat. 5 (1963) 215-265.

31. Hakkarainen, J.: Om Spadrisens Sjukdomar Under Nyfoddhetsperioden. (Diseases of the Pig in the Neonatal Period). Svensk. Veterinartidning 24 (1973) 527-536.

32. Hsu, S. T., Change, L. C., Lin, S. Y., Chuang, T. Y., Ma, C. H., Inoue, Y. K., and Okuno, T.: The Effect of Vaccination with a Live Attenuated Strain of Japanese Encephalitis Virus on Stillbirths in Swine in Taiwan. Bull. Wld. Hlth. Org. 49 (1972) 465-471.

33. Heard, C. R. C., Platt, B. S., and Stewart, R. J. C.: The Effect on Pigs of a Low Protein Diet with and without Additional Carbohydrates. Proc. Nutr. Soc. 17 (1958) xli-xlii.

34. Howe, J. M., Addis, P. B., Howard, R. D., and Judge, M. D.: Environment Induced Adrenocortical Lipid in "Stress-Susceptible" Pigs. J. Anim. Sci. 28 (1969) 70-72.

35. Illinois Symposium: Factors Producing Embryonic and Fetal Abnormalities, Death and Abortion in Swine. J. Am. Vet. Med. Assoc. 152 (1968) 37-40.

36. Itoh, H., and Hansard, S. L.: Factors Affecting Fetal Liver Glycogen Storage in Swine. J. Anim. Sci. 24 (1965) 889.

37. Jackson, P. G. G.: The Incidence of Stillbirth in Cases of Dystocia in Sows. Vet. Rec. 97 (1975) 411-412.

38. Judge, M. D., Briskey, E. J., Hoestra, C. E., and Forrest, J. C.: Adrenal Hormone Excretion in Relation to Pale, Soft, Exudative Porcine Muscle. J. Anim. Sci. 24 (1965) 864.

39. Leech, F. G.: Biological Factors Affecting Survival, with Particular Relation to Farm Animals. Brit. Vet. J. 132 (1976) 335-345.

40. Leman, A. D., Knudson, C., Rodeffer, H. E., and Mueller, A. G.: Reproductive Performance of Swine on 76 Illinois Farms. J. Am. Vet. Med. Assoc. 161 (1972) 1248-1255.

41. Leman, A. D., Sprecher, D. J.: Effects of Parasympathomimetics on Farrowing and Stillbirth Rate. 1976 Proceedings of The International Pig Veterinary Society. Ames, Iowa. D 20

42. Lee, C. J., and Dubo, R.: Testing Biological Effects of Early Environmental Influences. VIII. Effects of Neonatal Infection, Perinatal Malnutrition and Crowding on Catecholamine Metabolism of Brain. J. Exp. Med. 136 (1972) 1031-1042.

43. Lubs, H. A., and Ruddle, F. H.: Chromosomal Abnormalities in the Human Population. Science 169 (1970) 495-497.

44. Marsh, J. B., and Drabkin, D. L.: Metabolis Channeling in Experimental Nephrosis, Part III. J. Biol. Chem. 230 (1958) 1063-1071.

45. McAdaragh, J. P., and Robl, M. G.: Experimental Reovirus Infection of Pregnant Sows. 1976 Proceedings of the International Pig Veterinary Society, Ames, Iowa. DD1

46. McFeely, R. A.: Chromosome Abnormalities in Early Embryos of the Pig. J. Reprod. and Fert. 13 (1967) 579-581.

47. McLaren, A., and Mitchie, D.: Congenital Runts. In Ciba Foundation Symposium on Congenital Malformations. Edited by G. E. W. Wolstenholme and C. M. Oconnor. Churchill Ltd., London, 178-193.

48. Mengeling, W. L., and Cutlip, R. C.: Reproductive Failure of Swine Associated with Porcine Parvovirus Infection. 1976 Proceedings of the International Pig Veterinary Society, Ames, Iowa. DD4

49. Michna, S. W.: Abortion in the Sow Due to Infection by Leptospira Canicola. A Preliminary Report. Vet. Rec. 74 (1962) 917-919.

50. Michna, S. W.: Further Observations on Abortion of the Sow Due to Infection by Leptospira Canicola. Vet. Rec. 77 (1965) 802-809.

51. Michna, S. W.: Animal Leptospirosis in the British Isles, A Serological Survey. Vet. Rec. 80 (1967) 394-400.

52. Milner, R. D. G.: Nutrition and Growth of the Foetus and Newborn Infant. Nutrition 27 (1973) 215-265.

53. Milosavljevic, S., Sovljanski, B., Murganski, S., Radovic, B., and Trbojevic, G.: Dependence of the Incidence of Stillborn Piglets on the Age of Sows. Acta. Vet. 21 (1971) 237-242.

54. Moore, R. W.: Iron Deficiency Anemia as a Cause of Stillbirths in Swine. J. Am. Vet. Med. Assoc. 147 (1965) 746-748.

55. Morrill, C. C.: Studies on Baby Pig Mortality. XI. A Note on the Influence of Fasting on Body Temperature, Body Weight, and Liver Weight of the Newborn Pig. Am. J. Vet. Res. 13 (1952) 164-180.

56. Mount, L. E.: The Climatic Physiology of the Pig. London (1968).

57. Naeye, R. L.: Nutrition. Arch. Path. 79 (1965) 284-291.

58. Newland, H. W. McMille, W. N., and Reineke, E. P.: Temperature Adaptation in the Pig. J. Anim. Sci. 11 (1952) 118-133.

59. Nielsen, H. E., and Bendixen, H. J.: Undersogelser Over Fosterdod Hos Svin. (Foetal Death in Swine). Landok. Forsogslab. Arbog. Copenhagen (1969) 23-32.

60. Nielsen, N. C., Christensen, K., Bille, N., and Larsen, J. L.: Pre-Weaning Mortality in Pigs. I. Herd Investigations. Nord. Vet. Med. 26 (1974) 137-150.

61. Palludan, B.: The Teratogenic Effect of Vitamin A Deficiency in Pigs. Acta. Vet. Scand. 2 (1961) 32-59.

62. Perry, J. S., and Rowell, J. G.: Variations in Foetal Weight and Vascular Supply Along the Uterine Horn of the Pig. J. Reprod. Fert. 19 (1969)527-534.

63. Platt, B. S., and Stewart, R. J. C.: Experimental Protein Calorie Deficiency: Histopathological Changes in the Endocrine Glands of Pigs. J. Endocrin. 38 (1967) 121-142.

64. Platt, B. S., and Stewart, R. J. C.: Effects of Protein Calorie Deficiency on Dogs Reproduction, Growth and Behavior Development. Med. Child. Neurol. 10 (1968) 324.

65. Pomeroy, R. W.: Infertility and Neonatal Mortality in the Sow. III. Neonatal Mortality and Foetal Development. J. Agric. Sci. Camb. 54 (1960) 31-56.

66. Pond, W. G., Stracham, D. N., Sinha, Y. N., Walker, Jr., E. F., Dunn, J. A., and Barnes, R. H.: Effects of Protein Deprivation on Swine During All or Part of Gestation on Birth Weight, Postnatal Growth Rate and Nucleic Acid Content of Brain and Muscle of Progeny. J. Nutr. 99 (1969) 61-67.

67. Randall, G. C. B.: The Relationship of Arterial Blood pH and pCO_2 to Viability of the Newborn Piglet. Canad. J. Comp. Med. 35 (1971) 141-146.

68. Randall, G. C. B.: Observations on Parturition in the Sow, II. Factors Influencing Stillbirth and Perinatal Mortality. Vet. Rec. 90 (1972) 183-186.

69. Randall, G. C. B., and Penny, R. H. C.: Stillbirth in the Pig: An Analysis of the Breeding Records of Five Herds. Br. Vet. J. 126 (1970) 593-603.

70. Saffer, P., and Simon, D. L.: Zur Abhangigkeit Der Ferkelgewichte Wahrend Der Saugeperiode. (On the Dependence of Piglet Weights During the Suckling Period). Zuchtungskunde. 42 (1970) 122-134.

71. Schain, R. J., Carver, M. J., Copenhaver, J. H., and Underdahl, N. R.: Protein Metabolism in the Developong Brain: Influence of Birth and Gestation Age. Science 156 (1967) 984-985.

72. Sprecher, D. J., Leman, A. D., Dziuk, P. D., Cropper, M., and DeDecker, M.: Causes and Control of Swine Stillbirths. J. Am. Vet. Med. Assoc. 165 (1974) 698-701.

73. Sprecher, D. J., Leman, A. D., and Carlisle, S.: Effects of Parasympathomimetics on Porcine Stillbirth. Am. J. Vet. Res. 36 (1975) 1331-1333.

74. Stanton, H. C., and Carroll, J. K.: Potential Mechanisms Responsible for Prenatal and Perinatal Mortality or Low Viability of Swine. J. Anim. Sci. 38 (1974) 1037-1044.

75. Stanton, H. C., Brown, L. J., and Mueller, R. L.: Interrelationships Between Maternal and Neonatal Factors and Thermoregulation in Fasted Neonatal Swine. (Sus domesticus). Comp. Biochem. and Physiol. 44A (1973) 97-105.

76. Stave, U.: Maturation, Adaptation and Tolerance. In Physiology of the Neonatal Period. Vol. I., U. Stave, Editor, Appleton, Century, Crofts, New York (1970) 29-40.

77. Topel, D. F., Bicknell, E. J., Preston, K. S., Christian, L. L., and Matsushima, C. Y.: Porcine Stress Syndrome. Mod. Vet. Pract. 49 (1968) 59-60.

78. Tumbleson, M. E., Tinsley, O. W., Mulder, J. B., and Flatt, R. E.: Undernutrition in Young Sinclair S-I Miniature Swine: Body and Organ Weights. Growth 34 (1970) 401-418

79. Ullery, D. E., Sprague, J. I., Becker, D. E., and Miller, E. R.: Growth of the Swine Fetus. J. Anim. Sci. 24 (1965) 711-717.

80. Wallach, D. P., Newland, H. W., and McMillen, W. N.: Stome Studies on Temperature Adaption in the Baby Pig. Mich. State Univ. Agric. Exp. Sta. Quart. Bull. 49 (1947) 30-31, 277-282.

81. Wenham, G., McDonald, I., and Elsley, F. W. H.: A Radiographic Study of the Development of the Skeleton of the Foetal Pig. J. Agri. Sci., Camb. 72 (1969) 123-130.

82. Widdowson, E. M.: Intra-uterine Growth Retardation in the Pig. I. Organ Size and Cellular Development at Birth and After Growth to Maturity. Biol. Neonate. 19 (1971) 329-340.

83. Wigglesworth, J. S.: Foetal Growth Retardation. Brit. Med. Bull. 22 (1966) 13-15.

84. Yuwiler, A.: Stress. In Handbook of Neurochemistry., Vol. 6, Edited by A. Lajtha, Plenum Press, New York. (1971) 103-171.

APPENDIX

Table 1: Means and Standard Errors Measured in Grams for All the Recorded Classes and Subclasses

		Body		Thymus		Liver		Brain		Spleen		Femur	
Class	No.	Mean	S.E.	Mean	S.E.	Mean	S.E.	Mean	S.E.	Mean	S.E.	Mean	S.E.
Normal	10	1434.08	98.16	0.70	0.30	47.65	3.35	31.74	1.44	1.19	0.29	4.01	0.09
Normal Male	6	1458.16	124.16	0.63	0.38	52.63	4.24	31.56	1.82	1.18	0.37		
Normal Female	4	1409.99	152.07	0.77	0.47	42.67	5.19	31.92	2.23	1.19	0.45		
Stillborn NND	200	1094.40	22.27	1.18	0.07	30.91	0.77	25.33	0.48	1.45	0.07	3.74	0.35
Neonatal Death	67	1076.36	42.15	1.09	0.14	30.14	1.46	26.80	0.62	1.42	0.13		
Stillborn	133	1108.30	28.73	1.23	0.09	30.84	0.99	26.45	0.42	1.45	0.09		
Herd 1 SNND	132	1069.89	28.79	1.29	0.10	30.86	1.00	25.65	0.43	1.46	0.09	3.73	0.35
Herd 2 SNND	68	1114.77	42.21	1.03	0.14	30.12	1.46	27.61	0.61	1.41	0.13	3.76	0.37
Male SNND	108	1071.69	36.30	1.18	0.12	29.49	1.26	26.55	0.53	1.43	0.11	3.70	0.38
Female SNND	92	1112.97	35.95	1.14	0.12	31.49	1.25	26.70	0.52	1.43	0.11	3.79	0.31
NND Herd 1	47	1051.26	46.01	1.26	0.16	30.26	1.60	25.20	0.70	1.47	0.14		
NND Herd 2	20	1101.45	70.87	0.91	0.24	30.02	2.46	28.41	1.02	1.36	0.22		
Still. Herd 1	85	1088.52	34.61	1.31	0.11	31.46	1.20	26.09	0.51	1.45	0.11		
Still. Herd 2	48	1128.08	45.88	1.14	0.15	30.21	1.59	26.81	0.66	1.45	0.14		
NND Male	32	1001.75	62.00	1.03	0.21	26.98	2.15	26.09	0.91	1.35	0.19		
NND Female	35	1150.97	57.41	1.14	0.19	33.30	1.99	27.51	0.84	1.48	0.18		
Still. Male	76	1141.62	37.79	1.33	0.12	31.99	1.31	27.01	0.55	1.52	0.12		
Still. Female	57	1074.97	43.29	1.13	0.14	29.68	1.50	25.88	0.63	1.38	0.13		
Herd 1 M SNND	72	1061.56	39.85	1.29	0.14	30.73	1.38	25.64	0.61	1.56	0.12		
Herd 1 F SNND	60	1078.22	41.55	1.29	0.14	31.00	1.44	25.65	0.62	1.36	0.13		
Herd 2 M SNND	36	1081.81	60.69	1.07	0.20	28.25	2.11	27.47	0.88	1.31	0.19		
Herd 2 F SNND	32	1147.72	58.69	0.98	0.19	31.98	2.04	27.75	0.85	1.50	0.18		
NND Herd 1 M	23	1019.95	65.76	1.19	0.23	28.74	2.28	24.63	1.02	1.59	0.20		
NND Herd 1 F	24	1082.58	64.37	1.33	0.22	31.79	2.23	25.76	0.97	1.35	0.20		
NND Herd 2 M	9	873,55	105.12	0.86	0.35	25.23	3.65	27.55	1.52	1.11	0.33		
NND Herd 2 F	11	1219.36	95.09	0.96	0.32	34.81	3.30	29.27	1.38	1.61	0.30		
Still. Herd 1 M	49	1103.18	45.05	1.38	0.15	32.72	1.56	26.64	0.66	1.52	0.14		
Still. Herd 1 F	35	1073.85	52.56	1.24	0.18	30.20	1.82	25.55	0.77	1.38	0.16		
Still. Herd 2 M	27	1180.07	60.69	1.28	0.20	31.27	2.11	27.39	0.88	1.51	0.19		
Still. Herd 2 F	21	1076.09	68.82	1.01	0.23	29.15	2.39	26.22	0.99	1.39	0.21		

Table 2

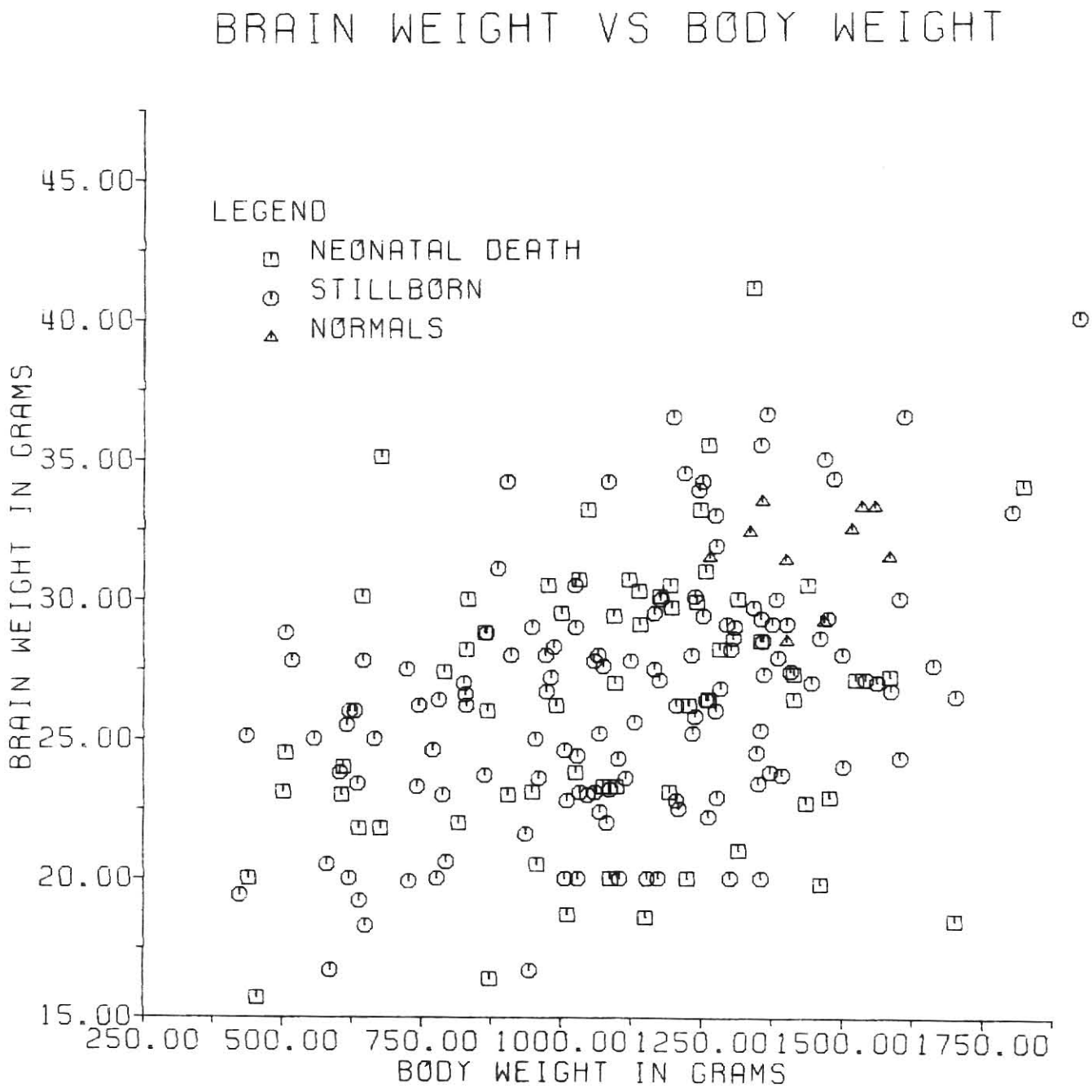

BRAIN WEIGHT VS BODY WEIGHT
LEGEND
NEONATAL DEATH
STILLBORN
NORMALS
BRAIN WEIGHT IN GRAMS
45.00
40.00
35.00
30.00
25.00
20.00
15.00
250.00
500.00
750.00
1000.00
1250.00
1500.00
1750.00
BODY WEIGHT IN GRAMS

Table 3

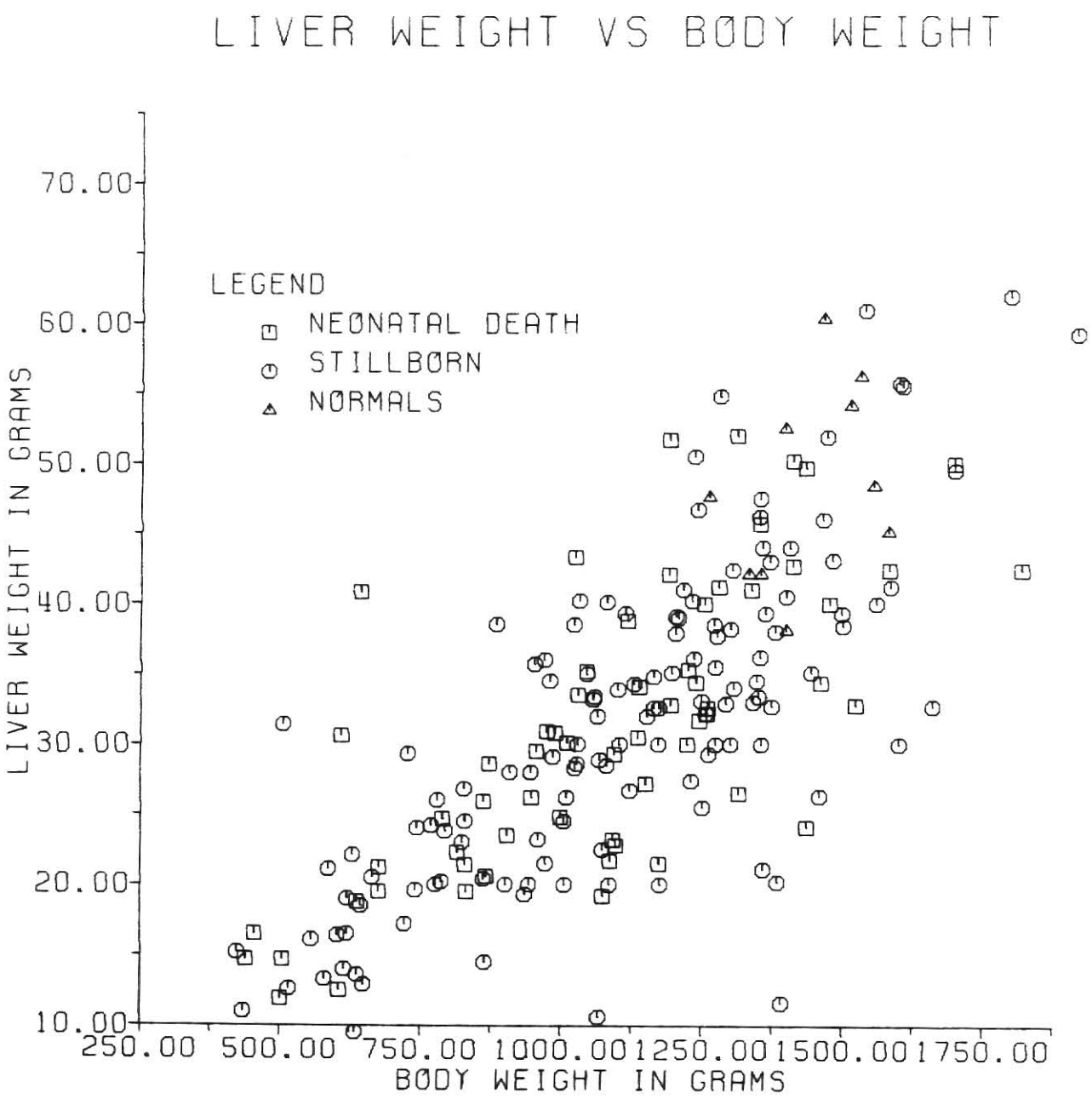

LIVER WEIGHT VS BODY WEIGHT
LEGEND
NEONATAL DEATH
STILLBORN
NORMALS
LIVER WEIGHT IN GRAMS
70.00
60.00
50.00
40.00
30.00
20.00
10.00
250.00
500.00
750.00
1000.00
1250.00
1500.00
1750.00
BODY WEIGHT IN GRAMS

Table 4

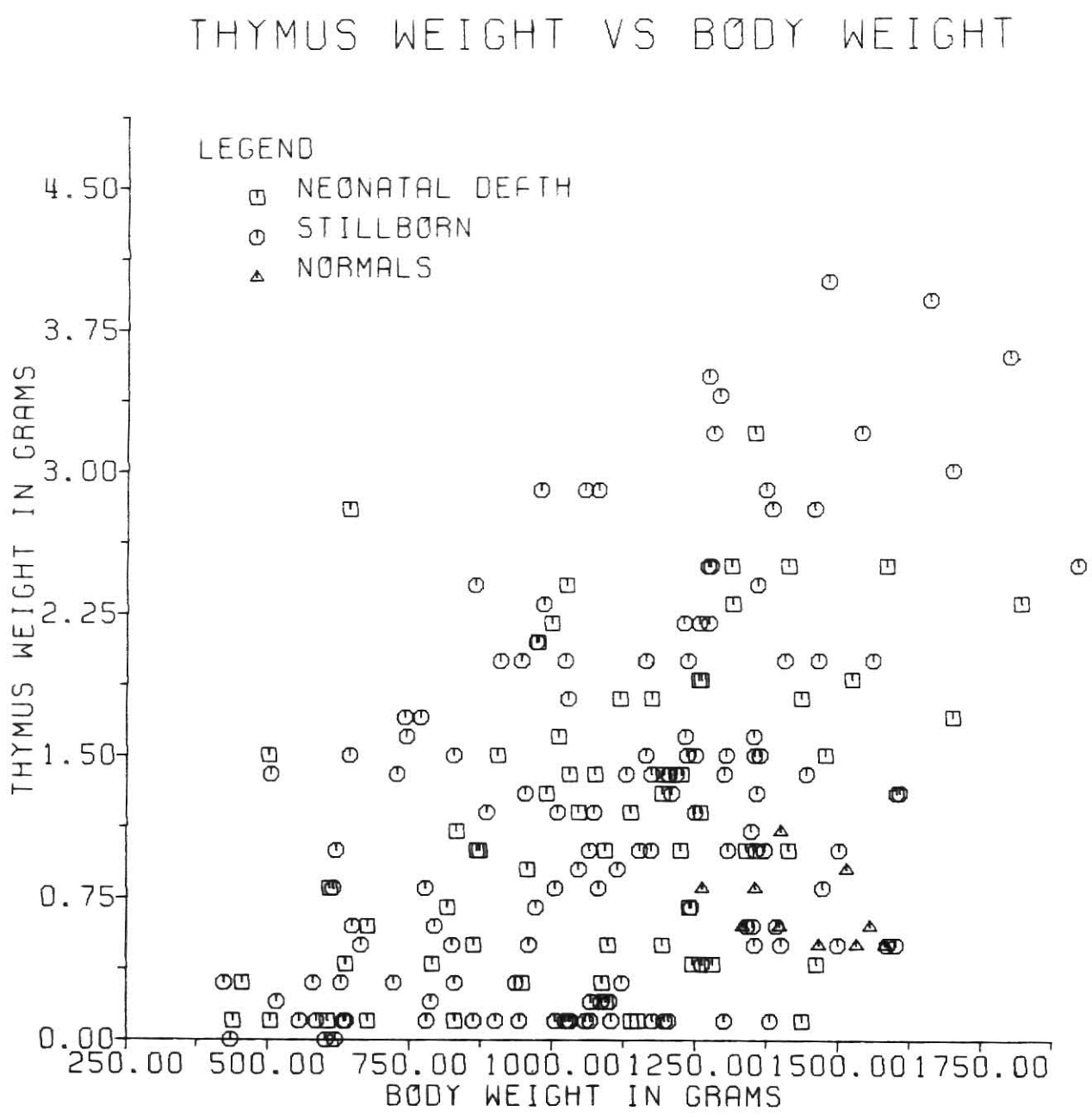
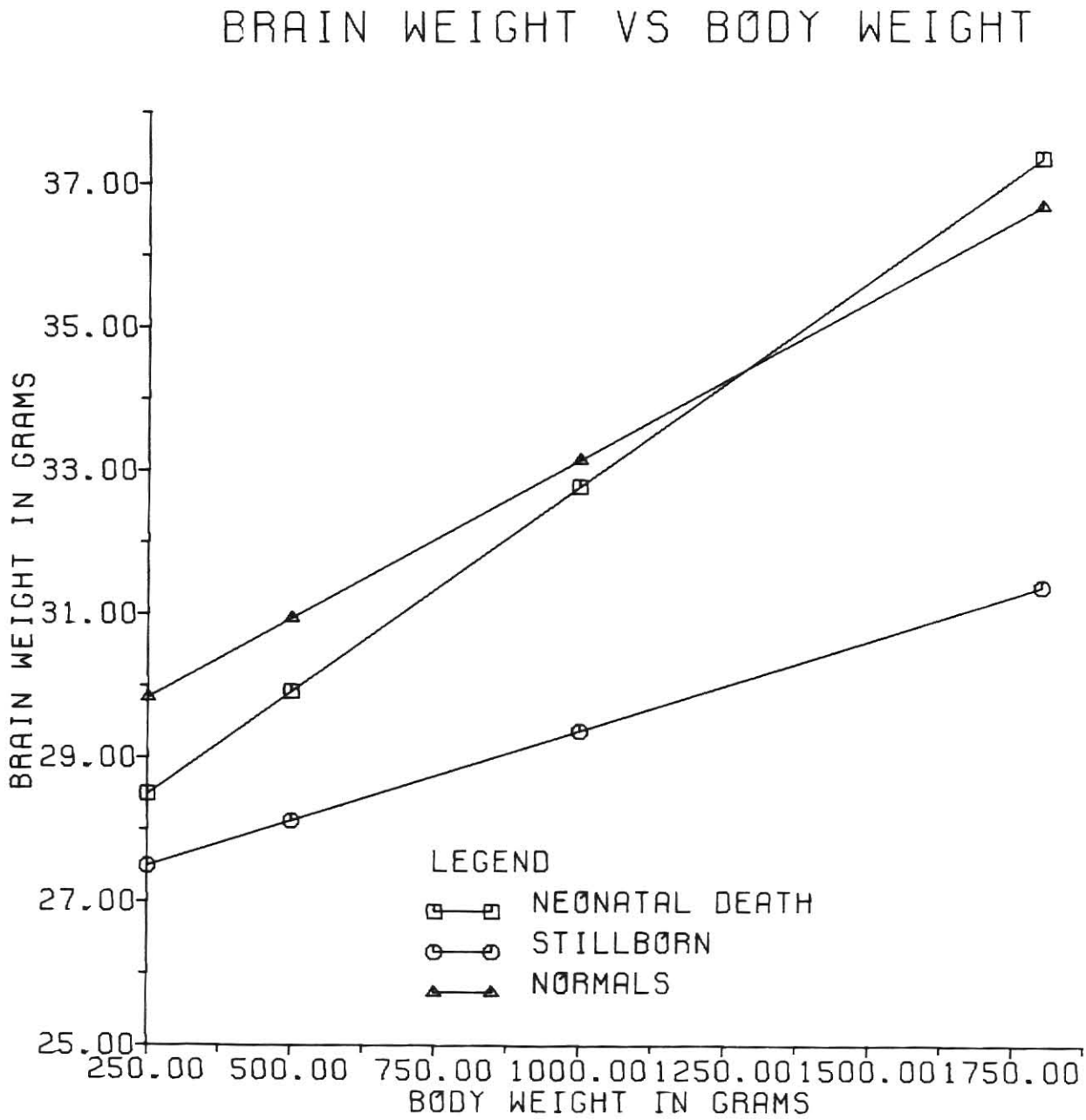
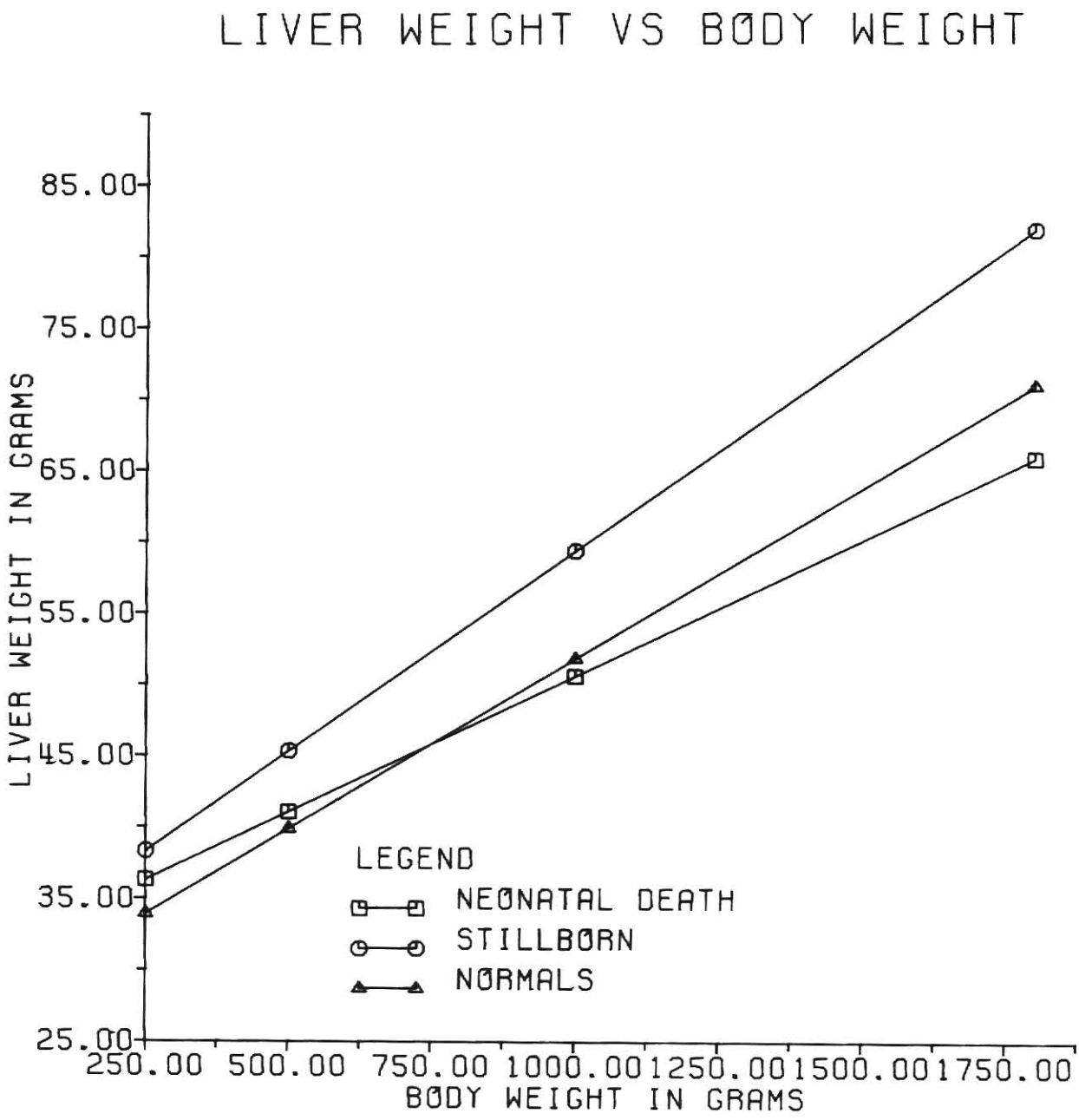

THYMUS WEIGHT VS BODY WEIGHT
LEGEND
NEONATAL DEATH
STILLBORN
NORMALS
THYMUS WEIGHT IN GRAMS
4.50
3.75
3.00
2.25
1.50
0.75
0.00
250.00
500.00
750.00
1000.00
1250.00
1500.00
1750.00
BODY WEIGHT IN GRAMS

Table 5


BRAIN WEIGHT VS BODY WEIGHT
BRAIN WEIGHT IN GRAMS
37.00
35.00
33.00
31.00
29.00
27.00
25.00
250.00 500.00 750.00 1000.00 1250.00 1500.00 1750.00
BODY WEIGHT IN GRAMS
LEGEND
NEONATAL DEATH
STILLBORN
NORMALS


LIVER WEIGHT VS BØDY WEIGHT
LIVER WEIGHT IN GRAMS
85.00
75.00
65.00
55.00
45.00
35.00
25.00
250.00 500.00 750.00 1000.00 1250.00 1500.00 1750.00
BØDY WEIGHT IN GRAMS
LEGEND
NEØNATAL DEATH
STILLBØRN
NØRMALS

THIS BOOK CONTAINS SEVERAL DOCUMENTS THAT ARE OF POOR QUALITY DUE TO BEING A PHOTOCOPY OF A PHOTO.

THIS IS AS RECEIVED FROM CUSTOMER.

Figure 1

Femoral radiographs from stillborn, neonatal death
Class Nos. 1-50 (omit 21 and 22)

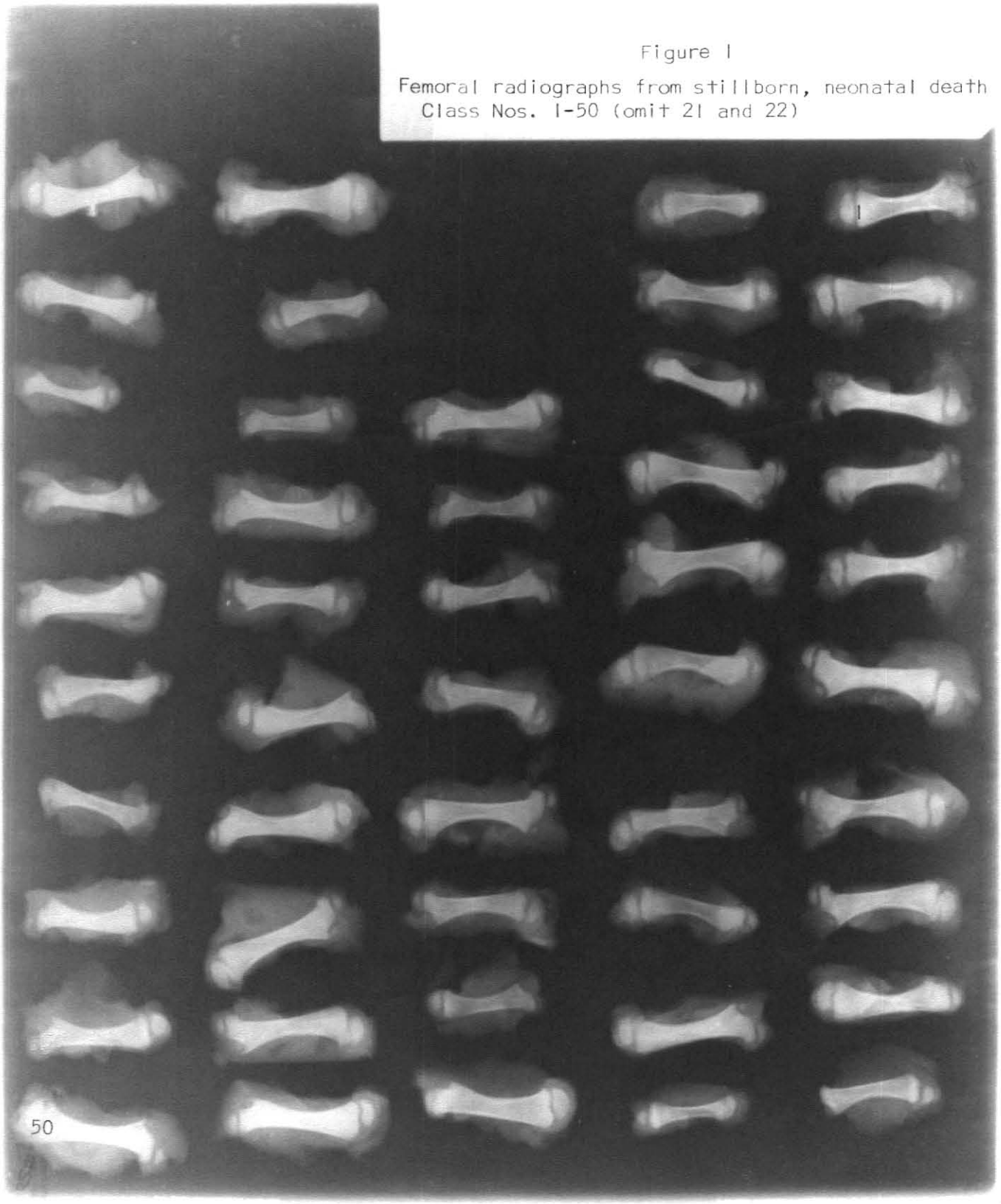


Figure 2

Femoral radiographs from stillborn, neonatal death Class Nos. 51-100 (omit 78, 94 and 96)

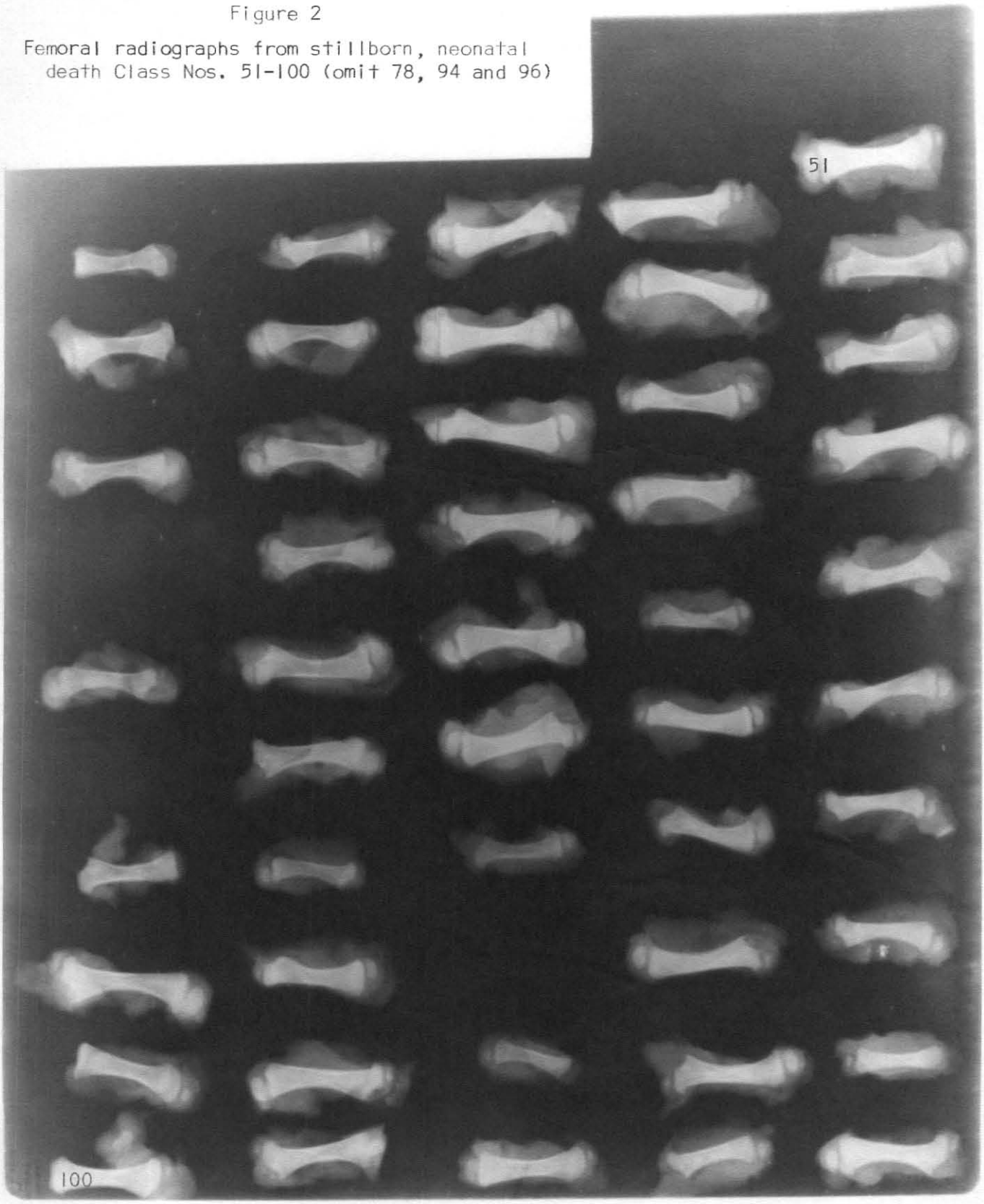


Figure 3

Femoral radiographs from stillborn, neonatal death Class Nos. 101-150 (omit 146)

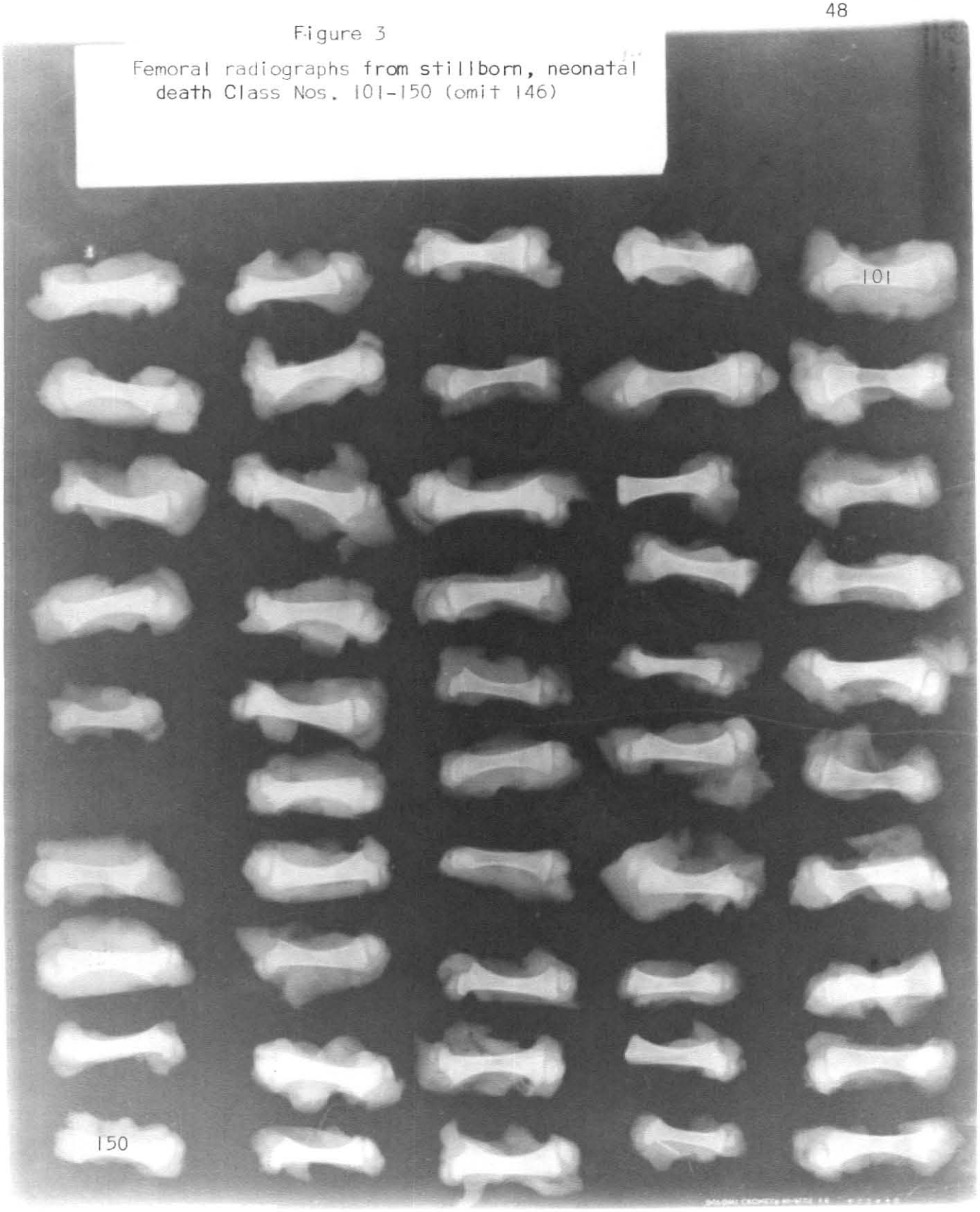
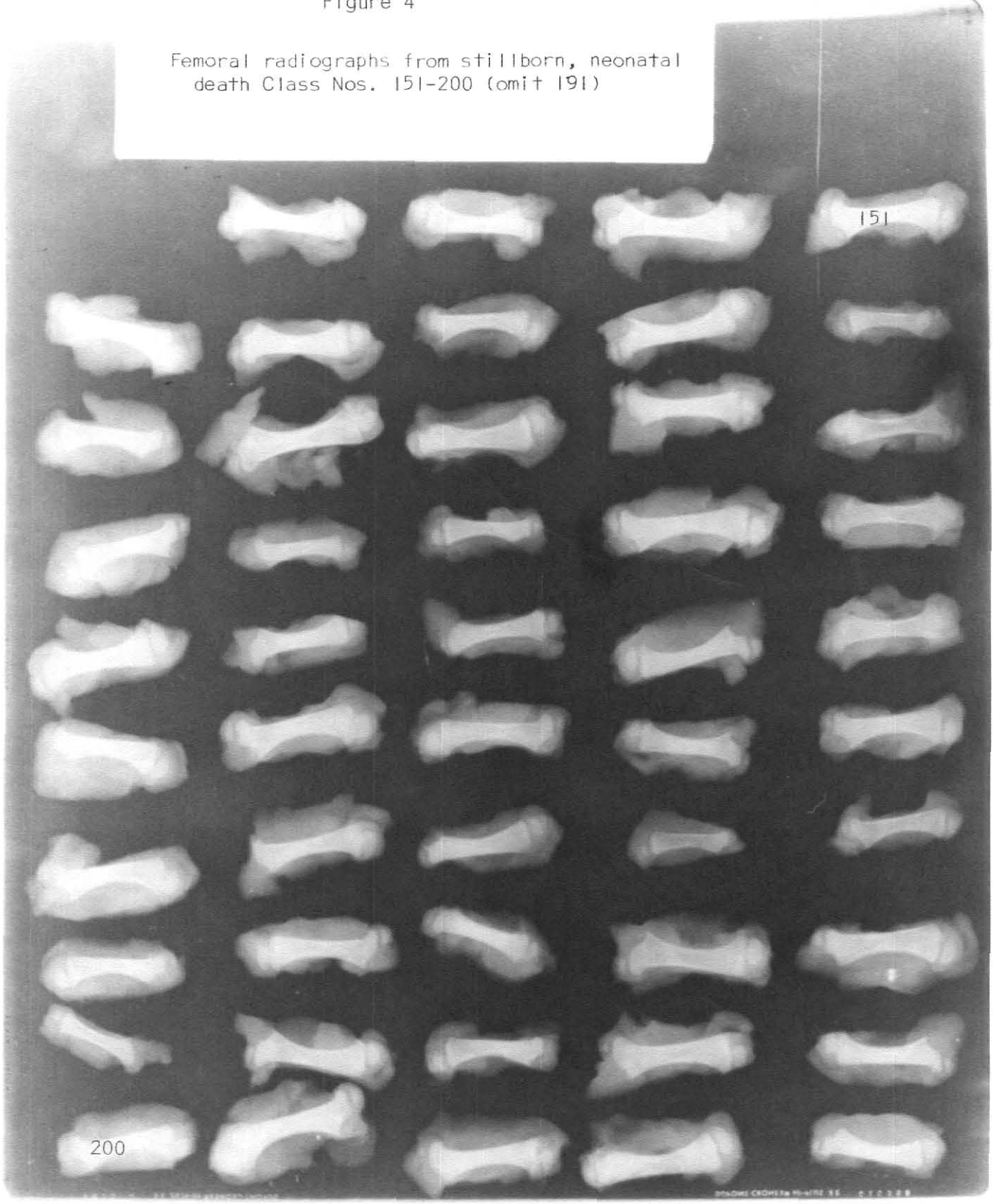


Figure 4

Femoral radiographs from stillborn, neonatal death Class Nos. 151-200 (omit 191)



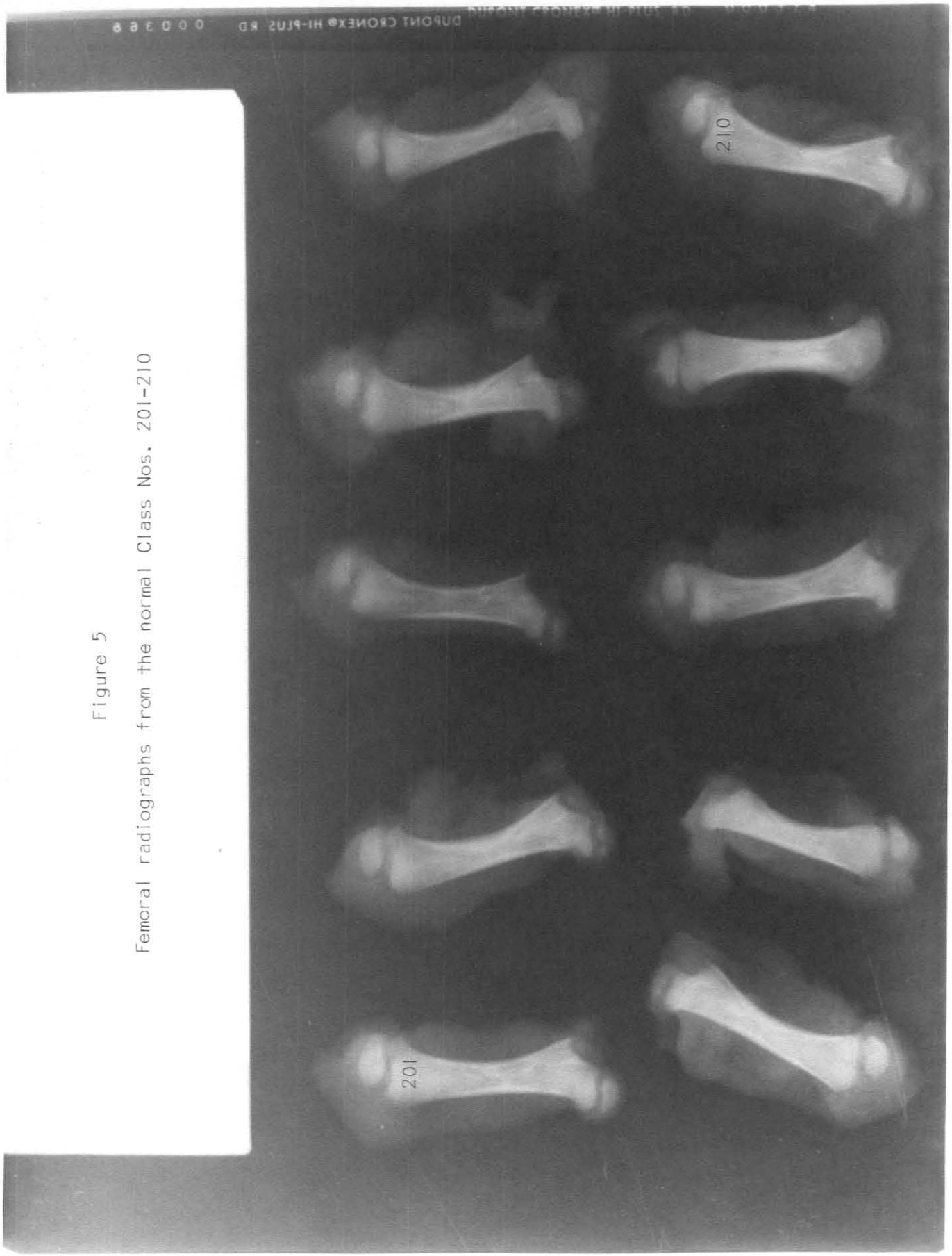


Figure 5

Femoral radiographs from the normal Class Nos. 201-210

BODY WEIGHTS AND SELECTED ORGAN MEASUREMENTS FROM PIGS STILLBORN OR DYING NEONATALLY

by

CHARLES EDWARD HERREN

B.S., Kansas State University, 1954
D.V.M., Kansas State University, 1954

AN ABSTRACT OF A MASTER'S THESIS

submitted in partial fulfillment of the

requirements for the degree

MASTER OF SCIENCE

Department of Surgery and Medicine

KANSAS STATE UNIVERSITY
Manhattan, Kansas

1977

ABSTRACT

This study was undertaken in an effort to shed some light on the swine industry's continuing problem of stillbirth and early neonatal death. 200 full term stillborn pigs and those dying within 48 hours post-partum were taken from two farrowing units, weighed and necropsied according to a prescribed procedure. The thymus, spleen liver and brain were removed and weighed. The femur was disarticulated, trimmed and radiographed to permit measurement. A pericardial fluid sample was collected by sterile technique and examined for the presence of spirochaetes.

The data was subjected to an unequal subclass analysis of variants. The parameters for class division were: 1. Stillborn - pigs with atelectic lungs, 2. Normal - active pigs euthanatized before 48 hours of age, 3. Neonatal death (NND) - as evidenced by aerated lungs and/or food in the stomach.

The results of this study may be summarized as follows. The normal sexual grouping of pigs of 52% males and 48% females was found to be altered to 54% males and 46% females in the stillborn, neonatal death (NND) group. The males in this group were 41.38 grams lighter than the females. Stillborn pigs were 32 grams heavier than liveborn pigs dying within 48 hours (NND). Stillborn males weighed more than stillborn females and females dying neonatally (NND) weighed more than males in the same class.

Herd differences in body weights and organ measurements were insignificant. Least Significant Differences of the mean body, liver and brain

weights for the 3 important subclasses are as follows:

	LEAST SIGNIFICANT DIFFERENCES		
	Body Weight	Brain Weight	Liver Weight
Neonatal Death over Stillborn	111.19	1.70	3.79
Neonatal Death over Normals	213.68	3.17	7.29
Stillborn over Normals	205.56	3.02	7.02

Regression lines of organ weights as a function of body weights were not linear and demonstrated poor confidence limits because of the extreme dispersion of the data. A formula was devised to fit this function. When plotted, liver weights for the stillborn pigs were insignificantly greater than the liver weights of the neonatal death (NND) and normal groups. The brain weights of the stillborn group, when plotted, were significantly smaller than brain weights of either the normal or the live born pigs dying within 48 hours.

Results of the study suggest that while intrapartum anoxia may be the immediate cause of nonepidemic stillbirth and neonatal death, an underlying cause of diminished brain size may predispose at least stillbirth. We might hypothesize that the decreased brain size found in the stillborn pigs may be due to reduced uterine vasculature causing fetal and placental undernutrition to certain occupants of the uterine horn. This avenue of investigation needs to be explored further.