HYPERTROPHIC PYLORIC CANAL STENOSIS IN THE NEWBORN PIG

With Reference to the Gross and Microscopic Anatomy and Physiology of the Stomach of the Normal Newborn Pig

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TO E. A. SHARP, M. D., WHO GUIDED ME THROUGH MY APPRENTICESHIP IN MEDICAL RESEARCH AND WHOSE FRIENDSHIP I AM PRIVILEGED TO ENJOY, THIS THESIS IS DEDICATED.

TABLE OF CONTENTS

		Page
I.	THE PROBLEM	1
	Preface	1
	Statement of the Problem	7
	Limitation of the Study	9
	Clarification of Terms	10
II.	REVIEW OF THE LITERATURE	11
	Baby Pig Disease (baby pig hypoglycemia)	11
	Hypertrophic Pyloric Canal Stenosis in Domestic Animals	15
	Hypertrophic Pyloric Canal Stenosis in Man	16
	Gross Anatomy of the Stomach of the Adult Pig	21
	Gross Anatomy of the Stomach of the Newborn Pig	22
	Gross Anatomy of the Stomach of the Newborn Infant	22
	Comparative Physiology of the Stomach of the Newborn Pig and Infant	23
	Gastric Emptying Time	23
	Volume of Gastric Juice	23
	Free Hydrochloric Acid	24
	pH of Gastric Juice	24
	Pepsin and Rennin	24
	Physical Character of Milk Curd	25
	Blood Sugar Level of the Newborn Pig	25
	Liver Glycogen of the Newborn Pig	25
III.	DISTRIBUTION OF GROSS PATHOLOGY IN SEVENTY-SIX PIGS ELIMINATED FOR VARIOUS REASONS FROM THE COLLEGE HERD DURING THE FARROWING SEASON OF SPRING 1956	26
IV.	INVESTIGATIONAL PROCEDURE AND RESULTS	29
	Fixation of the Stomach in Situ During Life	29
	Gross Anatomy of the Stomach of the Normal Newborn Pig	31
	Pylorus	31
	Pyloric Canal	32
	Microscopic Anatomy of the Stomach of the Normal Newborn Pig	66
	Pylorus	66
	Pyloric Canal	68

Page

Physiology of the Stomach of the Normal Newborn Pig 86
Gastric Emptying Time 86
Volume of Gastric Juice 88
Free Hydrochloric Acid 88
pH of Gastric Juice 89
Pepsin and Rennin 90
Physical Character of Milk Curd 91
Blood Sugar Level 93
Liver Glycogen. 93
Gross Anatomy of the Stomach of the Newborn Pig with Hypertrophic Pyloric Canal Stenosis. 95
Pylorus 95
Pyloric Canal 95
Microscopic Anatomy of the Stomach of the Newborn Pig with Hypertrophic Pyloric Canal Stenosis. . . 112
Pylorus 112
Pyloric Canal 112
Physiology of the Stomach of the Newborn Pig with Hypertrophic Pyloric Canal Stenosis 123
Gastric Emptying Time 123
Free Hydrochloric Acid. 123
pH of Gastric Juice 123
Pepsin and Rennin 124
Physical Character of Milk Curd 124
Blood Sugar Level 124
Liver Glycogen and Fat. 125
Behaviorism of the Newborn Pig with Hypertrophic Pyloric Canal Stenosis. 126

V. OTHER PATHOLOGICAL CONDITIONS INVOLVING THE STOMACH AND PYLORIC CANAL IN NEWBORN PIGS 130

Atony of the Stomach and Obstruction of the Pyloric Canal by a Tenacious Plug of Mucus in a Full-term Stillborn Pig 130
Atrophy of the Greater Curvature Musculature of the Pylorus in a Newborn Pig Three Days of Age. . . . 134
Increased Thickness of the Greater Curvature Musculature of the Pylorus and Pyloric Canal in a Full-term Stillborn Pig 134
Malformed "Intra-gastric" Pyloric Canal in a Full-term Stillborn Pig. 135
Pyloric Ulcer and Pyloric Canal Occlusion by Interstitial and Mucosal Edema in a Newborn Pig Three Days of Age 139

VI. DISCUSSION AND SUMMARY 143

REFERENCES CITED. 147

VITA 155

LIST OF TABLES

Table		Page
1.	Distribution of Gross Pathology in Seventy-six Pigs Eliminated for Various Reasons from the College Herd During the Farrowing Season of Spring 1956. . . .	28
2.	Range and Average Dimensions of the Gross Anatomical Structures of the Pyloric Canal of the Stomach of Pigs at Birth (twenty-five pigs), and Tenth Postnatal Day (ten pigs)	59
3.	Range and Average Blood Sugar Concentration of Normal Newborn Pigs at Birth, Fifth and Tenth Postnatal Day in mg/100 ml. of Whole Blood	94
4.	Physical, Biochemical and Histochemical Data of Ten Newborn Pigs with Hypertrophic Pyloric Canal Stenosis	129

LIST OF FIGURES

Figure | Page

Normal Gross Anatomy

1. Stomach of a pig ten days of age 38
2. Pylorus of a pig ten days of age 40
3. Distal end of pyloric vestibule of a pig ten days of age . 42
4. Mucosal folds in the pyloric canal of a newborn pig . . . 44
5. Torus pyloricus longitudinalis and mucosal folds in a pig ten days of age 44
6. Longitudinal mucosal folds in a starving newborn pig . . . 46
7. Longitudinal mucosal folds in a suckling pig ten days of age 46
8. Torus pyloricus longitudinalis of a pig at birth 48
9. Shapes and positions of the torus pyloricus longitudinalis in a pig at birth 48
10. Duodenum and tail of torus pyloricus longitudinalis in a newborn pig 50
11. Transverse section through pyloric canal of a newborn pig 50
12. Stomach of a 90 mm. pig fetus 52
13. Stomach of a pig at birth 54
14. Pyloric canal (normal standard) of a pig at birth 56
15. Schematic diagram and mean measurements of pyloric canal (normal standard) of a pig at birth 58
16. Schematic diagram and mean measurements of the torus pyloricus longitudinalis (normal standard) of a pig at birth 58

Figure Page

17. Pylorus of a pig thirty days of age 61

18. Schematic diagram and major divisions of the stomach of a pig ten days of age 63

19. Schematic diagram of an adult pig stomach 65

20. Schematic diagram of a newborn pig stomach 65

21. Schematic diagram of the human stomach 65

Normal Microscopic Anatomy

22. Section through gastric sphincter and concave curvature of pyloric canal of a pig at birth 73

23. Section through the duodenal sphincter and concave curvature of pyloric canal of a pig at birth 73

24. Section through pyloric vestibule of a pig at birth 75

25. Section through mucous membrane of pylorus of a pig at birth 75

26. Transitional glands in the pyloric canal of a newborn pig . 77

27. Glands of torus pyloricus longitudinalis of a pig at birth. 77

28. Gastric sphincter connective tissue septal pattern of a pig at birth (color) 79

29. Concave curvature connective tissue septal pattern of a pig at birth (color) 79

30. Duodenal sphincter connective tissue septal pattern of a pig at birth (color) 79

31. Torus pyloricus longitudinalis of an adult pig 81

32. Torus pyloricus longitudinalis of a pig at birth 81

33. Torus pyloricus longitudinalis connective tissue septal pattern of a pig at birth (color) 83

34. Muscularis mucosae discontinuity in the torus pyloricus longitudinalis of a pig at birth (color) 83

35. Elastic fibers in the torus pyloricus longitudinalis of a pig at birth 85

Figure Page

36. Glycogen distribution in the liver of a newborn pig 85

Abnormal Gross Anatomy

37. Reference transverse planes of the pyloric canal of a pig at birth 99

38. Reference transverse sections of the pyloric canal at level of planes 1, 2, and 3 99

39. Transverse sections of stenosed pyloric canal (duodenal sphincter) of a pig six days of age 101

40. Transverse sections of stenosed pyloric canal of a pig one day of age 101

41. Twenty-four hour radiograph of abdominal region of a pig six days of age with pyloric canal stenosis. 103

42. Twenty-four hour radiograph of the abdominal region of a pig six days of age (normal control) 103

43-A Transverse sections of pyloric canal of a pig six days of age (normal control) 105

43-B Transverse sections of stenosed pyloric canal of a pig six days of age 105

44. Twenty-four hour radiograph of stomach of a pig eleven days of age with pyloric canal stenosis 107

45. Transverse stomach and pyloric canal of a pig eleven days of age with pyloric canal stenosis 109

46. Stenosed pyloric canal of a pig eleven days of age 111

47. Pyloric canal of a pig ten days of age (normal standard of comparison) 111

Abnormal Microscopic Anatomy

48. Hypertrophy of the middle circular muscle coat of the pyloric canal 116

49. Hypertrophy of pyloric canal glands 116

Figures Page

50. Hyperplasia of collagenic fibers in the torus pyloricus longitudinalis 118

51. Elastic fibers in the greater curvature mucosa 118

52. Hyperplasia of connective tissue in the concave curvature (color) 120

53. Hyperplasia of connective tissue in the torus pyloricus longitudinalis (color) 120

54. Fatty liver 122

Pathologic Conditions of the Stomach

Involving the Pyloric Canal

55. Distal portion of pyloric vestibule of an atonic stomach. . 133

56. Pyloric canal of the atonic stomach shown in Fig. 55. . . . 133

57-A Atrophy of greater curvature musculature of the pyloric canal in a full-term stillborn pig 138

57-B Hypertrophy of the greater curvature musculature of the pyloric canal in a full-term stillborn pig 138

58. Malformed "intra-gastric" pyloric canal in a full-term stillborn pig. 138

59. Lesser curvature pyloric ulcer in a pig three days of age . 142

60. Stenosed pyloric canal in a pig three days of age with a pyloric ulcer (Fig. 59). 142

I. THE PROBLEM

"Der Tag der letzten Hypothese wäre
auch der Tag der letzten Beobachtung"
Friedrich Gustav Jakob Henle

PREFACE

The purpose of this thesis is to present evidence of the occurrence of "hypertrophic pyloric canal stenosis" in the newborn pig, heretofore not reported in this species.

We believe that "hypertrophic pyloric canal stenosis" is a distinct clinical entity in the pig. The disease may be defined as an enlargement of one or more parts of the pyloric canal through hypertrophy (increase in size), hyperplasia (increase in quantity), or both, of tissue constituents without a visible causative lesion, resulting in stenosis (abnormal narrowing) of the lumen.

Hypertrophic pyloric canal stenosis was the cause or a contributory cause of baby pig losses in the herd of the Oklahoma Agricultural and Mechanical College during the farrowing seasons of the fall of 1955 and the spring of 1956. The term "newborn" as used in this thesis covers the pig from birth to the tenth postnatal day. During this span of life hypertrophic pyloric canal stenosis has been observed in the College herd. This anomalous condition of the pyloric canal was discovered during an autopsy survey made for the purpose of supplementing existing meager data pertaining to the conditions bringing about baby pig losses in the college herd. It is noteworthy that pigs afflicted with hypertrophic pyloric

canal stenosis were isolated from those clinically spoken of as having "baby pig disease" (hypoglycemia).

Because data pertaining to the normal gross and microscopic anatomy of the pyloric canal in the newborn pig could not be found in the literature and because such data are imperative for the understanding of pathological alterations, the major anatomical features of the pyloric canal at certain developmental stages are illustrated and described. Thirty-five plates comprising sixty figures, some in color, supplement the text. Many legends carry a "comment" in which corroborative data pertaining to the subject illustrated are presented.

The terminology employed for the designation of normal and abnormal anatomy has been carefully scrutinized. In the main the terminology follows closely that used by mammalian and human anatomists and pathologists. The terms "gastric sphincter of pyloric canal" and "concave curvature of pyloric canal," however, had to be coined to cover structures heretofore not described. The major gross divisions of the stomach of the newborn pig presented in Figure 1 compare well with those worked out for the human stomach by Forssell (1913), Bauer (1923), Torgersen (1942), Alvarez (1948), and Barborka and Texter (1955).

We are cognizant of the fact that some of the normal and abnormal anatomy of the pyloric canal presented in this thesis may fall short of correctness when "standards of comparison" other than our own become available. In some instances we were handicapped by unpreparedness to decide conclusively whether a certain phenomenon was an actual condition or was caused by agonal or fixing stresses. This predicament is not at all new to those working in the field of dynamic biology. We are of the opinion that the over-all data constitute a foundation upon which further

research can be built.

Hypertrophic pyloric canal stenosis in the newborn pig is in many respects analogous to a condition known as "congenital infantile hypertrophic pyloric canal stenosis" in the human. The major similarities of these two gastric lesions are briefly mentioned in the section titled "Discussion and Summary." Without further qualifying the generalities stated in this paragraph, one may logically postulate that discovery of the underlying etiology of pyloric canal stenosis in the pig may resolve the obscureness of the pathogenesis of infantile hypertrophic pyloric canal stenosis. Should this hypothesis become a reality, the ultimate purpose of this thesis would be fulfilled.

I wish to express my sincere thanks to the members of my committee for the approval of the problem and sustained interest in my progress.

The investigation presented in this thesis has been supported in part by the Oklahoma Agricultural Experiment Station.

Thanks are extended to Dr. C. K. Whitehair, formerly of the Department of Veterinary Research and Animal Husbandry, for my appointment to research assistant in the Department of Physiology and Pharmacology.

I am deeply indebted to Dr. J. C. Hillier, Department of Animal Husbandry. It would have been difficult for me to achieve my aims had I not enjoyed his genuine interest and enthusiasm in my problem. It was on his suggestion that an autopsy survey of the college swine herd was made. His appreciation of the merit of the baby pig as a research tool in the field of comparative anatomy, physiology, and pathology has contributed much toward making the field work enjoyable.

The untimely death of Dr. H. W. Orr, Dean of the School of Veterinary Medicine, has deprived me of a good friend and teacher. His willingness

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The literature review was capably assisted by Dr. M. Grundmark, Stockholm, Sweden, Mr. A. P. Juhlin, Special Service Department, and Mr. C. M. Howland, Biological Science area, both of the Oklahoma Agricultural and Mechanical College Library. I wish to thank Miss E. G. Peebles for the many courtesies extended to me during my association with the School of Veterinary Medicine and for typing the final draft of this thesis. Thanks are expressed to Mrs. D. Mueller for the efficient handling of the preliminary typing work.

STATEMENT OF THE PROBLEM

It has been assumed in the past that the heavy loss of baby pigs in the Oklahoma Agricultural and Mechanical College herd was caused by baby pig disease (baby pig hypoglycemia). This supposition, however, was based on superficial rather than concrete clinical and autopsy data. It is generally inferred that baby pig disease is responsible for the loss of about 30 percent of all pigs farrowed each year (Wilcox, Carroll, and Hornung, 1933). Because of rising baby pig losses in the College herd an autopsy survey of all pigs eliminated from the herd irrespective of the reason was believed a worthwhile project. Such a survey was made during the fall of 1955 and the spring of 1956 farrowing seasons. During these two seasons 1,040 (536 males and 504 females) pigs were farrowed. Out of this total one hundred and twelve pigs were autopsied. Among them were seventeen pigs believed afflicted with baby pig disease. Only seven of them, however, presented the autopsy findings compatible with this condition. The remaining ten cases had "hypertrophic pyloric canal stenosis." Although hypertrophic pyloric canal stenosis was the cause or a contributory cause of the death of 1.0 percent of all pigs farrowed during the fall of 1955 and the spring of 1956 at Oklahoma Agricultural and Mechanical College, the disease is not an economic problem in swine production as long as the mortality remains at the 1.0 percent level. Should the malady, however, be a genetotrophic disease, the possibility must be kept in mind that under certain conditions the gastric lesion may cause loss of baby pigs of economic importance. The literature therefore, was

searched for data relevant to the occurrence of hypertrophic pyloric canal stenosis in domestic animals. With the exception of the dog no data were found pertaining to the subject under investigation. The literature of human medicine was then scrutinized. There it was found that hypertrophic pyloric stenosis constitutes not only a well documented clinical entity but also is the most common gastric lesion in infancy.

In summary, the autopsy survey revealed that baby pig disease was a minor cause of pig losses during the farrowing seasons studied. The survey brought to light a fatal gastric lesion producing symptoms characteristic of baby pig disease. It can be readily understood that superficially examined pigs afflicted with hypertrophic pyloric canal stenosis may be placed in the category of baby pig disease and thus falsely raise the percentage loss from the latter condition. In order that this additive error is not made in the future by those charged with ascertaining the cause of baby pig losses, our investigational data relevant to hypertrophic pyloric canal stenosis in the newborn pig are presented here. Further, lack of descriptive and pictorial data pertaining to the anatomy and meager references to the physiology of the stomach of the newborn pig suggested to us to make a limited study of the subjects just cited to obtain some idea of the "norm." This study revealed a striking analogy of the anatomy, physiology, and pathology of the newborn pig's stomach to that of the newborn human. On the basis of these analogies the hypothesis was formulated that discovery of the pathogenesis of hypertrophic pyloric canal stenosis in the newborn pig may not only reduce if not eliminate baby pig losses from this disease but also may resolve the obscureness concerning the pathogenesis underlying the malady in the human and in the dog. Toward this goal our future research is directed.

LIMITATION OF THE STUDY

This thesis is limited to the study of the readily demonstrable gross and microscopic anatomy and physiology of the stomach of normal newborn pigs and pigs with hypertrophic pyloric canal stenosis.

CLARIFICATION OF TERMS

Technical terms are defined whenever deemed necessary for a novitiate to get a better understanding of the pictorial record, anatomical planes, and dissecting procedures.

The term "newborn" as used in this thesis includes pigs from birth to the tenth postnatal day. For structures that can be seen with the unaided eye the term "gross anatomy" is applied, whereas for the finer detail the term "microscopic anatomy" is used. The terms "proximal" and "distal" express the distance, position, and direction of parts or an organ from the long axis of the body, and the terms "external" and "internal" indicate relations of depth in a cavity or organ. The term "pyloric canal" is applied to the most distal part of the pyloric vestibule through which chyme passes into the small intestine. The term "gastric sphincter" is used to designate the gastric orifice of the pyloric canal, and the term "concave curvature" identifies an incisura between the gastric and the duodenal sphincter of the pyloric canal. The term "duodenal sphincter" is synonymous with the term "pyloric sphincter." The latter term is applied to the duodenal orifice of the pyloric canal of the stomach of the adult pig.

II. REVIEW OF THE LITERATURE

Baby Pig Disease (baby pig hypoglycemia)

To allow the novitiate a clear understanding of the subject "hypertrophic pyloric canal stenosis" it seems appropriate to present a brief review of baby pig disease (baby pig hypoglycemia) from which the fatal gastric lesion was isolated.

Graham, Sampson, and Hester (1941) described a highly fatal malady in the newborn pig to which they gave the non-specific name "baby pig disease." Pigs afflicted with this disease shiver, lose their appetite and their weight, develop a rough hair coat, and exhibit a clammy skin. Hypoglycemia, coma, and death are the ultimate sequel occurring generally between twenty-four to thirty-six hours after the onset of the first symptoms. According to these workers 30 percent of all pigs farrowed are lost from the disease, and losses of from 75 to 100 percent may occur in some litters. The malady affects large and small litters and all breeds of swine. Sampson, Hester, and Graham (1942) noted that the disease occurred more frequently in early and late spring. They, however, offered no explanation for this preferential phenomenon. Graham, Sampson, and Hester (1941), and Sampson, Hester, and Graham (1942) ruled out hog cholera, influenza, erysipelas, and enteric disorders as a possible etiologic agent. They also reported absence of diagnostic gross pathology at autopsy. Their observation of a large cohesive milk mass in the stomach of some of the baby pigs is noteworthy since they interpreted

the phenomenon as presumptive evidence of gastrointestinal stasis. An empty stomach, more commonly found in field cases, was presumed by Graham and his associates to be the result of failure of the baby pig to suckle at all or to suckle only a little between the time of birth and death.

Young and Underdahl (1947) postulated that baby pig disease is the sequel of an exposure of pregnant sows to other diseased swine. They advanced the provocative hypothesis that baby pig disease (hypoglycemia) results from a "reverse anaphylactic shock," which occurs at the first feeding of the milk of the dam.

This concept requires antigens (viruses, proteins, or antigenic fractions of smaller size) to pass the placenta sensitizing the pig in utero. While the sow produces protective antibodies, they can not pass the placental barrier and thus can not neutralize antigens in the unborn animal. Ingestion of colostrum, which contains concentrated antibodies, adds an additional stress which can be fatal to the newborn pig. Young and Underdahl believe that the degree of shock is dependent upon the degree of intra-uterine sensitization, the amount of ingested colostrum, and the concentration of the antigen, in the latter. Their hypothesis appears to account for the loss of only one, several, or an entire litter of newborn pigs as well as elucidates the phenomenon of a sudden refusal of a newly born litter to suckle the sow after the first ingestion of colostrum. When transferred to another sow, the baby pigs will eagerly suckle the foster mother. This suckling response suggests incongruity of the mother's colostrum and compatibility of the milk of the foster mother.

Sampson (1950) reviewed the literature on spontaneous and experimental hypoglycemia in newborn pigs. He states that this condition in baby pigs results from one of three causes: "(1) failure of the sow to secrete

enough milk or (2) failure of the pigs to ingest enough milk because of inappetence associated with some form of digestive disturbance, such as scours, or (3) failure of the curd to pass from the stomach into the intestine for further digestion and absorption." He remarks that when no milk or other nutrients are ingested following birth, hypoglycemia develops generally within 24 to 48 hours. Sampson, Hester, and Graham (1942) state that fatal hypoglycemia will not develop in four-or five-day-old pigs until after 150 hours of fasting. They explain the difference of the onset of hypoglycemia in newborn and older pigs by the fact that a newborn pig requires several days before it can produce liver glycogen from non-carbohydrates, whereas gluconeogenesis is an effective source of liver glycogen and blood sugar in older pigs. For the alleviation of the hypoglycemic state these investigators recommend the administration of 3 to 5 ml. of a 5 percent glucose solution intraperitoneally.

Heidebrecht et al. (1950) investigated the possibility of a toxic factor or nutrient deficiency in the milk of sows nursing baby pigs afflicted with baby pig hypoglycemia. They found no evidence of a toxic factor or deficiency of a nutrient. The first symptoms in their animals were vomiting and diarrhea following ingestion of the sow's milk. Weakness, emaciation, dehydration, and a rough hair coat developed within a few hours even though the baby pigs suckled until they became too weak to stand. No blood sugar levels were given. Their observation of a yellowish milk curd in the stomach, gastroenteritis, and congested mesentary blood vessels is more in accord with "transmissible gastroenteritis" described by Doyle and Hutchings (1946) than with baby pig disease (baby pig hypoglycemia) reported by Graham, Sampson, and Hester (1941) and Sampson, Hester, and Graham (1942).

Morrill (1952 a) fasted and exposed newborn pigs to an environmental temperature ranging from 57.0° to 61.0° F. (13.8° to 16.0° C.) and 82° to 93° F. (27.7° to 33.8° C), respectively. He noted that fasted pigs kept in a cold environment developed a critical hypoglycemia sooner than those kept in a warm environment. Morrill advanced the opinion that one important effect of chilling may be a rapid depletion of available carbohydrates, whereas higher temperatures conserve carbohydrates through reduction in rate of heat loss. Morrill (1952 b) determined the blood sugar level of newborn pigs and found the sugar concentration to range from 49.72 to 158.20 mg. percent with an average of 102.6 mg. percent. In the moribund hypoglycemia-fasted pig he found the blood sugar level from 2.26 to 47.46 mg. percent with an average of 15.6 mg. percent. The comment of Morrill (1952 c) is noteworthy that it may be fruitful to study the relation of the gastric emptying time and composition of sow's milk because of the presence of milk curd in the stomach of some field cases which suggests failure of normal gastric and intestinal motility.

Newland, McMillen, and Reineke (1952) studied the temperature adaptation in baby pigs. They observed that the mechanism regulating the body temperature in the newborn pig is not fully developed. These workers show that the body temperature may drop from 3 to 13° F. during the first thirty minutes after birth. The temperature returns to normal, however, in about two days. They stress that the newborn pig is very sensitive to cold environment and chilling.

Kemény <u>et al</u>. (1955) fasted newborn pigs after allowing them to suckle from 15 to 96 hours. The animals were then exposed to an environmental temperature ranging from 14° C. (57.2° F.) to 24° and 28° C. (75.2° and 82.4° F.). The blood sugar level dropped in the majority of

pigs. Several pigs, however, did not become comatose. It is interesting to note that a few animals died when the blood sugar level was still high. These workers question that hypoglycemia is the true and only cause of the baby pig's death.

In summary, baby pig disease (baby pig hypoglycemia) may be defined as a disturbance of the carbohydrate metabolism seemingly initiated by chilling and/or anorexia. The disease affects pigs during the first few days of life, the mortality peak being between the third and fourth postnatal day. It affects all breeds and both sexes and occurs in large and small litters. An entire litter may succumb, or only one pig in a litter may have the disease. With the exception of hypoglycemia the disease has no specific symptomatology which allows its separation from other clinical entities affecting carbohydrate metabolism. Autopsy findings aid to some extent by establishing absence of gross pathology. Microscopic pathology of the liver, kidneys, adrenals, and other organs has been noted, but the changes are not specific. No pathogenic organisms or toxins have been identified as causally related to the disease. Thus, in the final analysis the pathogenesis of baby pig disease awaits further studies.

Hypertrophic Pyloric Canal Stenosis in Domestic Animals

Review of the literature revealed occurrence of pyloric stenosis in the dog. Kirk (1953) states that in three dogs the clinical symptoms were persistent projectile vomiting. This vomiting may occur some hours after ingestion of food. Vomiting may occur, however, when no meal has been consumed. Milk is vomited even when pepsinated. In the radiograph the stomach has a balloon-like shape caused by the obstructed pyloric sphincter. Little to none of ingested barium escapes into the intestine.

In the normal dog the barium enters the intestine in about five minutes after ingestion. Kirk found the pyloric canal tightly constricted at autopsy. There were no gross lesions that accounted for the stenotic condition of the pyloric canal. Kirk recommends methyl atropine nitrate (Eumydrin 1:10,000 solution) 0.25 to 1.5 ml. to be given about a half hour before the meal.

Vine (1954) reported the occurrence of pyloric canal stenosis in a young dog. He alleviated the stenosis of the pyloric canal by surgical intervention.

The observation by Bachmann (1956) of pyloric stenosis in a ten-year-old dog is noteworthy because of the absence of a visible causal lesion.

Hypertrophic Pyloric Canal Stenosis in Man

Because of the striking clinical and anatomical similarities between hypertrophic pyloric canal stenosis in the newborn pig and the condition known as congenital infantile hypertrophic pyloric stenosis, in the human the literature pertaining to the latter disease was reviewed.

According to Mack (1942) the first case of hypertrophic pyloric stenosis in the human was reported by Fabricious Hildanus in 1627. Armstrong in 1777 focused attention upon the familial tendency of the malady (Euster-man and Balfour, 1935). DeBuys (1913) states that pyloric stenosis is the sequel of either hypertrophic pyloric stenosis, a permanent organic obstruction resulting from hypertrophy of the circular muscle layer subject to surgical intervention, or pylorospasm which is a functional obstruction amiable to dietary and/or drug therapy. Wallgren (1941) found that boys are more commonly afflicted with hypertrophic pyloric stenosis

than girls. He gives the incidence for boys as 1:154, or 0.65 percent, among 13,217 and for girls 1:770, or 0.13 percent, among 12,425. Ford, Ross, and Brown (1941) studied the birth rank incidence of 278 infants with hypertrophic pyloric stenosis. They report a predilection of the disease for the first-born. The first-born accounted for 51.8 percent, the second-born for 21.9 percent, the third-born 14.7 percent, and the fourth-born and over 5.8 percent of their series. Ford and his associates offered no explanation for the birth rank phenomenon. McKeown, MacMahon, and Record (1951) found the incidence of birth rank per 1000 cases of pyloric stenosis to be for the first-born 4.2, for the second-born 2.8, and for the succeeding siblings 1.4. Ford, Brown, and McCrary (1941) state that in the case of uniovular twins both are affected but in the case of binovular twins only one is affected by the disease. They postulate that an environmental handicap imposed before birth on members of multiple sets is a factor inducing the condition of pyloric stenosis. Sheldon (1938) presented surgical evidence of the presence of hypertrophic pyloric stenosis in only one identical twin. DeBuys (1913) postulated that pyloric stenosis may cause obstruction from a degree which is fatal to a slight hyperplasia which is compatible with life. Nelson (1950) remarks that "failure to demonstrate constriction of the lumen of the pyloric canal immediately after birth does not eliminate the possibility that the underlying factor is present at this time." He states that pyloric stenosis has been observed as early as the second postnatal day. Ladd, Ware, and Pickett (1946) advocate a hereditary background on the basis that pyloric stenosis has been noted in successive generations, in an infant seven months old (premature) and in a full-term stillborn infant. Wallgren (1946) examined roentgenologically 1000 newborn boys.

Five of them developed pyloric canal stenosis. The radiographs of the stomach of these boys showed a patent pyloric canal at the time of the first examination. McKeown, MacMahon, and Record (1952) suggest that a "recessive gene with low manifestations" may be involved in the production of pyloric stenosis. Nelson (1950) points out that clinically congenital infantile hypertrophic pyloric stenosis is characterized by projectile vomiting between the second and third week of postnatal life, loss of weight, constipation, hemoconcentration, and possible alkalosis. According to Nelson, the gastric lesion occurs in both breast-fed and artificially nourished infants. From the anatomical point of view hypertrophic pyloric stenosis is defined by Eusterman and Balfour (1935) as "enlargement or overgrowth of the pyloric muscle, without apparent causative or associated lesion." Niero (1950) states that "hypertrophy and disorderly arranged longitudinal muscle fibers in the duodenum are important factors in the pathogenesis of pyloric stenosis." Meuwissen and Slooff (1934) measured the length of the pyloric canal in newborn infants. They found that normally the canal averaged from 1 to 2 mm. in length. Obstruction occurs when the canal exceeds 6 mm. in length. Miller and Ostrum (1945) state that with complete or incomplete pyloric stenosis the stomach is unually large and atonic and that the intestine proximal to the point of obstruction is dilated. Buckstein (1948) believes that heaped-up mucosal folds in the canal, gastritis, inflammatory hyperplasia involving the mucosa, and hypertrophy of the muscular layer are other factors capable of causing various degrees of pyloric stenosis. As to the medical alleviation of hypertrophic pyloric stenosis, Sauer (1921) advocates administration of thick cereal, and Sadove et al. (1954) suggests the use of procaine amide. The preferred surgical measure is that employed

by Ramstedt (1912). The procedure calls for the separation of the tunica muscularis from the mucosa. Bell (1947), Coffey (1950), and Potter (1952) relate that congenital hypertrophic pyloric stenosis is the most common gastric lesion found in infancy. It is generally assumed that the lesion is present before birth and that the hypertrophy of the muscle fibers is the end stage of a pylorospasm (strain hypertrophy). Potter (1952) points out that pylorospasm and mild forms of hypertrophic pyloric stenosis respond to atropine or barbiturates. These drugs tend to relax the pyloric sphincter, and thus chyme may pass from the pylorus into the duodenum. When however, stenosis is caused by an extensive hypertrophy of the muscle fibers and hyperplasia of the connective tissue, the aforementioned drugs are ineffective, and the obstruction can be alleviated only by surgical intervention.

Nelson (1954) reviewed the case history of 95 adults who had pyloric stenosis in infancy. He found that 28 percent had either gastritis or a peptic ulcer, whereas in the control group only 12 percent were afflicted with these gastric lesions.

Schnohr (1932) noted a pronounced hypochloremia in both the blood and tissues in human cases with a severe degree of hypertrophic pyloric stenosis. He is of the opinion that hypochloremia may be used as a diagnostic criterion since the symptom is more severe than in any other gastrointestinal obstruction associated with vomiting.

Ladd, Ware, and Pickett (1946) found not only a low sodium chloride level but also a subnormal ascorbic acid (vitamin C) concentration in individuals with a severe form of pyloric stenosis.

From the review of the literature we are inclined to agree with Mack (1942) that the complete history of congenital infantile hypertrophic pyloric stenosis has yet to be written.

[1] Because no English translation of Martin's important reference work is available, it seemed worthwhile to cite verbatim the paragraphs quoted in this thesis in the original language, p. 57.

Der Sphincter pylori ist kein gleichmässig ausgebildeter Ring, denn er besitzt eine mondsichelförmige Verdickung, welche gegenüber dem in die Pyloruslichtung hereinragenden, schleimhautüberzogenen, 1 cm. hohen, 2 cm. breiten und 3.5 cm. langen Pyloruswulste, Torus pylori gelegen ist. Dieser besteht aus der Schleimhaut, radiär eindringenden Muskelfaserzügen und Fettgewebe. Der Wulst bilded einen longgestreckten, am konkaven Bogen angehefteten Zapfen, welcher mit der Sphincterverdickung zusammen den Pförtner fest abschliesen kann. Seine Länge beträgt ungefähr 3.5, seine Breite 1.5-2 cm. bei ungefähr 1-1.5 cm. Höhe."

Gross Anatomy of the Stomach of the Adult Pig

Marshall and Halnan (1953) state that the stomach of the pig represents a transition stage between the simple stomach of the dog and the complex stomach of ruminants. Sisson and Grossman (1953) describe the pig stomach as being a one compartment or simple stomach. The organ has several distinct coats, which from without inward are: longitudinal, circular, and oblique muscle layers, submucosa, muscularis mucosae, lamina propria, gastric glands, and surface epithelium. Grossly the stomach is divided into four major regions: (a) the cardiac gland region, in which is located a conical blind pouch, the diverticulum ventriculi, (b) the oesophageal region, in which is located the distal end of the oesophagus (esophagus), (c) the fundus gland region, and (d) the pyloric region, which incorporates the pyloric canal. In the latter a prominence (torus pyloricus) projects from the wall of the lesser curvature. Sisson and Grossman state that this structure diminishes the size of the pyloric canal orifice considerably. They give the dimensions of the torus as about one inch and a half (3 to 4 cm.) long and half an inch (1 cm.) high. It consists largely of fat, but fibers from the muscular coat extend into it (Figure 31). Martin (1923) states

> ". . . the pyloric sphincter is not a uniformly developed circle. It has a crescent-like thickening which opposes the mucosa-covered 1 cm. high, 2 cm. thick, and 3.5 cm. long torus pyloricus, which protrudes into the pyloric space. The torus pyloricus consists of a mucous membrane, radially penetrating muscle fibers, and adipose tissue. The swelling forms a longitudinal cone attached to the concave side, which together with the thickened sphincter can tightly close the pylorus. The length of the latter is about 3.5, the width 1.5 to 2.0 cm. and is between 1.0 to 1.5 cm. high."[1]

Torgersen (1942) noted absence of the longitudinal musculature on the lesser curvature of the stomach and remarks that in the pyloric canal actually one may see two sphincters which delimit an area in which the circular

musculature has a special structure, that is, a muscular knot (torus pyloricus) on the lesser curvature. Circular musculature loops are continuous in this muscle torus. The muscular sphincter is on the greater curvature. According to Torgersen the pylorus has arisen from two parts of the intestinal tract, of different origin, the parts united at the point known as pyloric canal (sphincter). This fusion of the gastric and the duodenal part accounts for the marked independence of the gastric (oral) and the duodenal sphincter. Torgersen advocates that the pyloric sphincter is as much a part of the duodenal bulb as of the pylorus.

Gross Anatomy of the Stomach of the Newborn Pig

Review of the literature failed to reveal descriptive or illustrative material pertaining to the gross anatomy of the newborn pig stomach, No plausible explanation was found for this lack of anatomical data.

Gross Anatomy of the Stomach of the Newborn Infant

Scammon (1923) states that the musculature of the stomach "aside from that of the pyloric canal and sphincter is only moderately developed at the time of birth." He points out that the longitudinal layer is incomplete over a part of the greater curvature. According to him both layers of the muscularis mucosae are present but the elastic tissue is poorly developed. Scammon found the mucous membrane to be thicker in the newborn than in the child.

Pisek and LeWald (1913) state "there is no definite normal type of stomach in the infant." Anson (1950) remarks that perennially copied stereotyped figures of the stomach are of little value when confronted

with natural aberrancies. His illustrations of variations of the shape of the human stomach in his Atlas of Human Anatomy, (p. 287), are to the point.

Comparative Physiology of the Stomach of the Newborn Pig and Infant

Gastric Emptying Time

The literature does not contain data pertaining to the emptying time of the stomach of the newborn pig. The radiological observation of Neimeier (1939) suggests that the stomach of the adult pig empties itself slowly. He obtained evidence of the presence of food in the stomach after a day's fast.

Ladd (1913) with the aid of radiological methods studied the gastric motility of infants one to sixteen and a half months old. He found that in the normal infant the stomach empties itself in from one a half to two and a half hours. Ladd made radiographs at half-hour intervals after ingestion of five ounces of radio-opaque media. He made another interesting observation, namely, that three hours were required by the stomach to empty itself of artificially fed milk in contrast to breast-fed milk, which was out of the stomach in two hours.

Volume of Gastric Juice

No data on the volume of gastric juice of newborn pigs has been found in the literature.

Gilde (1937) noted a great variation of gastric juice secreted by the adult pig. According to him the volume ranges from 25 to 300 ml. per hour.

Smith (1945) gives the volume secreted by the newborn infant as averaging 4 to 5 ml. during the first few hours of life.

Free Hydrochloric Acid

Kvasnitskii and Bakeeva (1940) studied the gastric secretion in the newborn pig with the aid of a gastric fistula. They found no free hydrochloric acid in the gastric juice, though hydrochloric acid was secreted between the twentieth and thirtieth postnatal day.

Smith (1945) found that hydrochloric acid may be demonstrated in the stomach of the human fetus between the fourth and the fifth month. Hess (1913) noted presence of free hydrochloric acid in the gastric juice of unfed infants a few hours old.

pH of Gastric Juice

No data pertaining to the pH of the gastric juice of the adult and newborn pig were found in the literature.

Smith (1945) gives the pH range of the gastric juice of the newborn infant between 1.3 and 4.6.

Pepsin and Rennin

Kvasnitskii and Bakeeva (1940) obtained evidence of the presence of pepsin and rennin in the gastric juice of the newborn pig.

Smith (1945) states that pepsin and rennin are present in the gastric secretion of the four-to five-months-old human fetus. Hess (1913) demonstrated the presence of pepsin and rennin in the gastric juice of the infant immediately after birth.

Physical Character of Milk Curd

No data were found pertaining to the physical character of milk curd in the stomach of newborn pigs.

Hess (1914) states that "Pyloric casts of milk aspirated from the duodenum of infants have a diameter of 1 to 2 mm. and denote the normal diameter of the pyloric canal during the passage of milk from the stomach into the duodenum."

Blood Sugar Level of the Newborn Pig

Morrill (1952 b) determined the blood sugar level of twenty-six newborn pigs. He found the blood sugar concentration to range from 49.72 to 158.20 with an average of 102.6 mg. per 100 ml. of blood.

Liver Glycogen of the Newborn Pig

Morrill (1952 b) estimated the liver glycogen concentration of fourteen newborn pigs. He found the glycogen to range from 1.85 to 9.00 percent with an average of 5.2 percent on a fresh basis.

III. DISTRIBUTION OF GROSS PATHOLOGY IN SEVENTY-SIX PIGS ELIMINATED FOR VARIOUS REASONS FROM THE COLLEGE HERD DURING THE FARROWING SEASON OF SPRING 1956

Pig losses before weaning are generally said to be between 30 and 35 percent of all pigs farrowed each year. The first toll taken from a litter consists of pigs born dead according to Carroll and Krider (1950). Wilcox, Carroll, and Hornung (1933) found the causes of death to be (a) crushing by the sow (44 percent) and (b) miscellaneous and unknown causes (18 percent). The latter two categories are of questionable value to the swine producer, for neither aids the swine industry to reduce pig losses. While it is admitted that the exact cause of death can not be ascertained in every instance, we are of the opinion that the patho-anatomical data presented in Table 1 should give the novitiate some idea of the reasons of pig losses. It is noteworthy that in spite of the great variety of gross pathology found in the college herd the pig loss amounted to only 14 percent of the 539 pigs farrowed during the spring of 1956.

In summary, one can truthfully say that the autopsy records revealed that baby pig disease was not a major cause of pig losses in the College herd as previously assumed but rather a minor cause during the farrowing season of the fall of 1955 and the spring of 1956. The data also point up the value of a systematic autopsy survey of all pigs eliminated from a herd irrespective of the reason. Adherence to this policy led

to the discovery of "hypertrophic pyloric canal stenosis" as an anomalous condition of the stomach heretofore not reported as an entity or cause of death of newborn pigs. Preliminary studies of this gastric lesion suggested a parallelism to a condition known in man as "congenital infantile hypertrophic pyloric stenosis." Because of this discovery a detailed though limited investigation of hypertrophic pyloric canal stenosis in the newborn pig was believed to constitute a worthwhile problem.

TABLE 1

Distribution of Gross Pathology in Seventy-six Pigs Eliminated for Various Reasons from the College Herd During the Farrowing Season of Spring 1956*

Gross Patho-Anatomical Diagnosis	Number of Pigs	Gross Patho-Anatomical Diagnosis	Number of Pigs
Stillborn	7	Hemopericardium (needle puncture)	1
Premature	7	Sero-fibrinous pericarditis	1
Runt	8	Gastric ulcer (pylorus)	4
Crushed by sow	3	Perforated stomach (wood splinter)	1
Baby pig disease (hypoglycemia)	3	Hemorrhagic gastritis	1
Anemia (bleeding from navel)	3	Peritoneal abscess and peritonitis	1
Anopthalmia	1	Gut edema	2
Agnasis renalis (left)	1	Strangulation gangrene of ileum	1
Atresia ani	1	Necrotic enteritis	1
Double common bile duct leading into pylorus	1	Constrictive enteritis	1
Umbilical hernia	1	Rectal abscess	1
Scrotal hernia	1	Liver abcess	6
Congenital megacolon	1	Ruptured gall bladder	1
Congenital hepatomegaly	1	Fatty liver	8
Hemangiomata of liver	1	Paralysis of hind legs (compression fracture)	1
Bilateral renal cystic disease	1	Fracture of femur	1
Hypertrophic pyloric canal stenosis	6	Fracture of skull	1
Cholera (vaccination break)	1	Uric acid infarcts (kidney)	2
Influenza	22	Swollen knee joints (infected deep skin ulcer)	2
Bronchopneumonia	23	Pad ulcer of sole	1
Hydrothorax	13	Suffocation (stone in throat)	1
Pulmonary edema	7	Pitch poisoning	1
Pleural adhesions	5	Parakeratosis	1
Sero-fibrinous pleuritis	1	Eperythrozoonosis	1
Pulmonary amniotic fluid embolism	1		
Pulmonary zinc crystal embolism from drenching	1		

*The number autopsied (seventy-six) represents approximately 14 percent of the 539 pigs farrowed.

IV. INVESTIGATIONAL PROCEDURE AND RESULTS

Fixation of the Stomach in Situ during Life

Unavailability of data pertaining to the gross and the microscopic anatomy of the stomach of the newborn pig forced us to spend considerable time designing and experimenting with methods for determining the general contour of the stomach and preventing at the same time artifacts in the musculature and mucosa. We were particularly handicapped by the lack of a "standard of comparison" upon which our results could be evaluated in terms of "norm." Thus it became our first concern to establish a "normal contour" of the stomach of newborn pigs. Formol-saline fixing fluid (formaldehyde 10 ml., physiological saline 90 ml.) was introduced into the stomach with the aid of a small polyethylene tubing immediately after death. While the method gave some idea of the contour, over dilatation frequently occurred and caused the obliteration of the mucosal pattern. The method finally employed for the demonstration of the external and the internal gross anatomy of the stomach of newborn pigs was one worked out by the writer. The method referred to is a "spray fixation technique" and is given below.

The baby pig was placed in a restrainer in such a manner that the abdominal area presented itself for a midline incision. The animal was given 0.5 ml. of sodium pentobarbital (1 ml. = 1 grain) intraperitoneally. When the animal was relaxed, the skin was infiltrated with a 1 percent procaine solution along the midline of the abdomen. A median

incision was made with a Bard-Parker knife blade, and all bleeding points were checked with a hemostat. The peritoneum was exposed by blunt dissection. When the light-colored stomach wall was plainly visible below the liver edge, the peritoneum was incised just enough to allow manipulation of the stomach. The left gastro-epiploic artery was brought into view, and the stomach was incised about 1 centimeter medial to the artery. The incision was made just large enough to permit introduction of the nozzle of a DeVilbiss Atomizer to which the stomach wall was secured. A clinical thermometer was introduced into the abdominal cavity to ascertain the internal temperature. About 50 ml. of formol-saline fixing solution were warmed to the thermal state of the abdomen, and 10 ml. of the fluid were injected slowly into the abdominal cavity. The fixing fluid was then sprayed into the stomach in amounts of 1 to 2 ml. at intervals of several minutes until the animal stopped breathing. The dual fixation minimized (a) peristalses of the stomach musculature creating a better chance of obtaining reliable data pertaining to the contour, and (b) the mist of the fixing fluid introduced into the stomach produced rapid and uniform fixation of the mucosa. About half an hour after the initial fixation was completed, the abdomen was opened, and the esophagus and the most distal part of the duodenum were tied off and severed near the tie. The stomach was then dissected out and submerged in a formol-saline fixing fluid for twenty-four hours at room temperature. The stomach was then opened with a sharp pair of scissors along the greater curvature about 2 mm. above the gastro-epiploic vessels. The incision was extended from the incisura angularis of the lesser curvature to the beginning of the pylorus on the greater curvature. The visceral part of the longitudinal stomach was fastened to a board with

dissecting needles in such a manner that the duodenum pointed to the left. The free longitudinal part of the stomach was lifted up. With a sharp brain knife the pylorus was cut horizontally and slightly above the midline. When this cut is properly executed, the pyloric canal is laid open just above the torus pyloricus. A representative example of the result obtained with the simultaneous external and internal fixation method of the stomach in situ during life is shown in Figure 1.

Gross Anatomy of the Stomach of the Normal Newborn Pig

Although the contour of the stomach shown in Fig. 1 is "ovoid" we hasten to say that this is only one of the many shapes of the stomach of newborn pigs encountered in the course of our investigation. The great variation of form of the organ nullified any attempt to establish a normal shape of the stomach of newborn pigs. We are thus in full agreement with Anson (1950) that the natural aberrancies of the contour of the stomach do not allow assigning a specific form to the organ. We are also inclined to underline the statement of Huntington (1903) that the size and the shape of the stomach are determined by the anatomy of the abdominal cavity, an observation that we were able to confirm by topographical studies carried out on our series of newborn pigs.

Pylorus

Applying the four major gross anatomical segments of the simple human stomach, namely, longitudinal stomach, transverse stomach, greater curvature, and lesser curvature to the stomach of the newborn pig, we find that the diverticulum ventriculi, esophagus, and incisura angularis are in the longitudinal stomach, whereas the sphincter antri, pylorus,

and pyloric canal are in the transverse stomach (Fig. 1, 18). Since the relationships of the pylorus to the pyloric canal is our immediate concern these structures will be described in detail. The pylorus begins at the incisura angularis and at the first portion of the ascending transverse stomach on the greater curvature (Fig. 1, 18). Proximally the pylorus is limited by the sphincter antri and distally by the pyloric canal. Between these boundaries is the pyloric vestibule (antrum). The sphincter antri is a ring-like muscular structure located at the distal portion of the longitudinal stomach. Distal to the sphincter antri is the pyloric vestibule (antrum), which is in the ascending transverse stomach (Fig. 2, 18). A semicircular mucosal fold is at the base of the pyloric vestibule (Fig. 3). The pyloric canal begins at the semicircular fold and terminates at the proximal part of the duodenum (Fig. 2). The pyloric canal consists of seven distinct anatomical structures: (a) greater and lesser curvature musculature, (b) pyloric mucosa (mucous membrane), (c) torus pyloricus longitudinalis, (d) gastric sphincter, (e) duodenal sphincter, (f) concave curvature, and (g) longitudinal mucosal folds (plicae mucosae).

Pyloric Canal

In Fig. 2 is shown a longitudinal section of the pylorus and pyloric canal of the stomach of a pig ten days of age. The musculature of the greater and lesser curvature (letter i', i'' in Fig. 2) consists of muscle coats. The three muscle coats comprising the muscular wall of the pyloric canal are the outer longitudinal, middle circular, and the inner oblique. The latter is grossly the least distinguishable of the muscle layers. As to the size relationship of the muscle coats, the middle

circular muscle layer is about twice the thickness of the outer longitudinal muscle coat (Fig. 24). The latter does not surround the lesser curvature (Torgersen, 1942) and may have hiatuses on the greater curvature (Fig. 23 d). The mucosa (letter d in Fig. 2) is fairly thick and thrown into distinct folds (plicae mucosae). The longitudinal mucosal folds (letter c in Fig. 2) may run through the entire length of the pyloric canal or terminate above the duodenal sphincter (Figs. 4, 5, 6, 7). The gastric sphincter (letter e in Fig. 2) is a thickening of the musculature, a sort of compressed reserve muscle tissue. This muscular mass is a transient structure being stretched to a point of complete disappearance when the growth of the stomach becomes accelerated to accommodate increased food intake (Fig. 17). The gastric sphincter is semicircular and opposes the torus pyloricus longitudinalis (letter h in Fig. 2). The sphincter may be considered as the gastric orifice of the pyloric canal and the beginning of the latter. The gastric sphincter longitudinally sectioned has a somewhat cone-like appearance. The distal end of the sphincter tapers off into a distinct indentation of the greater curvature musculature "concave curvature" (letter f in Fig. 2). This narrow portion of the pyloric canal musculature demarcates the distal end of the gastric sphincter and the beginning of the proximal portion of the duodenal sphincter (letter g in Fig. 2). The latter is a thickening of the pyloric canal musculature. Its muscular mass is smaller than that of the gastric sphincter. The duodenal sphincter is semicircular. The sphincter tapers off in thickness toward the lesser curvature (Fig. 5 e). Longitudinally sectioned, the duodenal sphincter has an ovoid shape. The sphincter begins at the distal part of the concave curvature and terminates at the most proximal part of the duodenum.

In contrast to the transient character of the gastric sphincter, the duodenal sphincter is permanent and is referred to in the adult pig as "pyloric sphincter." In the newborn pig the duodenal sphincter together with the torus pyloricus longitudinalis form the duodenal orifice of the pyloric canal. Because of the narrowing of the pyloric canal musculature "concave curvature" the gastric and duodenal sphincter can function independent of each other. The torus pyloricus longitudinalis (letter h in Fig. 2) is a solid muscular mass (Fig. 32) rather than mostly adipose tissue as in the adult pig (Fig. 31). The term "torus" means bulging projection (protuberance). The torus is located on the lesser curvature and extends vertically from the most distal part of the pyloric vestibule (antrum) through the pyloric canal into the proximal part of the duodenum (Figs. 2, 8, 10). Thus is derived the term "torus pyloricus longitudinalis." The muscle bundles in the torus run in all directions, which is in sharp contrast to the more orderly arrangement of the muscle bundles in the gastric sphincter (Fig. 28), the concave curvature (Fig. 29), and the duodenal sphincter (Fig. 30). The gastric portion of the torus pyloricus longitudinalis is normally level with the proximal part of the gastric sphincter. The body of the torus tapers off as it nears the duodenal sphincter. Its tail extends into the proximal portion of the duodenum (letter j in Fig. 2). The dipping of the tail into the duodenum is particularly well shown in Fig. 10. Because of its compact muscular structure the torus pyloricus longitudinalis can function indepently of the gastric and duodenal sphincter. In contrast to the local constricting action of the sphincters the torus can constrict, dilate, and move in a vertical direction (Fig. 9). While in general the torus py-

loricus longitudinalis is cone-shaped (Figs. 5, 16), there is a considerable variability of its form determined by its size and position in the pyloric canal lumen and the caliber of the latter (Fig. 9). The canal lumen is crescent-shaped (Fig. 11) because of the protrusion of the torus pyloricus longitudinalis into the canal lumen.

The gross anatomy described above for the stomach of the pig ten days old has been adopted by us as "standard of comparison," for at this developmental stage the cited anatomical landmarks may be readily identified. Thus normal and abnormal aberrancies of the stomach of newborn pigs may be recognized with some degree of certainty, and the gross anatomy of the stomach of the pig fetus (Fig. 12) may be easily studied. It is of interest that the gastric sphincter becomes more or less obliterated as the stomach of the newborn pig enlarges to accommodate increased food consumption. Thus the duodenal sphincter becomes the "pyloric sphincter" (Fig. 17).

In summary, the over-all gross anatomy of the stomach with the exception of the dual sphincter is basically that of the adult pig. The schematic diagram presented in Fig. 18 allows a ready review of the anatomy thus far described and illustrated. For the purpose of establishing a dimensional concept of the pyloric canal from birth to the tenth postnatal day, the average mean dimensions of the components of the pyloric canal are presented in Figs. 15, 16 and the range of the dimensions in Table 2. It may be appropriate here to insert a word of caution regarding the numerical values given in Table 2; that is, the data should be looked upon as suggestive of growth rather than static specific structural dimensions at a given age.

In Figs. 19, 20, 21 are shown the major gross anatomical differences

between the pyloric canal of the adult pig, the newborn pig, and the human stomach. It may be noted that the pyloric canal of the adult pig (Fig. 19) has a single sphincter "pyloric sphincter" but that the pyloric canal of the newborn pig has a dual sphincter "gastric" and "duodenal sphincter" (Fig. 20). The pyloric canal in the human stomach has a single sphincter, "pyloric sphincter" (Fig. 21). The latter is anatomically different, however, from the single sphincter of the adult pig in that no torus pyloricus longitudinalis protrudes into the canal lumen, and further a circular rather than semicircular thickening of the pyloric musculature forms the sphincter. The pyloric canal is actually a space in the antrum (pyloric vestibule) given the designation "canalis egistorius" by Forssell (1913). The "Muskelplatten" (plate-like hyperplastic muscle tissue) found by Bachmann (1952) within the lesser curvature musculature of the human stomach is a most intriguing observation from the academic point of view. In these benign muscle plates the muscle bundles run in all directions, which is characteristic of the torus pyloricus longitudinalis, and, in addition, the plates range in location from the antrum (pyloric vestibule) to the pyloric sphincter and in some instances within the latter. Keeping in mind that the "Muskelplatten" of Bachmann are located in an area occupied by the torus pyloricus longitudinalis, one may assume that the structures may represent a rudimentary torus in the human stomach.

Plate I.

Fig. 1. Longitudinal section of the stomach of a pig ten days of age. The stomach was fixed in situ during life.

a, Transverse stomach; b, Pylorus; c, Pyloric canal. At arrow, Incisura angularis. x 2.

Plate I

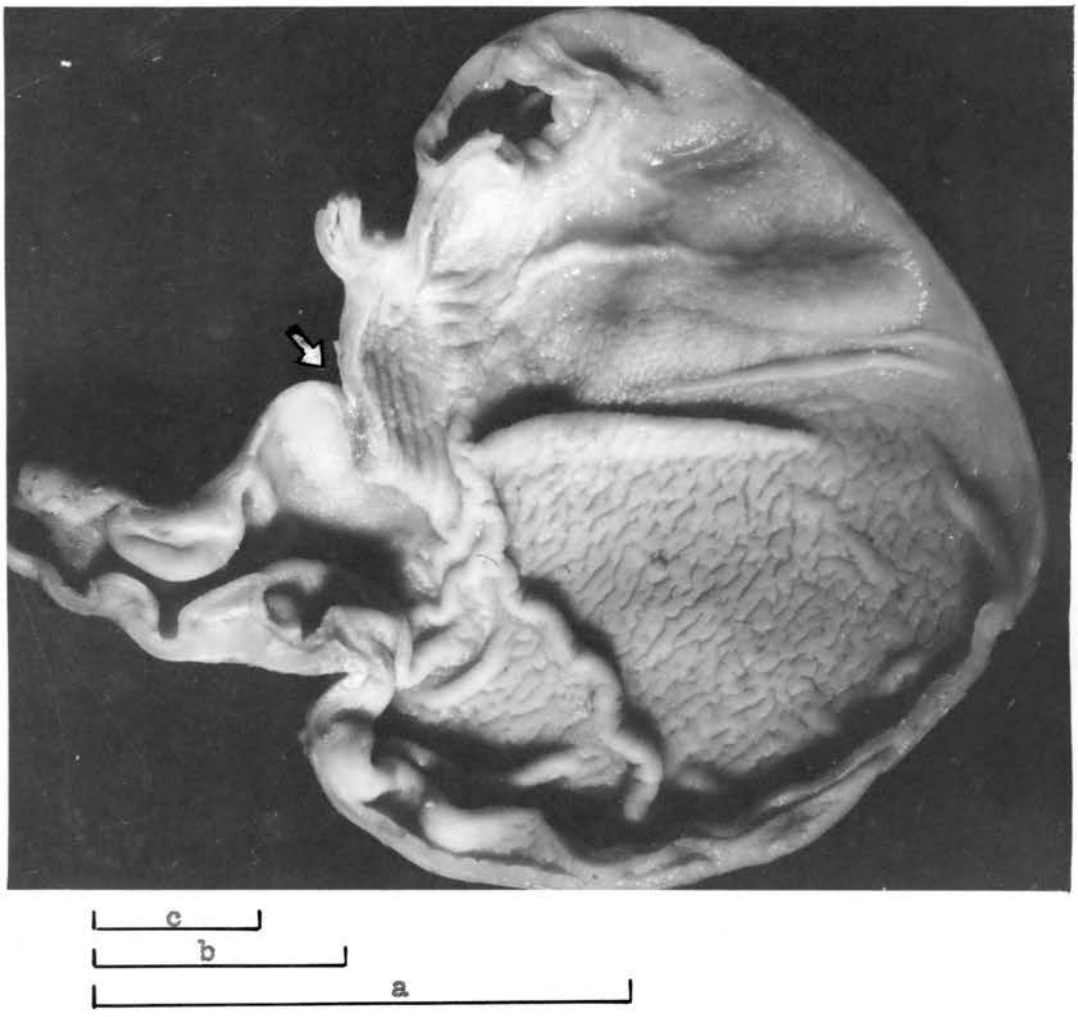


Fig.1

Plate II

Fig. 2. Longitudinal section through the pylorus of the stomach of a pig ten days of age (Fig. 1).

a. Sphincter antri; b, Pyloric vestibule (antrum); c. Longitudinal mucosal fold; d, Mucosa (mucous membrane); e, Gastric sphincter; f, Concave curvature; g, Duodenal sphincter; h, Torus pyloricus longitudinalis; i', Greater curvature musculature; i'', Lesser curvature musculature; j, Duodenum; k, Semicircular mucosal fold. At arrow, beginning of the pyloric canal and the canal lumen. x 4.

Plate II

Fig.2

Plate III

Fig. 3. Transverse section through the distal end of the pyloric vestibule of the stomach of a pig ten days of age.

a, Torus pyloricus longitudinalis; b, Semicircular mucosal fold; c, Longitudinal mucosal fold. x 5.

Comment.

The semicircular mucosal fold is in general not so prominent as is shown in Fig. 3. The fold is located at the base of the pyloric vestibule. The fold is capable of contraction but apparently does not close the gastric orifice of the gastric sphincter. The function of the fold is still to be clarified.

Plate III

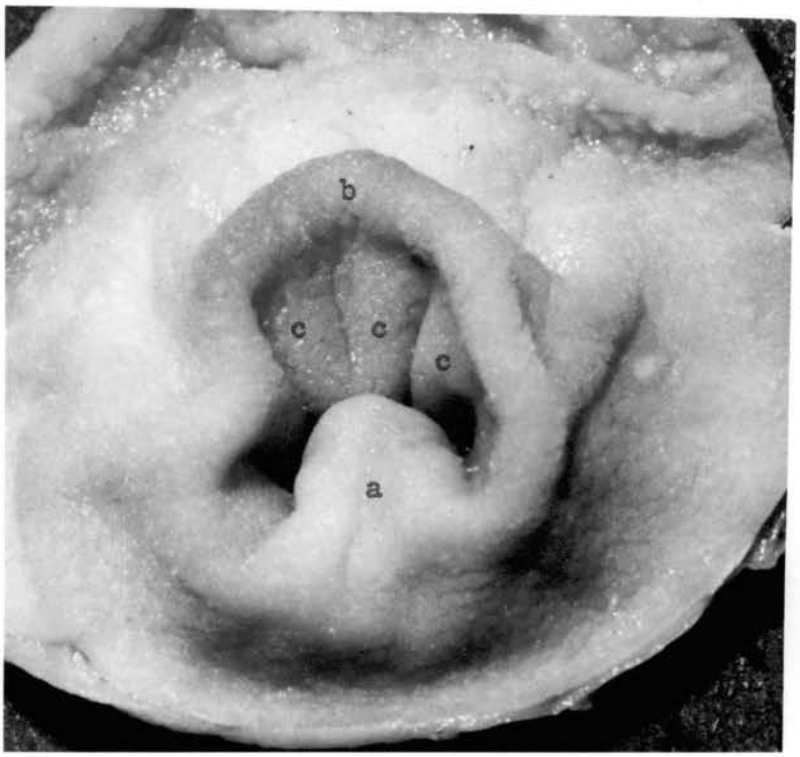


Fig.3

Plate IV

Fig. 4. Pyloric canal of the stomach of a pig at birth.

a, Longitudinal mucosal folds; b, Torus pyloricus longitudinalis; c, Duodenal sphincter; d, Longitudinal mucosal fold extending into the duodenum. x 6.

Fig. 5. Pyloric canal of the stomach of a pig ten days of age.

a, Longitudinal mucosal folds; b, Torus pyloricus longitudinalis; c, Gastric sphincter; d, Concave curvature; e, Duodenal sphincter; f, Duodenal mucosal fold. x 5.

Plate IV

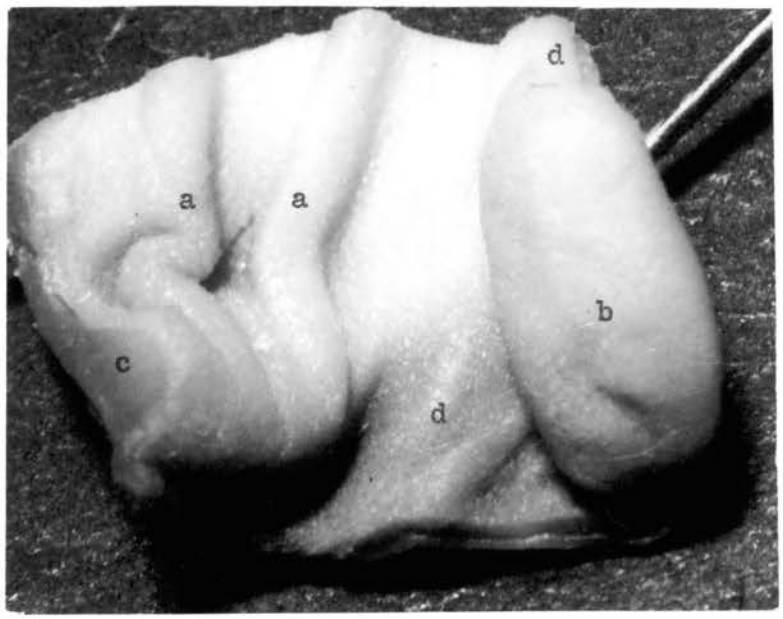


Fig.4

Fig.5

Plate V

Fig. 6. Longitudinal section through the pyloric canal of the stomach of a pig three days of age.

a, Longitudinal mucosal folds; b, Torus pyloricus longitudinalis; c, Gastric sphincter; d, Concave curvature; e, Duodenal sphincter; f, Duodenum; g', Wall of pylorus (greater curvature); g", Wall of pylorus (lesser curvature. x 5½.

Fig. 7. Longitudinal section through the pyloric canal of the stomach of a pig ten days of age.

a, Longitudinal mucosal fold; b, Torus pyloricus longitudinalis; c, Gastric sphincter; d, Concave curvature; e, Duodenal sphincter; f, Duodenum. x 5½.

Comment.

The pyloric canal shown in Fig. 6 is from a pig that received only water ad libitum since birth. Note that the canal lumen is obstructed by prominent edematous longitudinal mucosal folds (starvation phenomenon). Water passed very slowly through the canal lumen.

The pyloric canal shown in Fig. 7 is from a pig that was allowed to suckle since birth. Note that the lumen is patent and that the longitudinal mucosal folds are less prominent. Water passed rapidly through the canal lumen.

Plate V

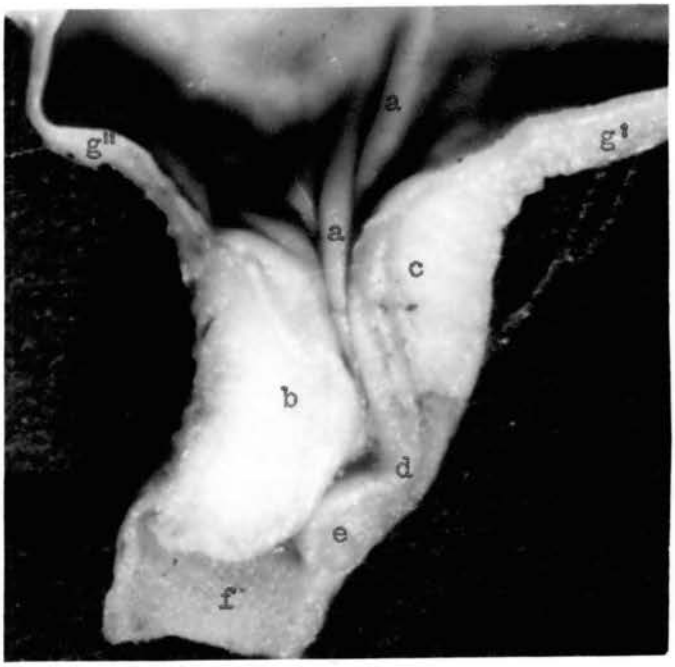


Fig.6

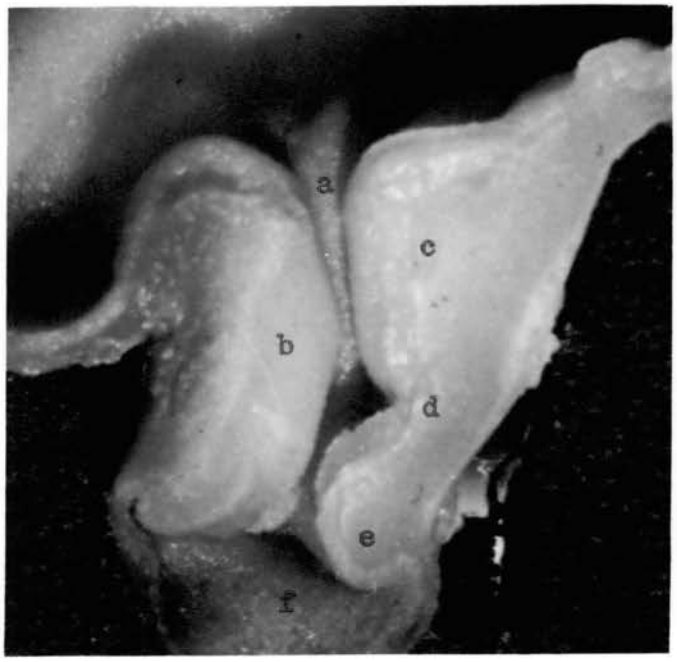


Fig.7

Plate VI

Fig. 8. Torus pyloricus longitudinalis of the stomach of a pig at birth.

a, Head; b, Body; c, Tail; d, Proximal part of duodenum; e, Longitudinal mucosal fold in pyloric canal lumen; e', Variable mucosal fold which may extend into the duodenum (Fig. 4 d); f, Wall of lesser curvature. x 6.

Fig. 9-A. Longitudinal section through the pyloric canal of the stomach of a pig at birth.

a, Torus pyloricus longitudinalis; b, Gastric sphincter; c, Concave curvature; d, Duodenal sphincter; e, Duodenal papilla. x 5.

Fig. 9-B. Longitudinal section through pyloric canal of the stomach of a pig at birth.

a, Torus pyloricus longitudinalis; b, Gastric sphincter; c, Concave curvature; d, Duodenal sphincter; e, Duodenal papilla. x 5.

Comment.

Note variation of the shape of the torus pyloricus longitudinalis determined by its size and position in the canal lumen. That the torus pyloricus longitudinalis moves up and down in the pyloric canal lumen is well shown in Fig. 9.

Runström (1939) states that the torus pyloricus functionalis is an anatomical preformed structure which participates to the same extent as the contraction and stratification of the muscularis propria.

Plate VI

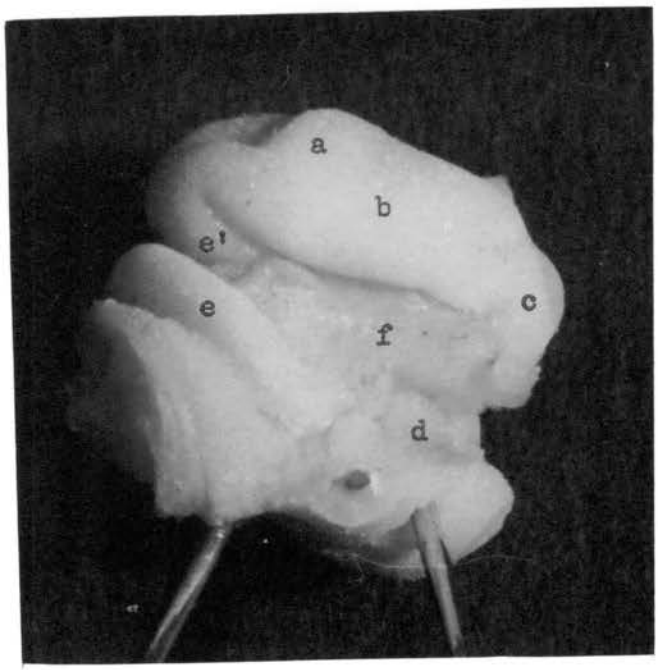


Fig.8

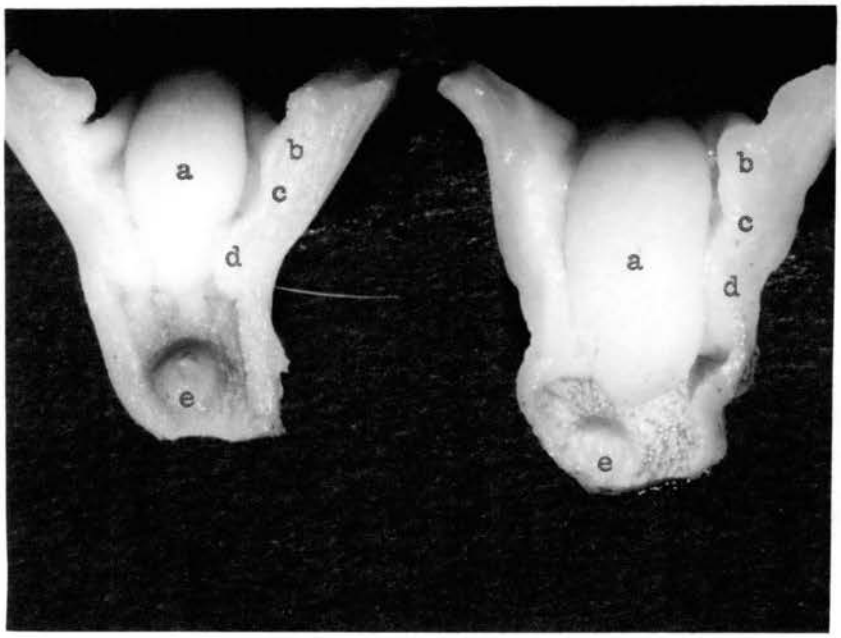


Fig.9

Plate VII

Fig. 10. Transverse section through the proximal part of the duodenum of the stomach of a pig at birth.

a, Duodenal sphincter; b, Tail of the torus pyloricus longitudinalis; c, Longitudinal mucosal fold extending into the duodenum; d, Duodenum. x 7.

Fig. 11. Transverse section through the pyloric canal below the head of the gastric sphincter of the stomach of a pig twenty-four hours of age.

a, Middle circular muscle coat; b, Torus pyloricus longitudinalis; c, Longitudinal mucosal folds; At arrow, Variable mucosal fold. x 6½.

Comment.

Note that the lumen of the pyloric canal is crescent-shaped because of the protrusion of the torus into the lumen. The longitudinal mucosal folds give the lumen the notched appearance. The diameter from the greater to the lesser curvature of this pyloric canal was 8.0 mm., and the transverse diameter 5.0 mm. The height of the torus pyloricus longitudinalis was 2.0 mm.

The actual bore of the lumen is difficult to determine because of the variation of the thickness of the pyloric canal musculature, height and width of the torus, and contraction of the muscle fibers during life and fixation. Because milk curds 1.0 and 2.0 mm. in diameter pass readily through the lumen, it may be assumed that the caliber of the lumen is about the same.

Hess (1941) states that pyloric casts found in the duodenum of infants range in diameter from 1.0 to 2.0 mm., which is indicative that the pyloric canal assumes this diameter during the passage of milk curds.

Plate VII

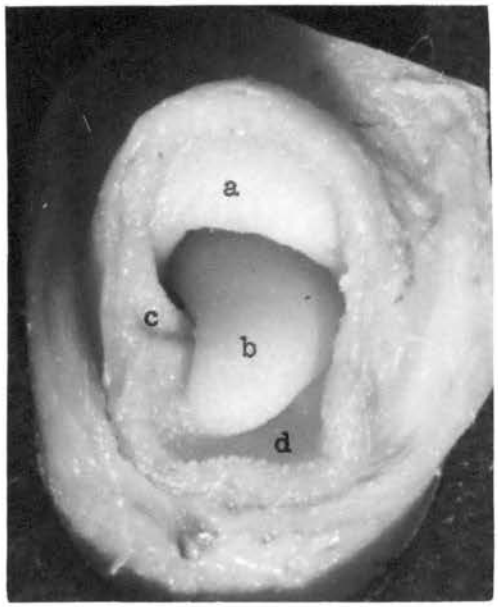


Fig.10

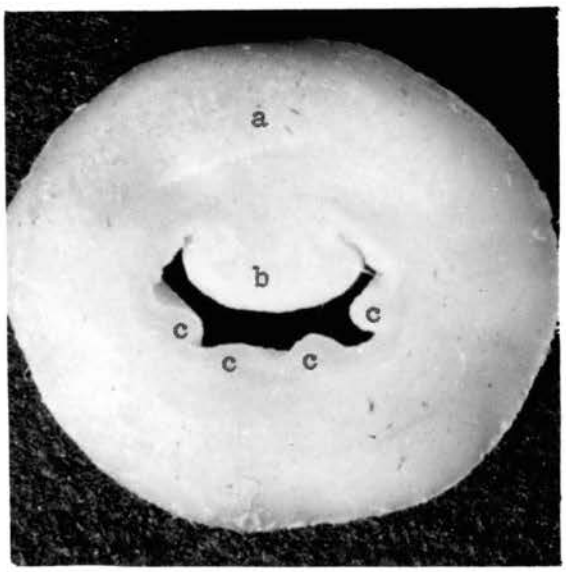


Fig.11

Plate VIII

Fig. 12. Longitudinal section of the stomach of a pig fetus C. R. 90 mm. (about 25 days of age).

a, Torus pyloricus longitudinalis; b, Gastric sphincter; c, Concave curvature; d, Duodenal sphincter; e, Incisura angularis; f, Magenstrasse; g, Esophagus; h, Diverticulum ventriculi; i, Longitudinal mucosal folds. x 5.

Comment.

At this developmental stage the pyloric canal is closed by a very tenacious plug of mucus. The same kind of mucus is present in the primitive pyloric vestibule. The gastric juice ranges in amount between 0.5 and 1.0 ml. at this fetal stage.

According to Sauer (1924) the stomach of the human fetus has no pyloric valve, and closure of the distal end of the stomach occurs by contraction of the entire pyloric segment known as canalis pylori.

Niero (1947) states that in the embryological development of the pylorus and the sphincter the circular muscle of the pyloric musculature develops first at the lesser curvature. Through thickening of the muscle coat at the distal portion of the pylorus the primitive pyloric sphincter is formed.

Torgersen (1942) postulates that the pyloric sphincter of the human stomach is part of the duodenal cap (bulb), which according to him is a "Zwischendarm." This concept is based on the phylogenetic hypothesis that the pyloric canal has arisen from two different parts of the intestinal tract which have united at the point known as pyloric canal.

Plate VIII

Fig.12

Plate IX

Fig. 13. Longitudinal section of the stomach of a pig at birth.

a, Torus pyloricus longitudinalis; b, Gastric sphincter; c, Concave curvature; d, Duodenal sphincter; e, Semicircular mucosal fold; f, Transverse bulge of lesser curvature musculature (primitive sphincter antri); g, Diverticulum ventriculi. x 3.

Comment.

Note the absence of mucosal folds in the longitudinal and transverse stomach. The stomach was distended with gas and contained 5 ml. of yellowish gastric juice. Tenacious mucus overlaid the gastric orifice of the pyloric canal lumen. The latter was patent (Fig. 14).

Plate IX

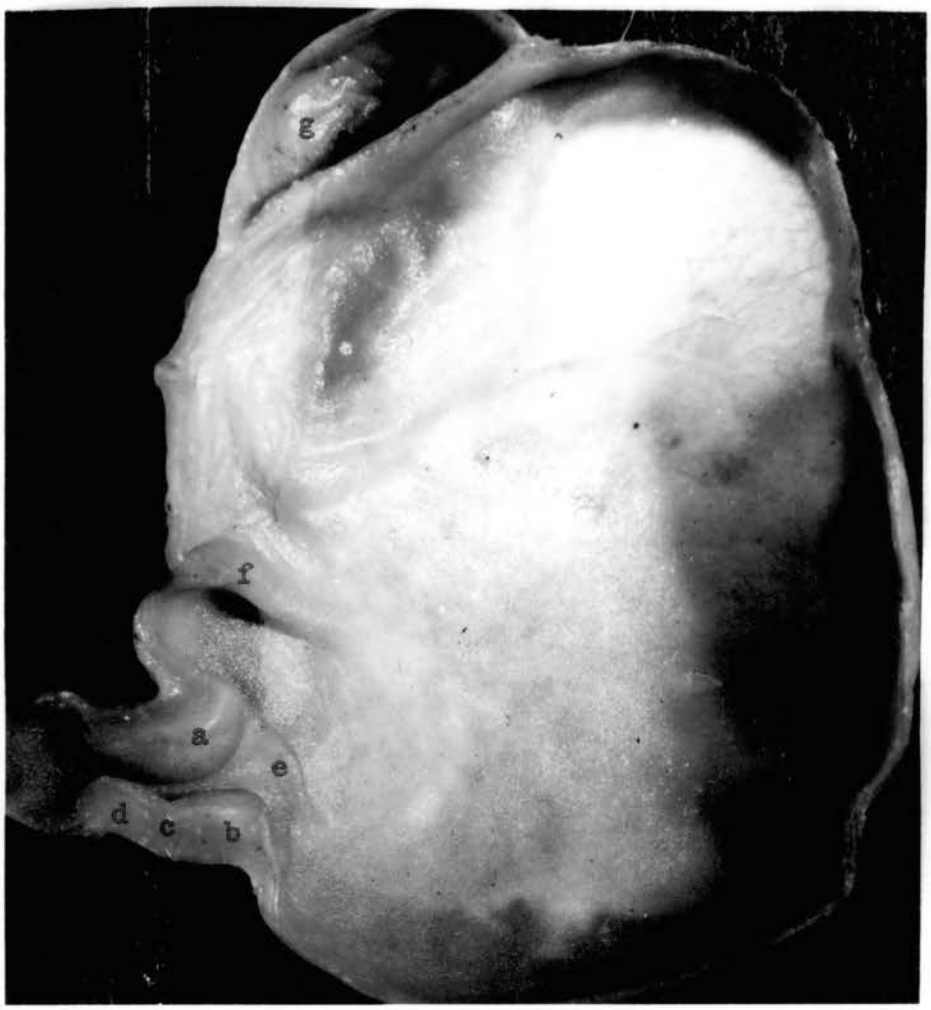


Fig.13

Plate X

Fig. 14. Longitudinal section through the pyloric canal of a pig at birth (Fig. 13).

a, Torus pyloricus longitudinalis; b, Gastric sphincter; c, Concave curvature; d, Duodenal sphincter; e', Greater curvature musculature; e", Lesser curvature musculature; f, Semicircular mucosal fold; g, Longitudinal mucosal fold; h, Mucosa (mucous membrane); i, Duodenum. Arrow is in the pyloric canal lumen which is patent. x 10.

Comment.

This pyloric canal is representative of the average "norm" and "standard of comparison" of the pyloric canal at birth. The dimensions of the components of the pyloric canal are presented in Fig. 15, 16. See also Table 2.

Plate X

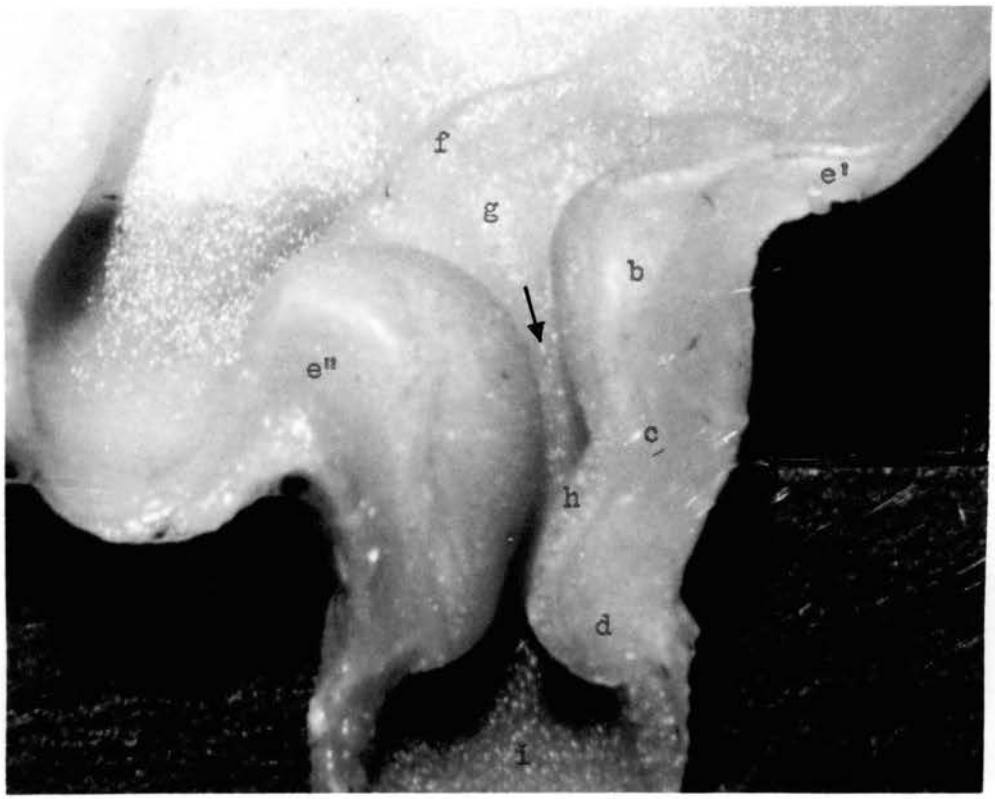


Fig.14

Plate XI

Fig. 15. Schematic diagram of a longitudinal section through the pyloric canal of a pig at birth (Fig. 14).

The dimensions are the rounded means of twenty-five pigs, a few minutes to five hours of age. Compare dimensions with those of the pyloric canal in a pig ten days of age (Table 2).

Fig. 16. Schematic diagram of frontal view of the torus pyloris longitudinalis of a pig at birth (Fig. 14).

Note the cone-like shape of the torus pyloricus longitudinalis. Additional dimensions of the structure are presented in Table 2.

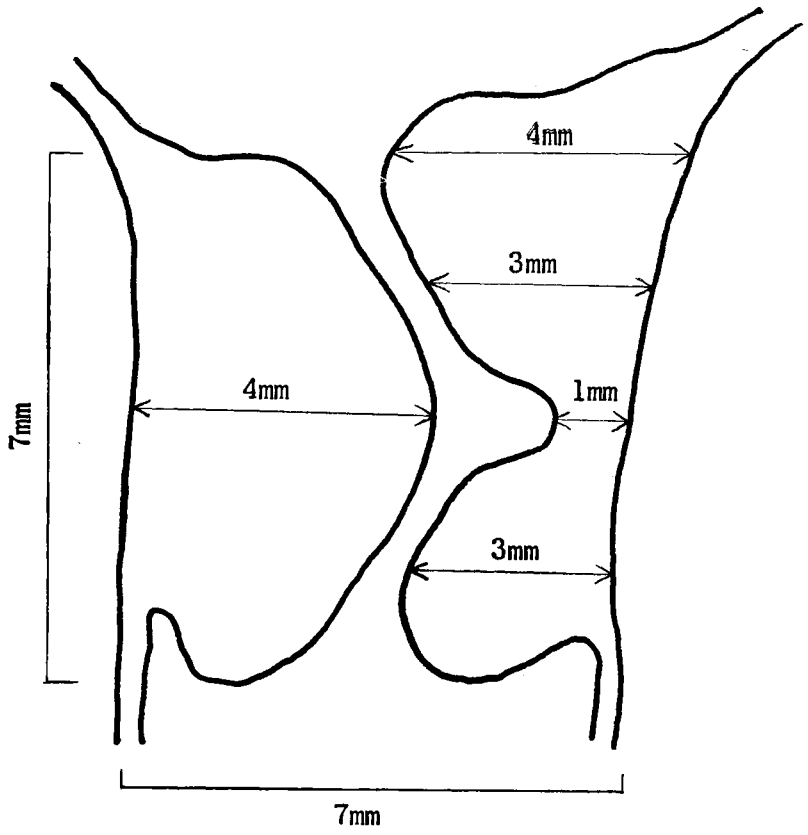


Fig. 15

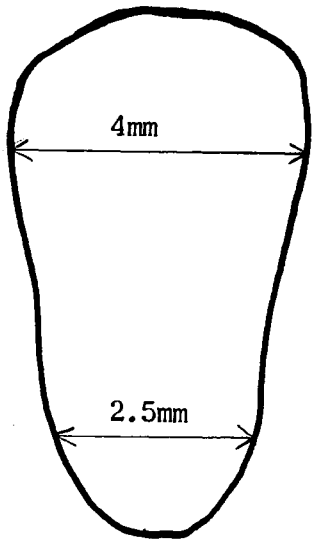


Fig. 16

TABLE 2

Range and Average Dimensions of the Gross Anatomical Structures of the Pyloric Canal of the Stomach of Pig at Birth (Twenty-five Pigs), and Tenth Postnatal Day (Ten Pigs).

Structure	At Birth		Tenth Postnatal Day	
	Range (mm.)	Average (mm.)	Range (mm.)	Average (mm.)
Torus pyloricus Longitudinalis				
Length	5.5-8.0	7.0	8.0-.0	9.0
Height	2.0-3.0	2.5	3.0-4.0	3.0
Width of head	3.0-4.0	4.0	4.0-5.0	4.0
Width of tail at entry of duodenum	2.0-3.0	2.5	2.0-4.0	3.0
Width of torus including lesser curvature	4.0-5.0	4.0	4.0-6.0	5.0
Gastric sphincter				
Width of head	3.0-4.0	4.0	4.0-6.0	5.0
At half length	2.0-3.0	3.0	4.0-5.0	4.0
At concave curvature	1.0-2.0	1.0	2.0-2.5	2.0
Concave curvature Width	0.8-1.0	1.0	1.0-2.0	1.5
Duodenal sphincter. Width	2.5-3.0	3.0	2.5-3.0	3.0
Pyloric canal				
Length	6.0-8.0	7.0	8.0-10	9.0
Total width from greater to lesser curvature	6.0-8.0	7.0	8.0-10	9.0

Plate XII

Fig. 17. Longitudinal section through the pyloric canal of the stomach of a pig thirty days of age.

a, Torus pyloricus longitudinalis; b, Pyloric sphincter (duodenal sphincter); c', Greater curvature musculature of the pylorus; c", Lesser curvature musculature of the pylorus; d, Sphincter antri; e, Longitudinal mucosal folds in the pyloric vestibule (antrum); f, Duodenum. x 5.

Comment.

The sphincter antri (d) is closed and thus shuts off the pyloric vestibule (antrum) from the rest of the stomach. The closure of the sphincter is believed to be the result of a deep contraction of the greater curvature musculature. Note that the pyloric sphincter is also closed. The simultaneous closure of these sphincters observed only in this pig may have been brought about by a pylorospasm resulting from prolonged mechanical manipulations of the stomach while fixing the latter extra-abdominally.

Plate XII

Fig.14

Plate XIII

Fig. 18. Schematic diagram of a longitudinal section of the stomach of a pig ten days of age.

The diagram is not drawn to scale. It is designed to aid identification of the gross anatomy of the stomach of newborn pigs as described and illustrated in the preceding pages of this thesis.

Note that the transverse stomach includes the pylorus, the proximal part of which is the sphincter antri, the pyloric vestibule (antrum), and the pyloric canal. The latter consists of four distinct structures, which are identified in Fig. 18.

Plate XIII

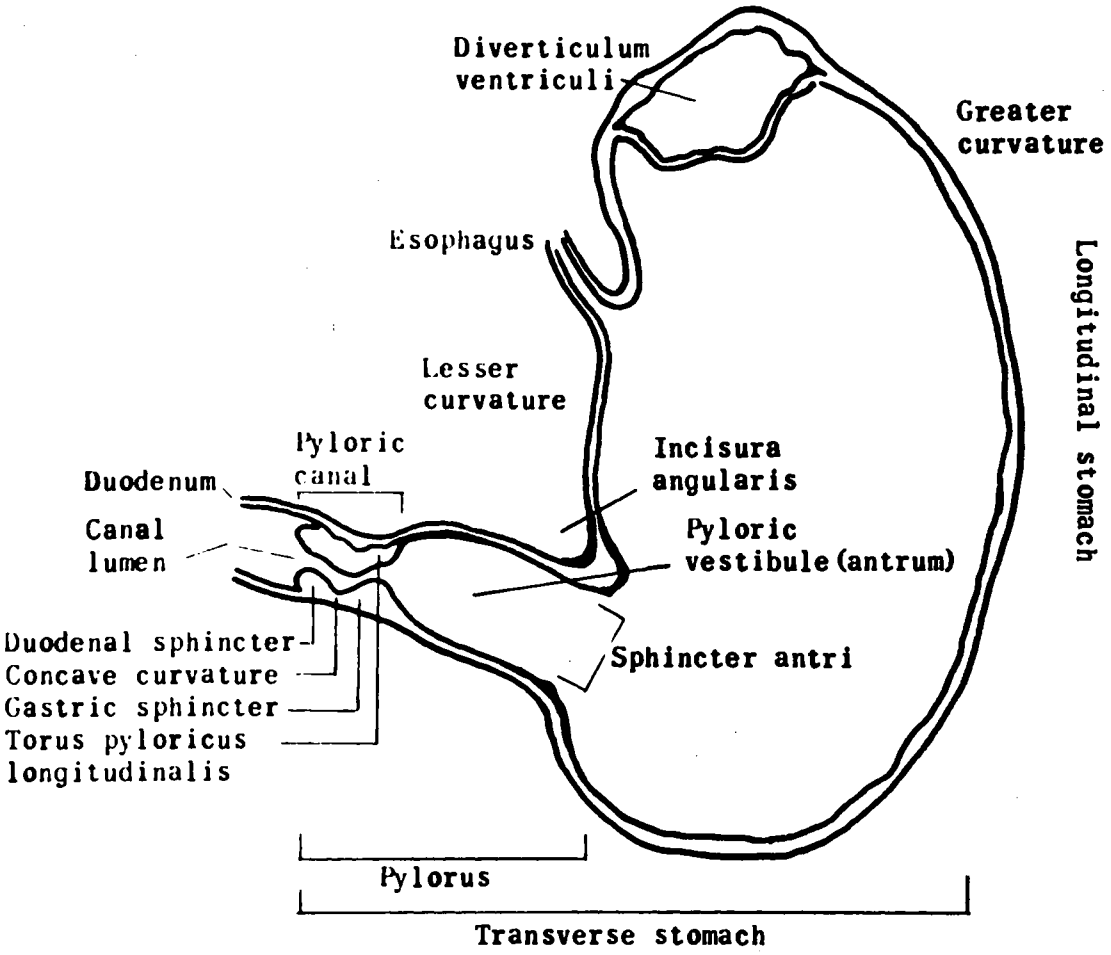


Fig.18

Plate XIV

Fig. 19. Schematic diagram of a longitudinal section of the stomach of a pig six months of age.

The pyloric canal of the stomach of the adult pig has only one sphincter. The sphincter corresponds to the duodenal sphincter of the pyloric canal of the newborn pig. The single sphincter together with the torus pyloricus longitudinalis (solid black) is known as the "pyloric sphincter."

With the exception of the presence of a torus the single sphincter of the stomach of the adult pig is analogous to that of the pyloric sphincter of the human stomach (Fig. 21).

Fig. 20. Schematic diagram of a longitudinal section of the stomach of a pig at birth.

The pyloric canal has two rather than one sphincter as in the adult pig. The sphincters are referred to in this thesis as "gastric" and "duodenal sphincter." The latter is the pyloric sphincter of the adult pig. The sphincters function independent of each other. Thus partial or complete patency of the canal lumen is determined by the degree of relaxation of either sphincter. The degree of stenosis results from the diseased state of the sphincters.

Fig. 21. Schematic diagram of longitudinal section of the stomach of adult human (After Anson, 1950).

The pyloric canal is a space within the antrum (pyloric vestibule) rather than a well defined structure as in the pig. The pyloric canal area is referred to by Forssell (1913) as "canalis egistorius." The pyloric sphincter is formed by a thickening of the greater and lesser curvature musculature of the pylorus. There is no suggestion of a torus pylorus longitudinalis although it has been mentioned in the text that the "Muskelplatten" of Bachmann (1952) may represent a displaced rudimentary torus pyloricus longitudinalis. No diverticulum ventriculi is present.

Plate XIV

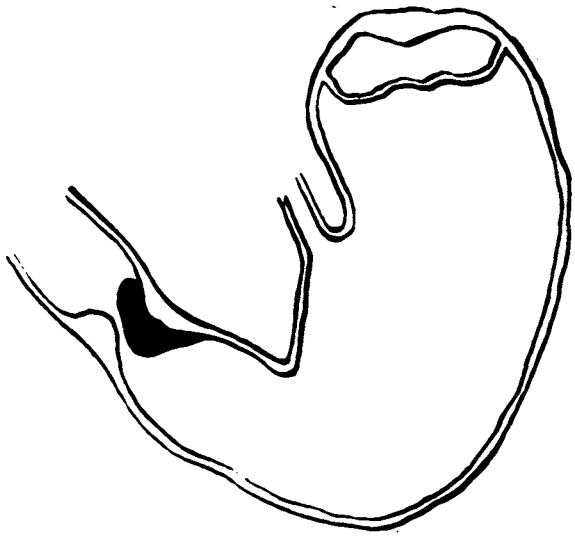


Fig.19

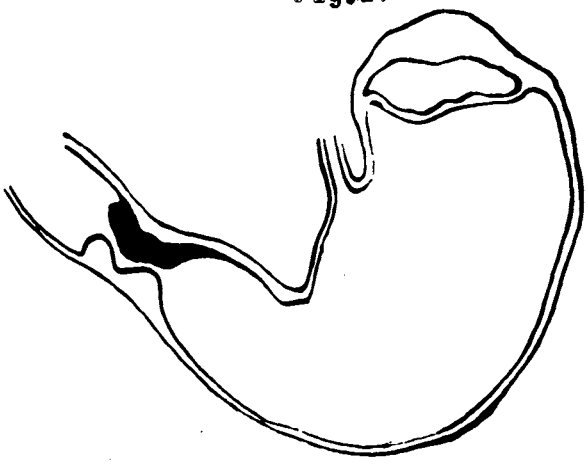


Fig.20

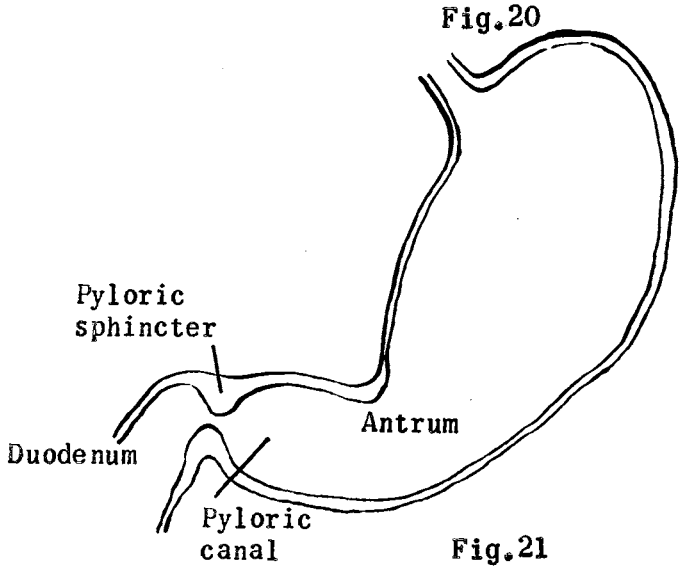


Fig.21

Microscopic Anatomy of the Stomach of the Normal Newborn Pig

Pylorus

The histology of the pylorus at the level of the head of the torus pyloricus longitudinalis (Fig. 22 f) conforms to the general structural plan of the digestive tube of man. The pylorus of the stomach of newborn pigs consists of eight layers: surface epithelium and glands, lamina propria, muscularis mucosae, submucosa, inner oblique muscle coat, middle circular muscle coat, outer longitudinal muscle coat, and adventitia or serosa. Commonly these layers are grouped into four structural units: mucous membrane (mucosa), submucosa, muscularis externa, and adventitia. The mucous membrane (mucosa) has three distinct components: surface epithelium and glands, lamina propria, and muscularis mucosae (made up of longitudinal and circular smooth muscle fibers). The pyloric glands (Fig. 25 a) are short and tortuous (coiled). The glands contain mucoid cells. No enzyme worthy of note is secreted by the pyloric glands. Fox and Castle (1942) found that an intrinisc factor is produced by the pyloric glands. The latter are embedded in the lamina propria, which consists of reticular, elastic and collagenic fibers, blood and lymph vessels, and muscle fibers derived from the longitudinal layer of the muscularis mucosae. The lamina propria connects with the muscularis mucosae which is a narrow band of smooth muscle. Coursing within the latter is a small quantity of connective tissue fibers. The muscularis mucosae is made up of two layers of muscle fibers, the outer longitudinal layer (toward the glands) and the inner circular layer (toward the submucosa). It is generally believed that the muscularis mucosae controls

the height and movement of the villi, allows localized movement of the mucous membrane (mucosa), and tends to throw the latter into folds. Rundström (1939) postulates that the movement of the mucous membrane results from contraction and stratification of the muscularis mucosae and filling of blood vessels and tissue interspaces. The submucosa (Fig. 25 d) consists of coarse, more or less loosely arranged connective tissue, blood and lymph vessels, and a ganglionated autonomic neural plexus (plexus of Meissner). The submucosa with its inherent pliability makes an ideal core for the longitudinal and the circular mucosal folds as well as strengthens the gastric and the duodenal sphincter of the pyloric canal (Figs. 22, 23). The muscularis externa is composed of three muscle coats, an inner oblique, middle circular, and outer longitudinal muscle layer. The latter does not extend over the lesser curvature and again may only surround the greater curvature in a patchy fashion (Fig. 23 d). Within and between these muscle layers are connective tissue septa and blood and lymph vessels. The action of these muscle coats is said to regulate the state of tonus of the muscularis externa and the size of the lumen of the pylorus and of the pyloric canal and to constitute the force which propels ingesta along the digestive tube. Between the longitudinal and the circular muscle layers and scattered within the muscle tissue is the motor myenteric plexus (plexus of Auerbach) ganglia which may be seen in Fig. 24 (arrow). The plexus of Auerbach together with the plexus of Meissner is referred to by Alvares (1948) as "musculoneural apparatus." This intrinsic communicating neural structure consists of both parasympathetic and sympathetic fibers. The adventitia (serosa), the outermost coat, is made up of connective tissue, lymph and blood vessels, areoler tissue, and nerves. The layer is covered by mesothelium.

Pyloric Canal

In Figs. 22, 23 is shown the over-all histology of the pyloric canal of the stomach of newborn pigs. The greater curvature has the same anatomical make-up as the pylorus. The gastric sphincter (Fig. 22 a) is covered by glands morphologically identical to those of the pylorus. The lamina propria, muscularis mucosae, and the submucosa, however, are more prominent than in the pylorus (Fig. 22 f), lesser curvature (Fig. 22 b), and duodenal sphincter (Fig. 23 a). The blood vascular system is also more abundant. There is a proportional increase of the reticular, elastic, and collagenic fibers. An average connective tissue septal pattern of the gastric sphincter is shown in Fig. 28. The degree of bulging of the greater curvature musculature (Fig. 22 c) varies with the developmental state of the stomach, the thickness of the inner oblique and middle circular muscle coat, and the width of the submucosa. The concave corvature resulting from a narrowing of the various layers making up the greater curvature is covered by glands similar to that of the pylorus and gastric sphincter. So-called "transitional" glands (Fig. 26) may be scattered among the glands of the concave curvature. The function of these aberrant glands is obscure. Although on one hand the glands may be merely aberrant pyloric glands, they may on the other hand represent a vestige of special glands with a phylogenetic background as yet not understood. The presence of a special gland would give strength to the postulate of Torgersen (1942) that the pylorus is a product of the union of two parts of the intestinal tract of different origin which have united at the point known as the pyloric canal as far as the stomach in man is concerned. It is noteworthy that, although a complete absence of the longitudinal muscle coat on the lesser curvature

as claimed by Torgersen (1942) is characteristic of the pig stomach, the longitudinal muscle coat of the stomach of the newborn pig has hiatuses on the greater curvature. A longitudinal muscle coat hiatus on the greater curvature may be seen just below the letter g in Fig. 23. The connective tissue septal pattern in the concave curvature differs from that in the gastric sphincter (Fig. 28) in that some septa tend to run somewhat oblique to the outer longitudinal muscle coat (Fig. 29). The oblique septa join the connective tissue bands only occasionally within the longitudinal muscle coat. The duodenal sphincter (Fig. 23 a) has a microscopic anatomy similar to that of the gastric sphincter. The submucosa, however, tends to vary in width from broader than the submucosa of the concave curvature to a narrow band of connective tissue (at letter a near bottom of Fig. 23). The connective tissue septal pattern tends to be obliquely oriented (Fig. 30). Although the orientation may represent a continuation of the concave curvature pattern, there is, however, a greater quantity of slender interwoven collagenic fibers in the duodenal sphincter than in the gastric sphincter (Fig. 28) and concave curvature (Fig. 29). It is generally inferred that numerous slender collagenic fibers interwoven in loose coarse connective tissue septa makes for a high degree of motility of a structure. Immobility is said to result from an increase of broad coarse septa, dense cords, and sheets of connective tissue (Fig. 49). The torus pyloricus longitudinalis is a solid muscular mass (Fig. 32). This is in sharp contrast to the microscopic anatomy of the torus in the adult pig, where the structure consists largely of adipose tissue, scattered muscle bundles derived from the middle circular muscle coat, connective tissue fibers and blood and lymph vessels (Fig. 31). The muscle bundles in the torus pyloricus longitu-

dinalis of the stomach of newborn pigs are derived from the middle circular muscle coat. The bundles vary in size and orientation. The former condition seems to be determined by the connective tissue septal pattern (black bands, Fig. 32). From the major (primary) septa which originate from the fibrous mucosa, arise numerous slender fibers forming an irregular network of collagenic fibers, which are in communication with the same type of fibers in the middle circular muscle coat of the pyloric canal. The submucosa has an abundant quantity of collagenic fibers. Blood vessels are plentiful, and many are embedded in broad connective tissue septa (Fig. 33). There is no musculoneural apparatus in the torus. Thus moveability of the torus pyloricus longitudinalis is dependent upon an inherent, independent tonus and rhythmic contraction of the muscle fibers rather than being initiated or accelerated by a neural system. Evans (1952) postulates that muscle fibers are likely connected together by fine intercellular bridges to form a syncytium by which the excitatory process can be transmitted from cell to cell, though under conditions which are at present unknown. The breaking up of the muscularis mucosae into small bundles and strands in the torus pyloricus longitudinalis (Fig. 34) is noteworthy. This dissolution of the muscularis mucosae accounts for the absence of a well defined lamina propria muscularis mucosae in the torus. Moveability of the mucous membrane, if it does occur, is thus dependent upon the quantity of muscle tissue and connective tissue fibers, in particular elastic fibers (Fig. 35), and orientation of the same in the submucosa. The over-all septal pattern of the pylorus differs from that of the gastric sphincter, the duodenal sphincter, and the concave curvature in that it seems to be a mixture of the septal pattern of the aforementioned structures (Fig. 33).

In summary, it may be said that the limited survey of the microscopic anatomy of the pylorus and pyloric canal carried out by us proved supportive of our current tendency to restrict the term "pyloric canal" to an anatomical area which appears to be grossly and microscopically a distinct structural entity in the stomach of the newborn pig. The study further revealed that the torus pyloricus longitudinalis in the stomach of the newborn pig is a solid muscular mass rather than a structure chiefly made up of adipose tissue as in the adult pig.

The staining methods employed for the study of the microscopic anatomy were those of Schleicher (1943, 1935), Gomori (1950), Weigert's elastic tissue stain, and iron-hematoxylin stain as cited by Cowdry (1948 a, 1948 b), and Mitchell and Wislocki (1944).

Plate XV

Fig. 22. Longitudinal section through the gastric sphincter and concave curvature of the pyloric canal of the stomach of a pig at birth.

a, Gastric sphincter; b, Concave curvature; c, Greater curvature musculature; d, Torus pyloricus longitudinalis; e, Pyloric canal lumen.
Photomicrograph. x 90.
Schleicher's hematoxylin-eosin stain.

Fig. 23. Longitudinal section through the duodenal sphincter and concave curvature of the pyloric canal of the stomach of a pig at birth.

a, Duodenal sphincter; b, Concave curvature; c, Greater curvature musculature; d, Outer longitudinal muscle coat with hiatus; e, Pyloric canal lumen; f, Duodenum; g, Adventitia; h, Torus pyloricus longitudinalis.
Photomicrograph. x 90.
Schleicher's hematoxylin-eosin stain.

Comment.

Note the striking variation in the width of the submucosa in the gastric sphincter, duodenal sphincter, and concave curvature (letters a and b are within the submucosa). A distinct hiatus of the outer longitudinal muscle coat on the greater curvature is at letter g in Fig. 23.

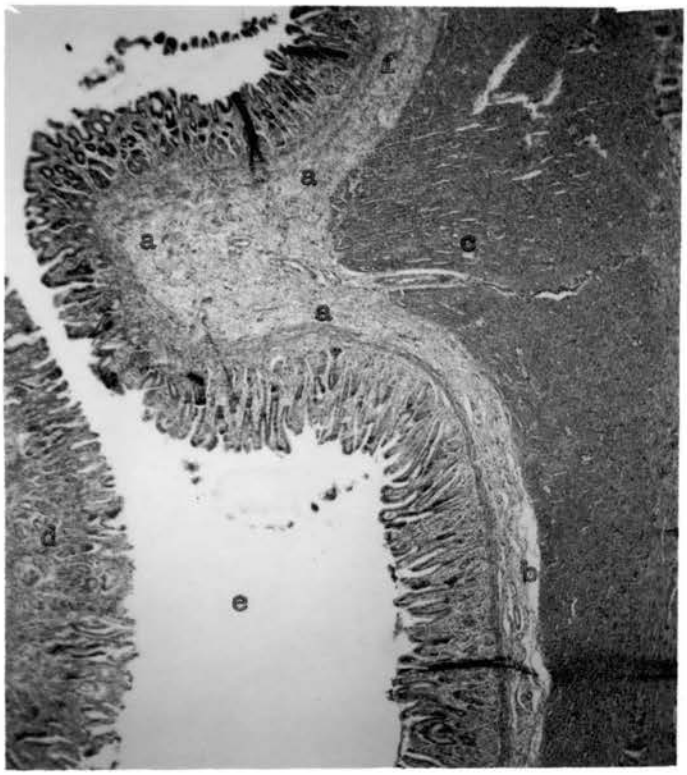


Fig. 22

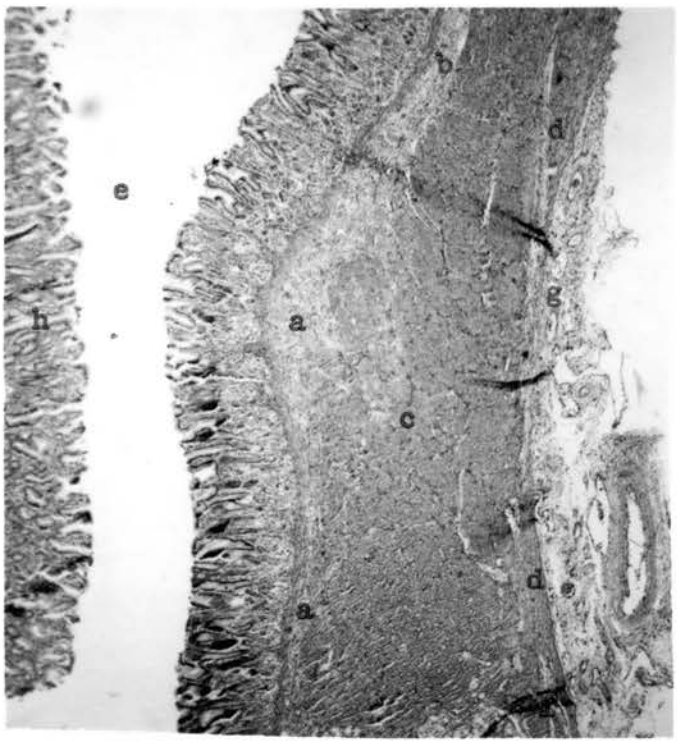


Fig. 23

Plate XVI

Fig. 24. Transverse section through the pyloric vestibule (antrum) at level of the head of the torus pyloricus longitudinalis of the stomach of a pig at birth.

a, Outer longitudinal muscle coat; b, Middle circular muscle coat; c, Submucosa; d, Adventitia; Arrows point at ganglion of plexus of Auerbach.
Photomicrograph. x 110.
Schleicher's hematoxylin-eosin stain.

Fig. 25. Longitudinal section through mucous membrane (mucosa) of the pyloric vestibule (antrum) near the gastric sphincter of the stomach of a pig at birth.

a, Pyloric glands; b, Lamina propria and muscle fibers derived from the longitudinal layer of the muscularis mucosae between the gland spaces; c, Muscularis mucosae (longitidinal fibers at gland site, circular fibers at submucosa site); d, Submucosa. Muscle fibers of the circular and oblique muscle coat are at lower right corner of the figure.
Photomicrograph. x 250.
Schleicher's hematoxylin-eosin stain.

Comment.

The terms "mucous membrane" and "mucosa" are used interchangeably. The terms include the surface epithelium, glands, lamina propria, and muscularis mucosae.

The muscularis mucosae allow independent moveability of the mucous membrane. Contraction and relaxation of the muscularis mucosae is chiefly myogenic (Rundström, 1939).

Plate XVI

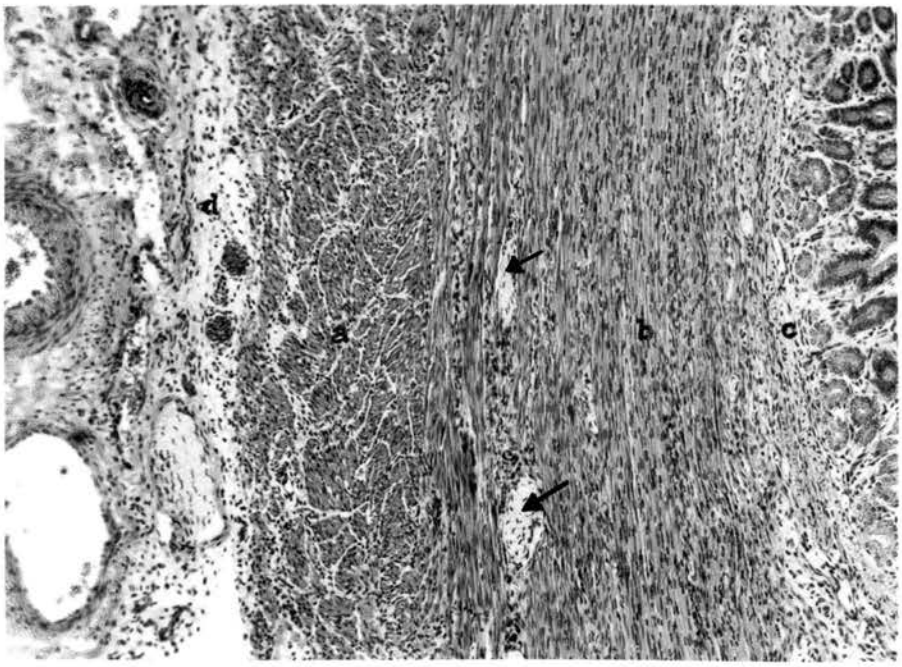


Fig.24

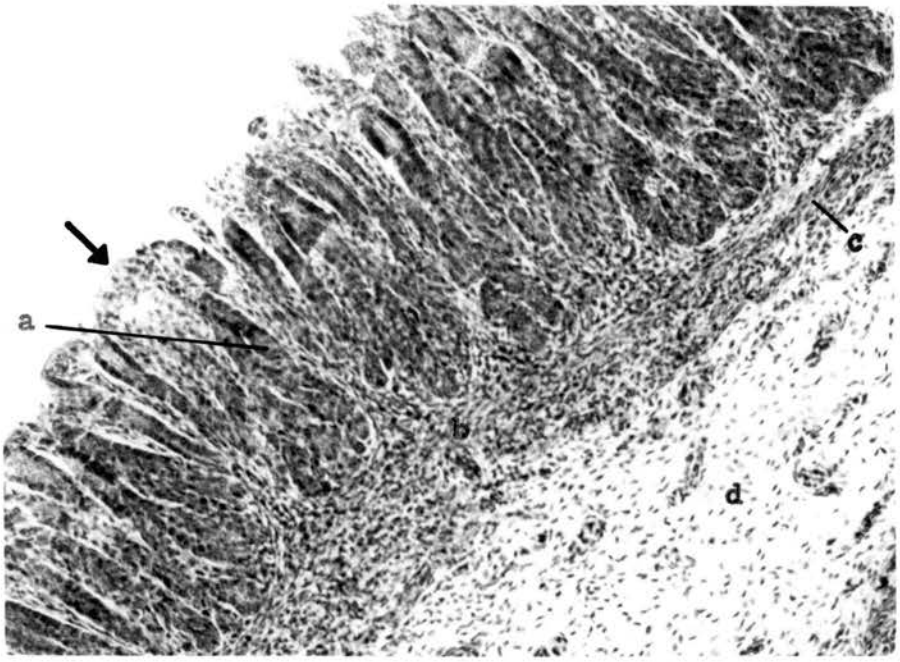


Fig.25

Plate XVII

Fig. 26. Longitudinal section through the mucous membrane (mucosa) of the concave curvature of the stomach of a pig at birth.

a, Transitional glands; b, Pyloric glands; c, Glands similar to torus pyloricus longitudinalis glands (Fig. 27).
Photomicrograph. x 250.
Schleicher's hematoxylin-eosin stain.

Comment.
The "transitional" glands are lighter in hue and the fundus has a triangular shape in contrast to the deeper hue and more bulb-like fundus of the surrounding glands. While the "transitional" glands may be modified pyloric or fundus glands they may represent a vestige of special glands with a distinctive phylogenetic background.

Fig. 27. Longitudinal section through the mucous membrane (mucosa) of the torus pyloricus longitudinalis of the stomach of a pig at birth.

The over-all morphology of the glands differs from that of the pyloric glands (Fig. 25) in that they are longer, are less tortuous (coiled), and do not rest upon a well defined lamina propria but are embedded in a very fibrous submucosa. There is no distinct muscularis mucosae in the torus pyloricus longitudinalis.

Plate XVII

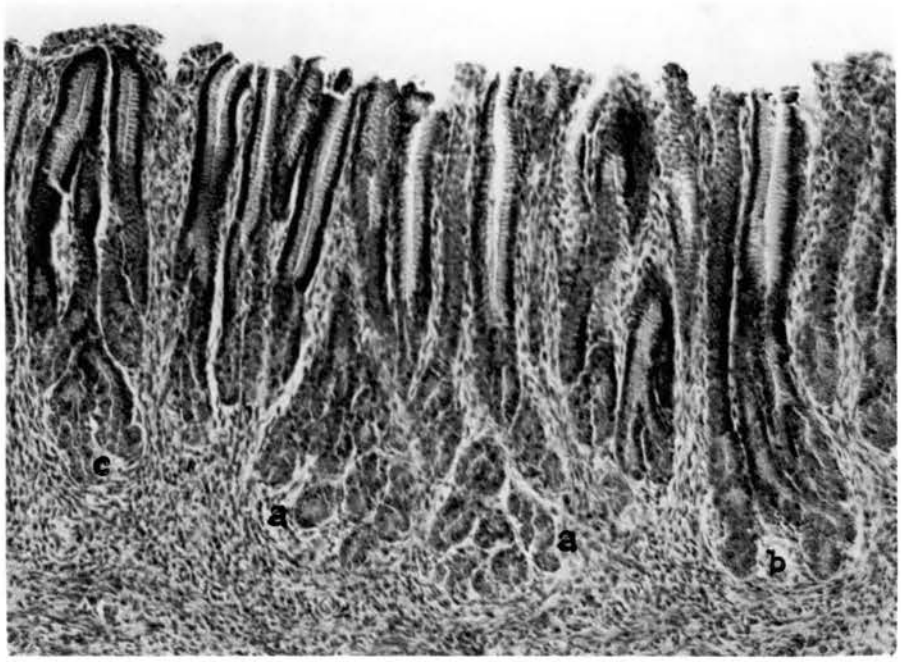


Fig.26

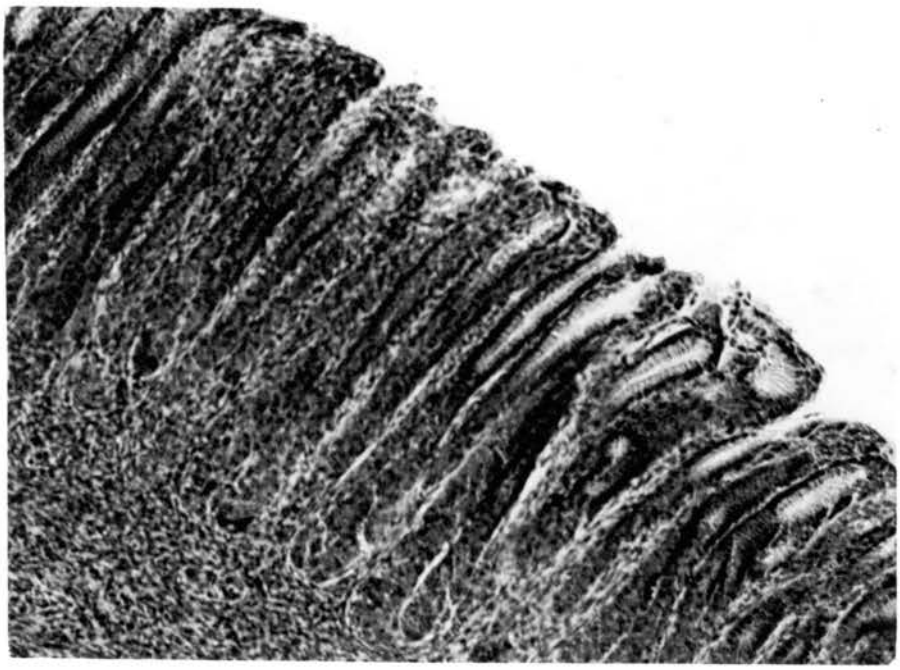


Fig.27

Plate XVIII

Fig. 28. Longitudinal section through the midportion of the gastric sphincter (pyloric canal) of the stomach of a pig at birth.

Normal average connective tissue septal pattern (blue strands). The fibrous tissue runs in all directions partitioning the musculature into various sized bundles giving the muscle layers a "braided appearance" (Alvarez, 1948). This arrangement of the connective tissue fibers not only strengthens but permits easy stretching of the sphincter.
Photomicrograph. x 90.
Schleicher's connective tissue stain.

Fig. 29. Longitudinal section through the midportion of the concave curvature (pyloric canal) of the stomach of a pig at birth.

Normal average connective tissue septal pattern (blue strands). The fibrous septa arise from the broad fibrous lamina (extreme left of picture). The fibrous strands have a tendency to run somewhat oblique to the outer longitudinal muscle coat (extreme right of the picture). The oblique fibers join occasionally the longitudinal connective tissue fibers in the longitudinal muscle layer. Stretching of the concave curvature longitudinally and horizontally is readily possible.
Photomicrograph. x 90.
Schleicher's connective tissue stain.

Fig. 30. Longitudinal section through the midportion of the duodenal sphincter (pyloric canal) of the stomach of a pig at birth.

Normal average connective tissue septal pattern (blue strands). Note the reorientation of the fibrous septa giving the musculature a braided appearance different from that of the gastric sphincter (Fig. 28) and the concave corvature (Fig. 29). It may be assumed, therefore, that stretching forces act differently on the duodenal, the gastric sphincter, and the concave curvature.
Photomicrograph. x 90.
Schleicher's connective tissue stain.

Comment.

The term "connective tissue" as used in this thesis includes three types of fibers: (a) collagenic, (b) elastic, and (c) reticular fibers. Orientation and abundance of the fibers determine the degree of motility of a structure.

Plate XVIII

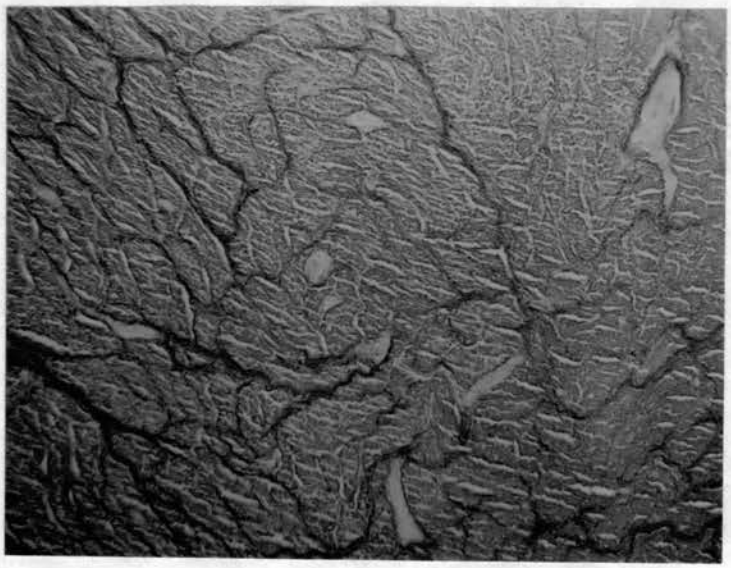


Fig. 28

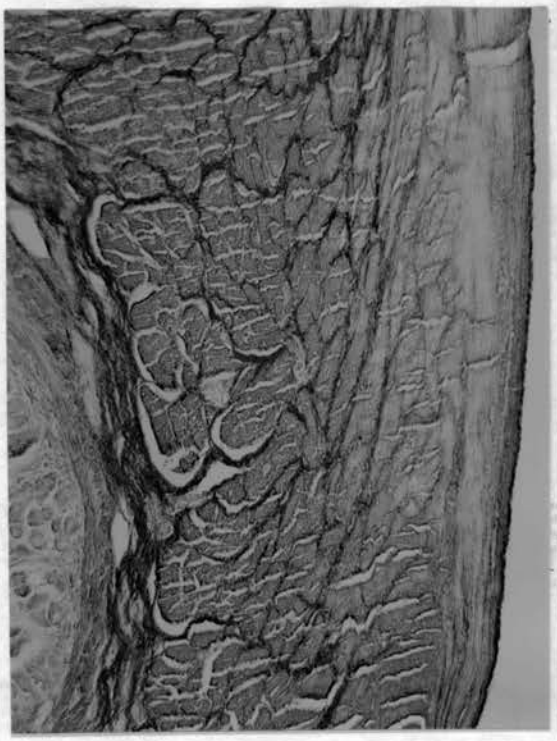


Fig. 29

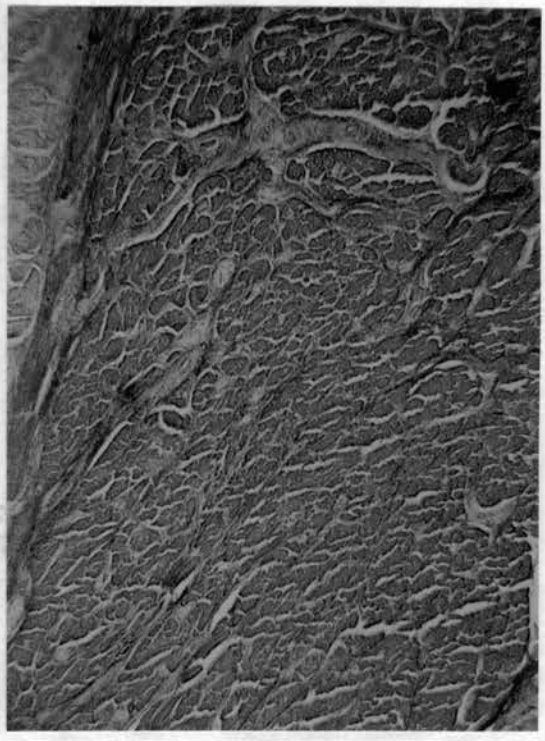


Fig. 30

Plate XIX

Fig. 31. Transverse section through the midportion of the head of the torus pyloricus longitudinalis of the stomach of a pig eleven months of age.

a, Adipose tissue; b, Muscle bundles; c, Connective tissue septa; c, Large blood vessel.

Fig. 32. Transverse section through the midportion of the head of the torus pyloricus longitudinalis of the stomach of a pig at birth.

a, Glands of torus pyloricus longitudinalis; b, Muscle bundles (note variation in size, shape and orientation). White arrow points at connective tissue lamina of the submucosa from which arise major (primary) connective tissue septa. Black arrows are in blood vessels. No adipose tissue is present.

Plate XIX

Fig.31

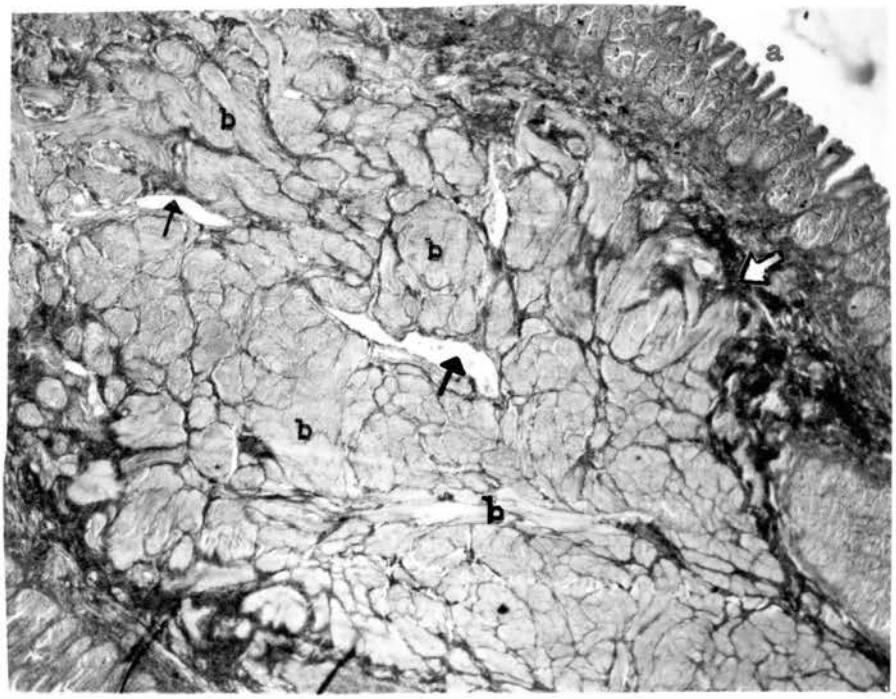


Fig.32

Plate XX

Fig. 33. Longitudinal section through the midportion of the torus pyloricus longitudinalis (pyloric canal) of the stomach of a pig at birth.

Normal average connective tissue septal pattern (blue strands). Compare the orientation of the septa with those in the gastric sphincter (Fig. 28), the concave curvature (Fig. 29), and the duodenal sphincter (Fig. 30). Note the marked variation of the size and the orientation of the muscle bundles. Observe that the torus glands rest upon a fibro-muscular lamina rather than upon a well developed lamina propria as in the pylorus (Fig. 25). The septal pattern allows contraction of the muscle bundles and vertical movement of the torus pyloricus longitudinalis.
Photomicrograph. x 180.
Schleicher's connective tissue stain.

Fig. 34. Transverse section through the midportion of the head of the torus pyloricus longitudinalis (pyloric canal) of the stomach of a pig at birth.

Note the variation of thickness, the orientation of the connective tissue septa (deep blue strands), and the conspicuous difference in size of the muscle bundles in the torus portion of the lesser curvature musculature (light greenish-blue areas left of arrow). The arrow which is in the glandular portion of the torus points at muscularis mucosae (narrow reddish-purple band), which breaks up into numerous small bands near the head of the torus (extreme right of photomicrograph). This phenomenon accounts for the absence of a distinct lamina propria and muscularis mucosae in the torus pyloricus longitudinalis.
Photomicrograph. x 180.
Gomori's trichrome stain.

Comment.

The over-all microscopic anatomy of the torus pyloricus longitudinalis suggests that the moveability of its mucous membrane (mucosa) is dependent upon the quantity of muscle strands and amount of connective tissue fibers in the submucosa.

Plate XX

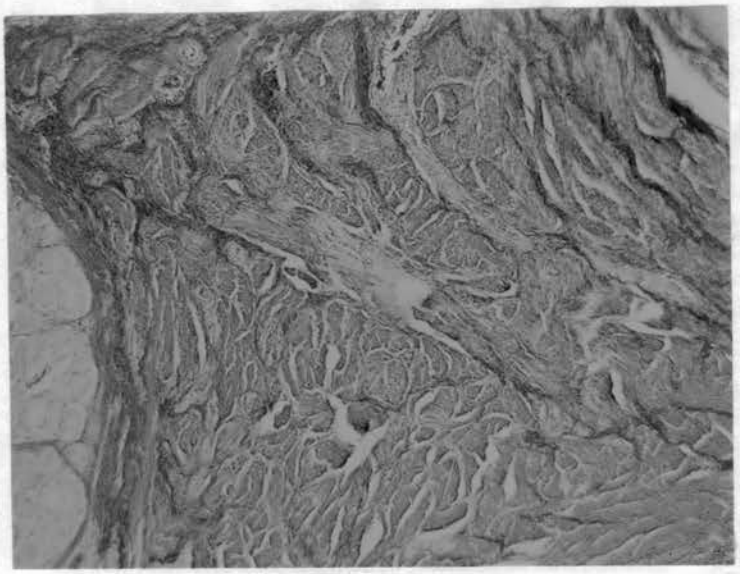


Fig. 33

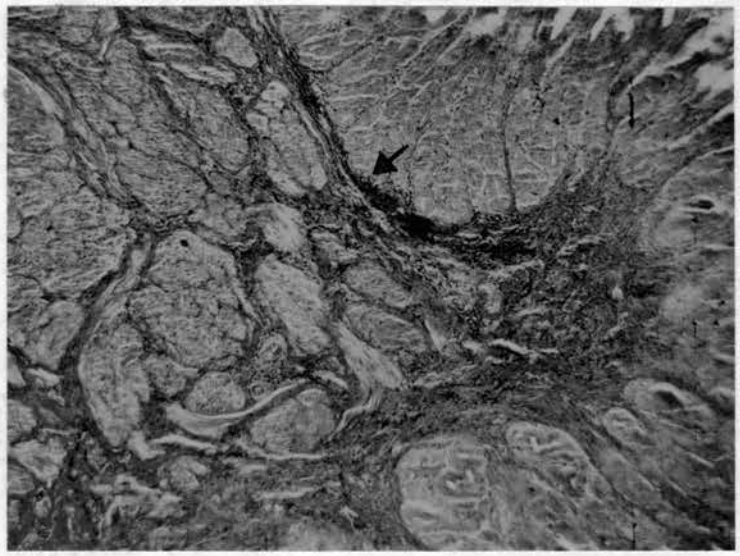


Fig. 34

Plate XXI

Fig. 35. Elastic fibers in the torus pyloricus longitudinalis of a pig ten days of age.

Orderly orientation of the elastic fibers (black strands) toward the glands (top of figure). x 100.

Comment.

Elastic fibers are less abundant in the components of the pyloric canal than collagenic fibers. Elastic fibers consist of polysaccharides of the hyaluronic acid type and their sulphuric acid esters. The fibers do not readily regenerate.

Fig. 36. Section of liver of a pig at birth (normal control).

Note the absence of fat droplets in the liver cells and the variation of density of the cells. The latter is caused by the quantity of glycogen present in the individual liver cell. x 450.
Bauer-Feulgen stain.

Plate XXI

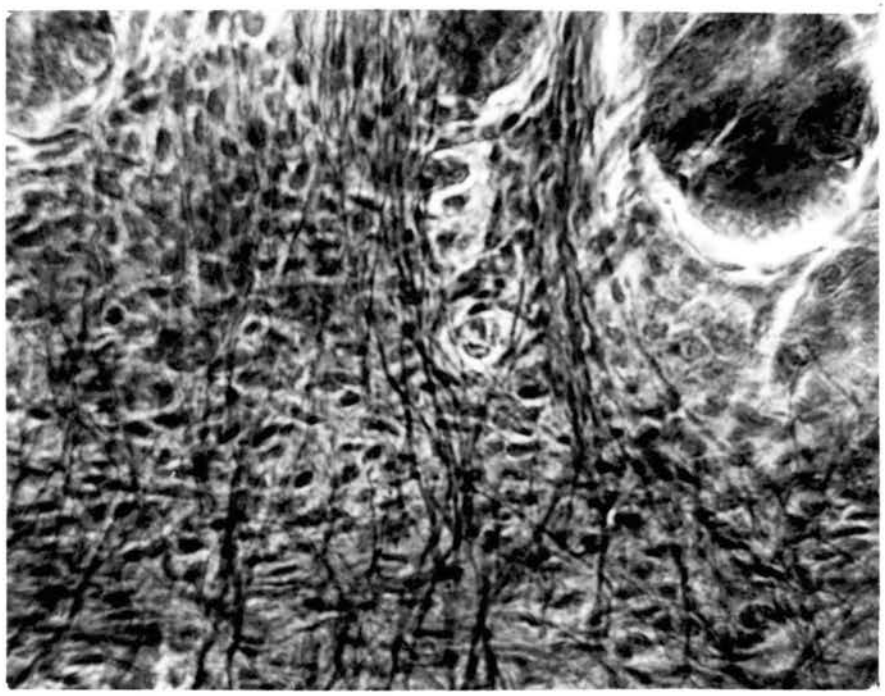


Fig.35

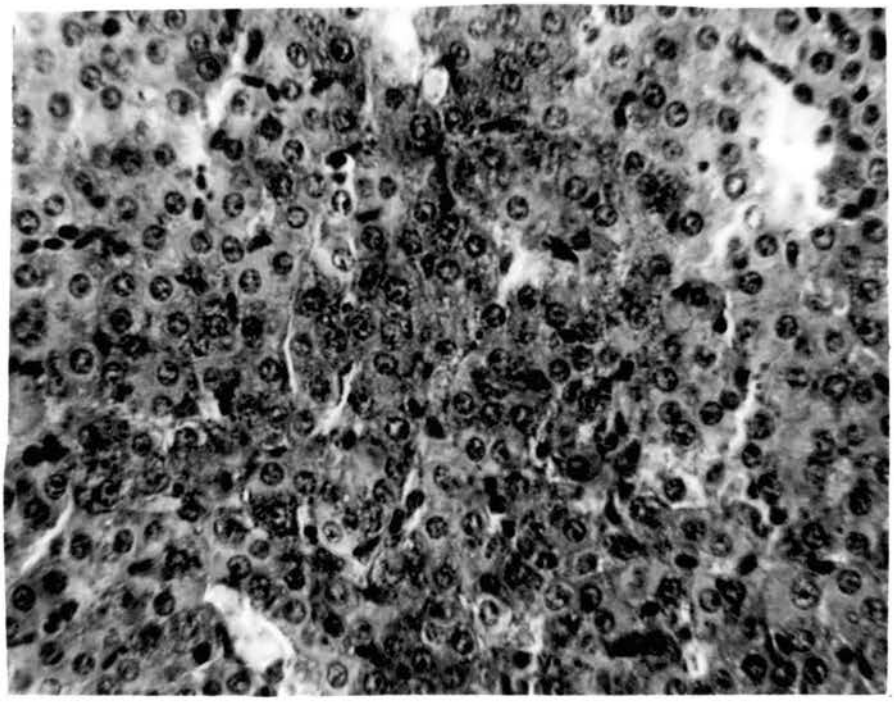


Fig.36

Physiology of the Stomach of the Normal Newborn Pig

Gastric Emptying Time

The gastric emptying time was determined with the aid of radio-opaque media in five full-term naturally-farrowed and five full-term pigs obtained by Cesarean section. To assure ourselves of reasonably reliable data on the gastric emptying time we made a preliminary study of the capacity of the stomach immediately after birth. The weight of the five naturally-farrowed pigs was ascertained before and after their first nursing period. The term "nursing period" stands for the act of lactation of the sow, which according to Hughes and Hart (1935) lasts about 30 seconds but which Gill and Thompson (1956) found to range from 14.2 to 49 seconds. Our preliminary data revealed that under the conditions prevailing at the time our observations were made the newborn pigs ingested from 30 to 55 grams of colostrum during a single nursing period. The amount ingested seemingly depended very much on the vitality of the pig and the environment prevailing at the time of suckling. Transcribing the quantity of colostrum ingested into "stomach capacity" the capacity of the stomach of the newborn pig is analogous to that of the newborn infant, which according to Smith (1945) has a capacity between 30 to 60 ml. He, however points out that an estimation of the gastric capacity is futile because of the flexibility of the stomach. We have focused attention on the fact that natural aberrancies of the contour and the size of the stomach make it very difficult to establish a gross anatomical norm for this organ. Adding to this handicap the fact that small amounts of milk and radio-opaque media leave the stomach within a few minutes after ingestion, one realizes that ascertaining the stomach capacity is a trying

matter indeed. Further, it must be remembered that factors such as size of the udder and the teat, the quantity of colostrum in the udder and the rate of flow of the colostrum, the ability of the pig to hang on to the teat and its suckling response (normal, greedy, or poor), and the compatibility of the colostrum play an important role as far as ingestion of colostrum during a single nursing period is concerned. Gill and Thompson (1956) noted that extreme changes in the environmental temperature influence the suckling response of the pig. These investigators found that at a temperature range between 30 to 70° F. (0 to 21° C.) suckling pigs ingested from 33.4 to 57.4 grams of milk during a single nursing period.

For the radiological demonstration of the gastric emptying time of the stomach of the pig at birth we used 50 ml. of a radio-opaque media consisting of 30 grams of barium sulphate, 30 ml. of Pet milk, and 30 ml. of water. The amount of 50 ml. was chosen because it seemed to be a representative quantity ingested by normal vigorous pigs during one nursing period. The radio-opaque media was administered with the aid of a plastic bottle fitted with a rubber nipple about the size of a teat. The five pigs obtained by Cesarean section were used for this study. The plastic bottle made it possible to squirt the media into the mouth when the suckling response became poor. Immediately after ingestion of the radio-opaque media, a left and a right oblique lateral and a dorsal-ventral radiograph of the abdominal region was made. The same series of radiographs was made every half hour for two consecutive hours and one series twenty-four hours after ingestion of the radio-opaque meal. The first half hour radiographs revealed presence of media in the intestines. At the end of the two-hour observation period no media was present in

the stomach. All of the media was expelled from the intestines in twenty-four hours. It is noteworthy that the ordeal caused no ill effects. No constipation resulted from the radio-opaque media.

In summary, it may be said that, although the number of pigs studied is small, our data allow one to presume that normally the stomach empties within two hours. Euthanasia of runt pigs two and three hours after suckling strengthened our radiological data pertaining to the emptying time of the stomach of pigs at birth.

It is not within the scope of this thesis to detail the various radiological techniques employed in our study. The reader, however, may obtain information pertaining to the techniques from the Section of Radiology, School of Veterinary Medicine.

Volume of Gastric Juice

The gastric juice of newborn pigs consists of gastric gland secretions, mucus, surface epithelium, swallowed amniotic fluid, regurgitated duodenal fluid, and bile.

For the estimation of the volume of gastric juice at birth five pigs were used ranging in age from a few minutes to two hours. The pigs were well anesthetized, the abdomen was opened, and a suture was placed about the gastric portion of the esophagus and the proximal part of the duodenum. The stomach was then removed, and its contents were allowed to drain into a graduate cylinder. With this method the amount of gastric juice ranged from 0.5 to 10 ml. with an average of 4.2 ml.

Free Hydrochloric Acid

The gastric juices of the preceding investigation were tested for

the presence of free hydrochloric acid. For this study Töpfer's reagent (0.5 percent alcoholic solution of dimethylamino-azobenzene) was used. No free hydrochloric acid could be demonstrated in the gastric juice samples with this reagent. The negative data were checked against gastric secretion samples obtained from three runt pigs which were given 0.1 ml. of a 1:1000 solution of histamine sulphate parenterally. No free hydrochloric acid was secreted under the influence of histamine, which according to Conway (1953) is a specific stimulator of parietal cells.

Thus our data support those of Kvasnitskii and Bakeeva (1940), who reported the absence of free hydrochloric acid in the gastric juice of newborn pigs.

pH of Gastric Juice

Determination of the pH of the gastric juice was made from the same samples on which volume and free hydrochloric acid determinations were made. For ascertaining the pH the Beckman hydrogen ion meter was employed. With this apparatus the gastric juice of the pig at birth ranged in pH from 2.0 to 3.0. It is to be kept in mind that these pH values represent actually the combined acidity of the organic acids rather than that of the gastric juice containing free hydrochloric acid. The presence of the latter gives the gastric juice a pH of from 0.5 to 1.0 and when mixed with mucus (Guyton, 1956) gives the juice a pH of 1.5 to 2.0.

It is noteworthy that the stomach of the pig at birth contains a variable amount of strongly alkaline mucus, which may readily neutralize small amounts of free hydrochloric acid secreted by the parietal cells. According to Babkin (1944) mucus has a pH of from 7.0 to 8.0.

Pepsin and Rennin

Samples from the gastric juices obtained for the previously cited determinations were used for ascertaining the pepsin and rennin content of the gastric juice of newborn pigs.

Pepsin

The activity of the enzyme was tested with Mett's egg white method. No pepsin activity could be demonstrated. Addition of hydrochloric acid to the test sample failed to induce pepsin activity. According to Kleiner (1954) pepsin has a maximum activity at pH 1.5 to 2.2.

Rennin

The milk coagulation test was employed for detecting rennin activity. With this test a strong positive reaction for rennin or a rennin-like substance was obtained. It is noteworthy that rennin activity occurred in gastric juices the pH of which ranged from 2.0 to 3.0. It is generally believed that a pH of 5.3 is necessary for optimal rennin activity.

In summary, it may be said that our data differ from those of Kvasnitskii and Bakeeva (1940) by demonstrating the absence rather than the presence of pepsin in the gastric juice of newborn pigs. Since the proenzyme pepsinogen requires the presence of free hydrochloric acid for conversion to pepsin, it thus would seem logical that rennin, which is active without the acid, should be the dominating proteolytic enzyme in the gastric juice of newborn pigs. Kleiner (1954) states that no rennin is present in adult human gastric juice. According to Bockus (1949) the proenzymes for pepsin and rennin are secreted by the pyloric glands, and thus in the absence of free hydrochloric acid in the gastric juice of the newborn infant the enzyme rennin should be present. It may well be possible that the presence of the absence of rennin suggests "species

specificity." In other words, there may be specific rennins which do a specific job yet to be demonstrated in both animal and man.

Physical Character of Milk Curds

The absence of free hydrochloric acid and pepsin in the gastric juice of newborn pigs and the presence of rennin or a rennin-like substance suggest that ingested milk is coagulated by the enzyme rennin or a closely related substance. Wolman (1946) states that the makeup of milk curds is conditioned by the composition of the gastric secretions, local motility of the stomach musculature, and temperature and inherent property of the milk itself. He noted that pepsin in the presence of hydrochloric acid tends to coagulate milk (pH 6.6) into small soft curds, whereas rennin in the absence of the acid produces large and sometimes hard curds. Wolman is of the opinion that large hard curds may be packed into masses too large to be pressed through the pyloric canal or to be vomited.

A preliminary study of the physical character of the curds in the stomach of the newborn pigs revealed small soft curds ranging in size from less than one millimeter to two millimeters in diameter. Keeping in mind that rennin coagulates human milk into corase, large, and hard curds, our data may suggest that species specific rennins exist, the purpose of which is to change the casein molecule into a readily digestible form particularly important for the fast growing pig.

It is noteworthy that the milk mass in the stomach of our series of newborn pigs with pyloric canal stenosis was made up of two layers. The outer layer consists of various sized and more or less packed soft and hard curds and an inner cohesive, dry, and hard layer (core). This physical make-up of the milk mass seems to be the result of two forces,

(a) packing of curds into large masses by the contractions of the stomach musculature (deep peristalses), which also remove the liquid portion of the mass, and (b) flowing of recently ingested milk about the hard core. Thus, one may deduce from the size and the physical appearance of the milk mass, the approximate onset and the degree of pyloric canal stenosis.

To learn whether hard granular material passes the pyloric canal readily we coagulated sow colostrum and cow milk in vitro with dilute hydrochloric acid. It is noteworthy that either milk formed large, hard curds. The curds were fixed in a formol-saline fixing solution, washed in distilled water, and submerged in waterproof India ink until stained deep-black. The curds were allowed to air-dry. The size of the curds ranged from less than one millimeter to five millimeters. Ten grams of these curd grains were suspended in water, and the mixture was squirted into the mouth of a newborn pig with the aid of a plastic bottle fitted with a large-holed rubber nipple. Two hours after ingestion of the grains the pig was euthanized. The stomach and the intestines were opened, and the distribution of the curd grains was noted. Most of the grains were well advanced in the intestines. A small coagulated mass of grains well coated with mucus was overlying the gastric orifice of the pyloric canal, and a small quantity of grains was embedded in the surface epithelium and in the pits of the glands of the concave curvature.

The findings suggest that hard grains pass the pyloric canal, but formation of grain masses may suggest that under certain conditions ingested milk may be coagulated into hard masses too large to be forced through the pyloric canal or to be vomited. Retention of the milk masses may encourage accumulation of injurious metabolic products from bacterial activities and degeneration of proteins. These products may bring

about aberrations of local chemical mechanisms that create a specific chemical lesion which may play an important role in progressive pyloric stenosis.

Blood Sugar Level

The blood sugar concentration of three groups of pigs, ten to each group, was determined with the LaMotte micro-blood sugar method. A blood sugar concentration of 50 mg. percent was considered indicative of hypoglycemia, for at this level and below this level loss of consciousness and coma resulted. The range and average blood sugar concentration of the three groups of pigs are recorded in Table 3. The average blood sugar concentration of Group I (at birth) of 102 mg. percent compares well with the mean of 102 mg. percent of twenty-six newborn pigs studied by Morrill (1952 b). The range and average blood sugar concentration in Group II (five days old) and of Group III (ten days old) agree with Morrill's figures for suckling pigs, the blood sugar of which ranged from 74.58 to 144.64 with a mean of 114.2 mg. percent.

Liver Glycogen

Because we were not immediately concerned with the problem of establishing a normal chemical range of the quantity of glycogen in the liver of newborn pigs, we made only a visual survey of the liver glycogen with the aid of histochemical methods. The latter allows a rough estimate of the glycogen concentration in liver cells. This information was considered adequate for the purpose of getting an idea of the glycogen content of the liver cells and the degree of glyconeogenesis. The plus and minus signs in the liver column in Table 4 represent,

TABLE 3

Range and Average Blood Sugar Concentration of Normal Newborn Pigs, at Birth, Fifth, and Tenth Postnatal Day in mg./100 ml. of Whole Blood

Group	Day of Postnatal Life	Range mg.	Average mg.	Number of Pigs
I	At Birth	80-150	108.0	10
II	Fifth Postnatal Day	90-150	110.0	10
III	Tenth Postnatal Day	75-120	101.0	10

therefore, a visual expression of the glycogen concentration, which does not necessarily reflect the actual quantity of glycogen in the liver. The four plus sign stands for the presence of glycogen in all liver cells within the boundary of the square of the Whipple eyepiece micrometer disc. The latter was used in combination with an eye piece 10 x and objective 10 x (16 mm.). The microscopic survey called for examination of ten successive microscopic fields of liver sections 5 micra in thickness. In Fig. 36 is shown an example of normal liver with the glucogen evenly distributed throughout the liver parenchyma.

The histochemical methods employed for the demonstration of glycogen in liver sections were those of Bauer-Feulgen cited by Cowdry (1948 b) and Mitchell and Wislocki (1944).

Gross Anatomy of the Stomach of the Newborn Pig with Hypertrophic Pyloric Canal Stenosis

Pylorus

In general, the pylorus is dilated. The degree of the latter is dependent upon the size of the milk mass and the amount of gas present in the stomach. We found the average thickness of the lesser curvature musculature of the pylorus to be 1.0 mm. (normal 1.3 mm.), and that of the greater curvature 2.3 mm. (normal 1.6 mm.). The difference in thickness of the pylorus musculature is shown in Fig. 46. The mucosa shows no gross evidence of gastritis or edema though hyperemia is common. Most mucosal folds (plicae mucosae) are obliterated.

Pyloric Canal

The pyloric canal as defined in this thesis is partially or completely

stenosed. Stenosis occurs either at the duodenal sphincter (Figs. 39, 57 B) or involvs both the duodenal and gastric sphincter with concomitant obliteration of the concave curvature (Figs. 40, 43 B, 46). If one consults Figs. 38, 43 A, which show transverse sections, and Fig. 47, which shows a longitudinal section of normal pyloric canals, one can readily understand the stenotic state of the pyloric canal.

The completely stenosed pyloric canal is grossly cylinder-like (Fig. 45) and has a stretched appearance (Fig. 46) and a fibromatous consistency. The latter is in sharp contrast to the normal pyloric canal, which is soft and flexible. The increased thickness of the greater curvature musculature is more prominent than in the pylorus. The increase of thickness of the pyloric canal musculature is caused by hypertrophy of the fibers of the middle circular and, to a lesser extent, the inner oblique muscle coat. The outer longitudinal muscle coat is involved only to a slight extent, if at all. This lack of participation of the muscle in the hypertrophic state may be explained on the basis that the muscle is not uniformly distributed over the pylorus and pyloric canal (Fig. 23), is not well developed at the time the fibers of the middle circular muscle coat undergo hypertrophy, and in many areas is made up of only a few muscle strands. It is, however, understood that the factors just cited may not play a role as far as the noninvolvement of the outer longitudinal muscle is concerned.

The fibromatous consistency is the result of an extensive hyperplasia of the connective tissue, particularly of collagenic fibers (Figs. 50, 52, 53). Concomitantly with the increase of thickness of the pyloric canal musculature the concave curvature becomes obliterated

(Fig. 46 c). The contraction of large masses of connective tissue causes stretching of the pyloric canal. The latter lengthens from 0.5 to 1.0 mm. depending on the developmental stage of the stomach, the anatomical make-up of the pyloric canal, the degree of stenosis, and the number of days the stenotic state existed.

It is of interest to note that Horwitz, Alvarez, and Ascanio (1929) calculated the thickness of the pyloric canal muscle in children from one to eight years old. They found the thickness to range from 2.6 mm. to 4.8 mm., the former figure in a one-year-old infant, and the latter in a child six years old.

Plate XXII

Fig. 37. Longitudinal section through the pyloric canal of the stomach of a pig at birth.

1, Horizontal plane at level of the head of the torus pyloricus longitudinalis and proximal portion of the gastric sphincter (gastric orifice of the pyloric canal); 2, Horizontal plane at the level of the body of the torus pyloricus longitudinalis and concave curvature; 3, Horizontal plane at the level of the tail of the torus pyloricus longitudinalis and duodenal sphincter (duodenal orifice of the pyloric canal). x 5.

Fig. 38. Transverse sections 2 mm. thick through planes 1, 2, and 3 (Fig. 37) of the pyloric canal of the stomach of a pig at birth (normal control).

Top. Transverse section through plan 1 (gastric sphincter).
a, Head of torus pyloricus longitudinalis. Pyloric canal lumen is patent.

Middle. Transverse section through plane 2 (concavature).
a, Body of torus pyloricus longitudinalis; b, Neck (base) of torus pyloricus longitudinalis. The neck gives the structure a mushroom-like appearance. Pyloric canal lumen is patent.

Bottom. Transverse section through plane 3 (duodenal sphincter).
a, Tail of torus pyloricus longitudinalis. Pyloric canal is patent. x 5.

Plate XXII

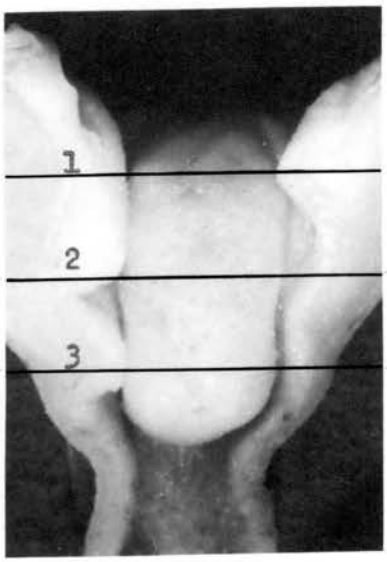


Fig.37

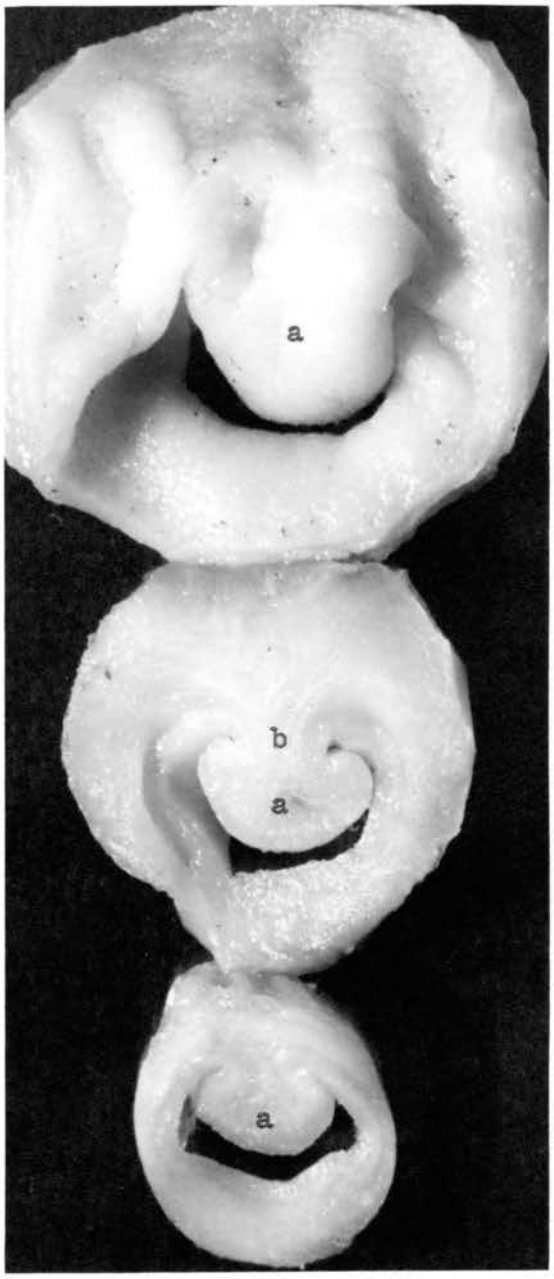


Fig.38

Plate XXIII

Fig. 39. Transverse sections 2 mm. thick through plane 1, 2, and 3 (Fig. 37) of the pyloric canal of the stomach of a pig six days of age with particl pyloric canal stenosis (pig 3, fall 1955).

Top. Transverse section through plane 1 (gastric sphincter). a, Head of torus pyloricus longitudinalis; b, Underdeveloped semicircular mucosal fold; c, Longitudinal mucosal folds. Pyloric canal lumen is patent.
Middle. Transverse section through plane 2 (concave curvature). a, Body of torus pyloricus longitudinalis. Note that the longitudinal mucosal folds are obliterated. Pyloric canal lumen is partially obstructed (stenosed).
Bottom. Transverse section through plane 3 (duodenal sphincter).
a, Tail of torus pyloricus longitudinalis. Pyloric canal lumen is completely stenosed. x 6.

Comment.
The partial stenosis was caused by an irregular hypertrophy of the middle circular muscle coat, connective tissue septa, and pyloric canal glands. This localized stenosis adds to the mystery of what starts and limits hypertrophy.

Fig. 40. Transverse sections 2 mm. thick through plane 1, 2, and 3 (Fig. 37) of the pyloric canal of the stomach of a pig one day of age with complete pyloric canal stenosis (pig 1, spring 1956)

Top. Transverse section through plane 1 (gastric sphincter). a, Head of torus pyloricus longitudinalis, b, Partial developed semicircular mucosal fold; c, Longitudinal mucosal folds. Pyloric canal lumen is stenosed.
Middle. Transverse section through plane 2 (concave curvature). a, Body of torus pyloricus longitudinalis; b, Bulging of the greater curvature mucosa (mucous membrane). Pyloric canal lumen is stenosed.
Bottom. Transverse section through plane 3 (duodenal sphincter). a, Tail of torus pyloricus longitudinalis. Pyloric canal lumen is stenosed. x 6.

Comment.
The pig (Fig. 40) never suckled. It became comatose about twenty hours after birth. The blood sugar level was 75 mg. percent. The pig failed to respond to glucose therapy.

Histologic sections revealed hypertrophy of the middle circular muscle coat and the pyloric canal glands (Figs. 47, 48).

The gastric juice contained no bile. Because a tenacious bile-stained mucus plug was present in the duodenum, it was presumed that the stenotic state was manifested in utero. It is not likely that a birth stress pylorospasm produced the pathology of the pyloric canal.

Plate XXIII

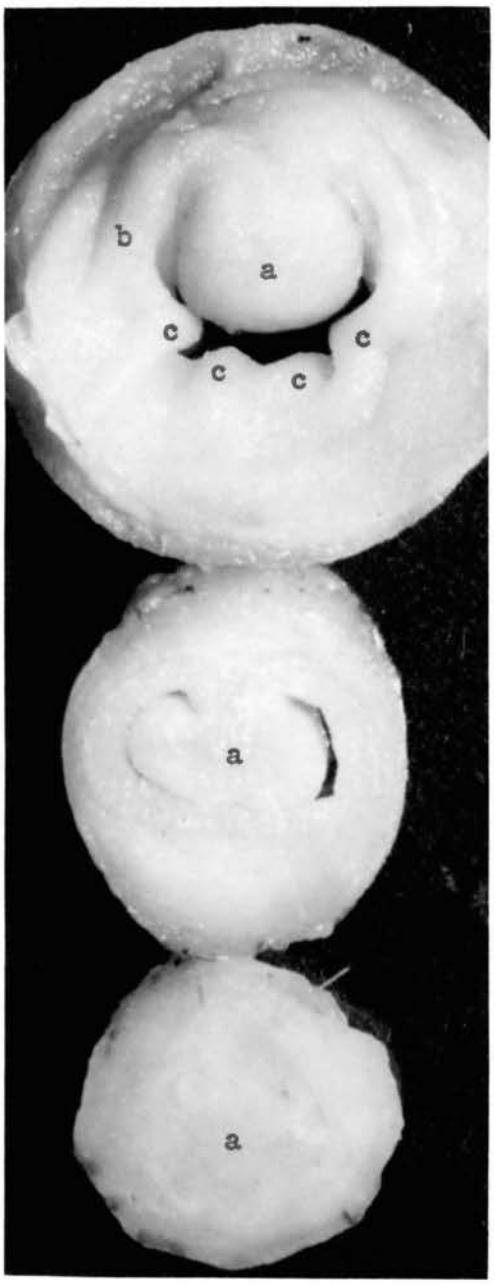


Fig.39

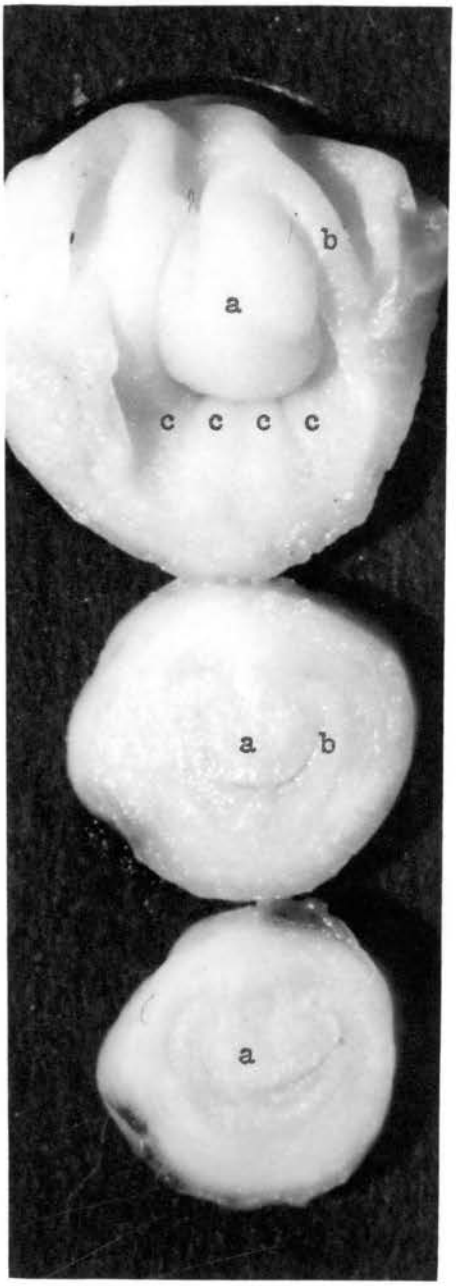


Fig.40

Plate XXIV

Fig. 41. Oblique lateral radiograph of the stomach of a pig six days of age with partial pyloric canal stenosis, twenty-four hours after ingestion of 50 ml. radio-opaque media (pig 3, fall 1955).

Note the shape and position of the stomach. The arrow is in the airspace of the stomach and points at the meniscus of the barium (black mass). Small amounts of barium have escaped from the stomach and are scattered throughout the intestines (arrows left of stomach).

Fig. 42. Oblique lateral radiograph of the stomach of a pig six days of age, twenty-four hours after ingestion of 50 ml. radio-opaque media (normal control).

Absence of barium throughout the entire gastro-intestinal tract.

Comment.

On the basis of the retention of practically all of the barium in the stomach after twenty-four hours and absence of barium from the gastro-intestinal tract of the control pig, a diagnosis of pyloric canal stenosis was made (pig 3, fall 1955).

The pig was given 1.0 ml. of 1:10,000 atropine solution, to be repeated every five hours. The animal became moribund, however, seven hours after the radiograph was made. At autopsy the gastric sphincter (gastric orifice of the pyloric canal) was found to be in a relaxed state; that is, the lumen was patent, but the duodenal sphincter was completely stenosed. It is not likely that the atropine (2 ml.) the animal received before death brought about a relaxation of the gastric sphincter musculature. The muscle fibers of the latter were, however, somewhat less hypertrophied than those in the duodenal sphincter. The possibility of a pylorospasm may be ruled out on the basis of the hypertrophy of the muscle fibers, connective tissue, and glands.

Potter (1953) states that pylorospasm is not associated with hypertrophy of the musculature.

Plate XXIV

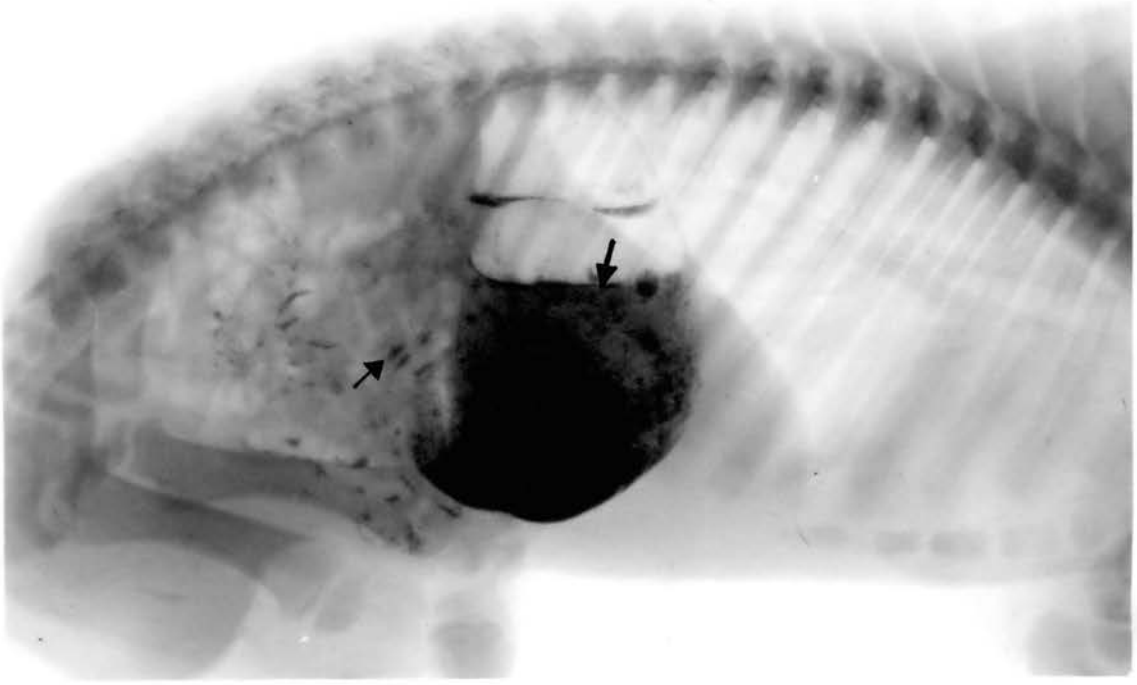


Fig.41

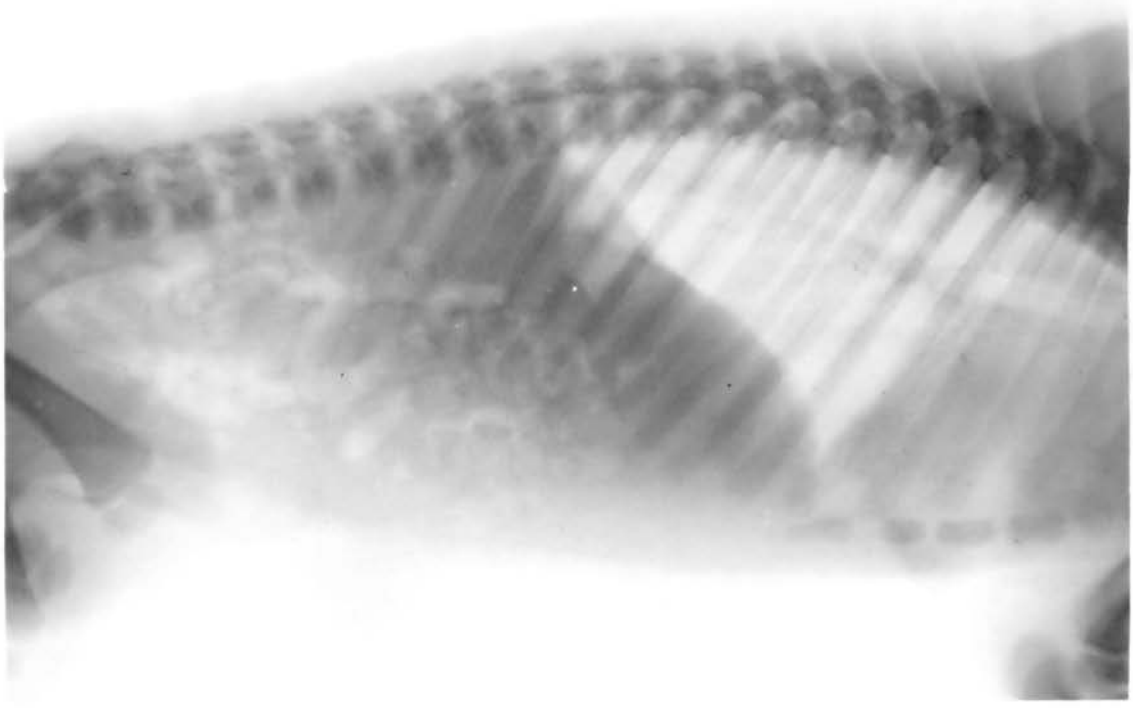


Fig.42

Plate XXV

Fig. 43-A. Transverse sections 2 mm. thick through the midportion of the gastric and duodenal sphincter of the pyloric canal of the stomach of a pig six days of age (normal control).

Top. Transverse section through the gastric sphincter. a, Middle circular muscle coat; b, Torus pyloricus longitudinalis; c, Longitudinal mucosal folds. Pyloric canal is patent.

Bottom. Transverse section through the duodenal sphincter. a, Middle circular muscle coat; b, Torus pyloricus longitudinalis; c, Longitudinal mucosal fold; d, Bulge of greater curvature musculature forming duodenal sphincter. Pyloric canal lumen is patent. x 5½.

Fig. 43-B. Transverse sections 2 mm. thick through the midportion of the gastric and duodenal sphincter of the pyloric canal of the stomach of a pig seven days of age with pyloric canal stenosis (pig 5, spring 1956).

Top. Transverse section through the gastric sphincter. a, Hypertrophic middle circular muscle coat; b, Torus pyloricus longitudinalis; c, Longitudinal mucosal folds. Pyloric canal lumen is stenosed.

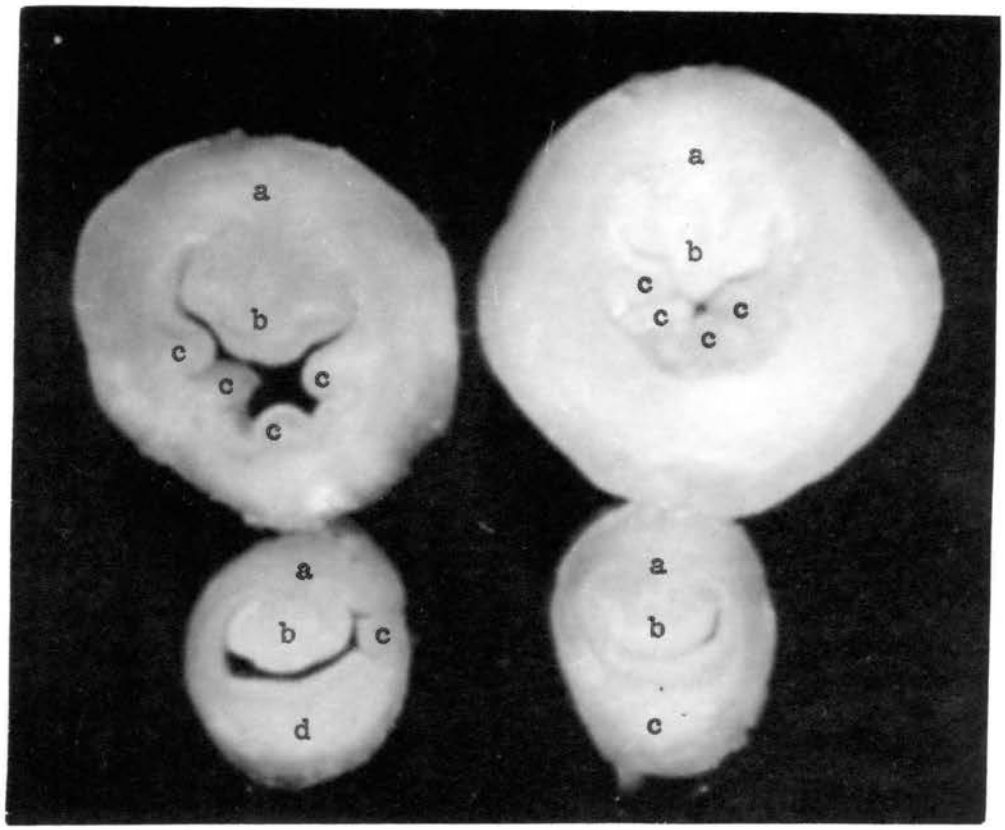
Bottom. Transverse section through the duodenal sphincter. a, Hypertrophic middle circular muscle coat; b, Torus pyloricus longitudinalis; c, Hypertrophic greater curvature musculature forming duodenal sphincter. Pyloric canal lumen is stenosed. x 5½.

Comment.

Note the striking difference in the diameter of the normal control (Fig. 43-A) and stenosed pyloric canal (Fig. 43-B). The increased size of the latter is the sequel to hypertrophy of the fibers of the middle circular muscle coat and the mucosa. The diameter of the stenosed pyloric canal exceeded by 1 mm. the diameter of five normal controls.

Sauer (1924) made wax models (reconstructions of wax serial sections) of stenosed pyloric canals obtained at autopsy and found that in the infant pyloric canal stenosis resulted from hypertrophy of the pyloric musculature.

Plate XXV



A B

Fig.43

Plate XXVI

Fig. 44. Oblique lateral radiograph of the stomach of the pig eleven days of age with pyloric canal stenosis, twenty-four hours after ingestion of 50 ml. of radio-opaque media (pig 6, spring 1956).

Note the balloon-like appearance of the stomach and air filled diverticulum ventriculi. Dense black area in the transverse stomach is caused by settled barium. Arrow points at pyloric canal. No barium is in the intestines.

Comment.

Apathy, poor suckling response, and a prominent potbelly drew attention to the pig on its eight postnatal day. The pig was given 50 ml. of warm water to which charcoal was added as a marker. No charcoal was present in the small amounts of feces passed during the following two days. Thus, a tentative diagnosis of intestinal obstruction was made. A mixture of barium, water, and milk was given until the animal vomited. Fluoroscopic examination revealed that the stomach was large, almost globular. The barium mixture appeared to flow about a mass but failed to enter the duodenum. The radiograph (Fig. 44) made twenty-four hours later definitely suggested obstruction in the pyloric canal rather than in the intestines. This interpretation of the radiograph was substantiated at autopsy (Fig. 46).

Buckstein (1948) states that hypertrophic pyloric stenosis in the human produces a characteristic radiograph. The stomach is greatly dilated (cup-shaped), and only a little barium may escape into the small intestine during twenty-four hours.

Plate XXVI

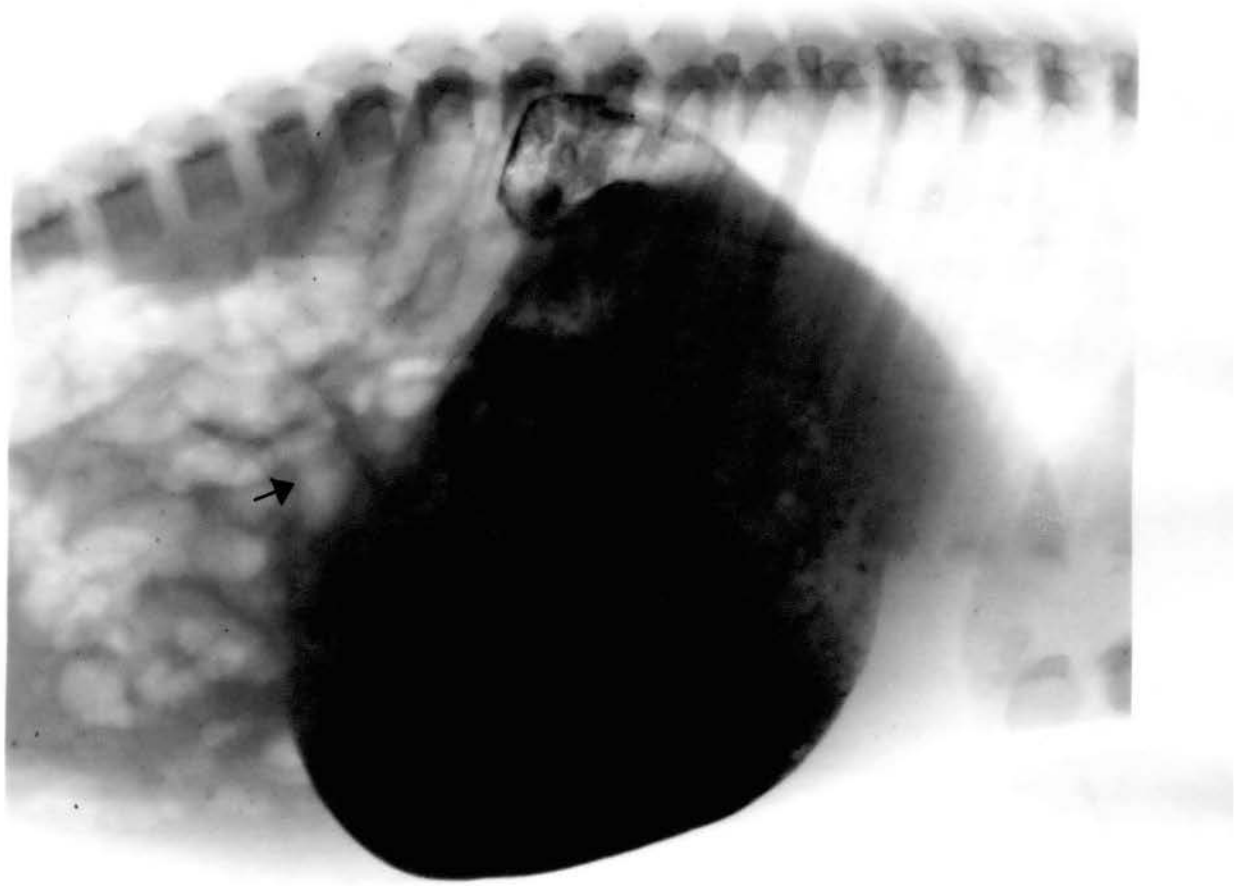


Fig.44

Plate XXVII

Fig. 45. Transverse stomach fixed in situ of the pig eleven days of age with pyloric canal stenosis (pig 6, spring 1956).

Note the cylinder-like shape of the pyloric canal (white arrows), globular appearance of the transverse stomach and engorged blood vessels. x 2.

Comment.

The pyloric canal had a fibromatous consistency. The latter resulted from hypertrophy of the pylorus musculature (Fig. 46) and hyperplasia of the connective tissue chiefly of the collagenic fibers (Figs. 52, 53).

Plate XXVII

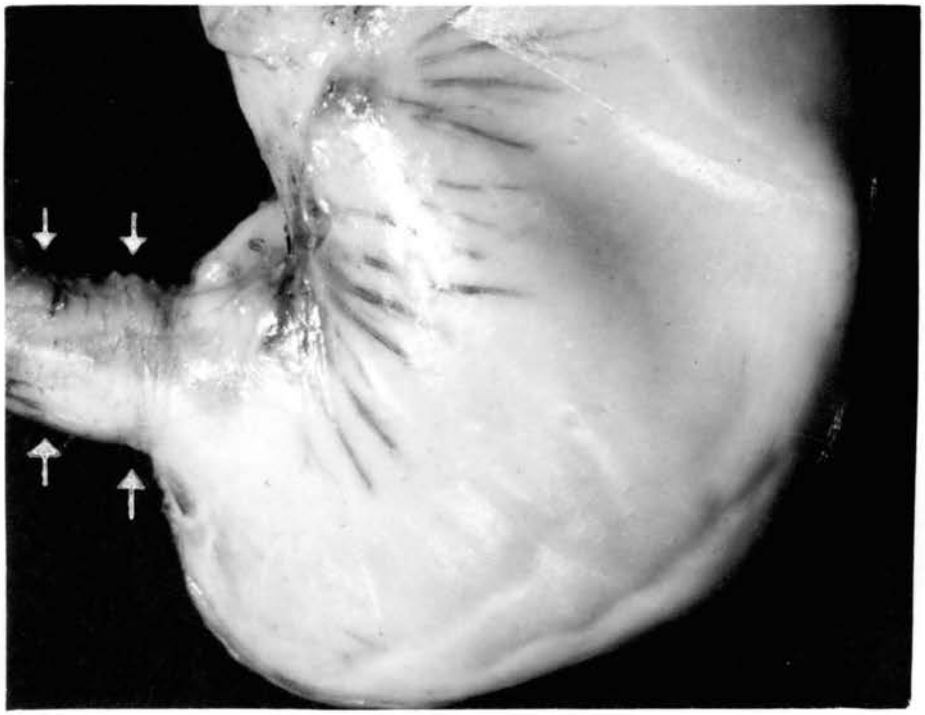


Fig.45

Plate XXVIII

Fig. 46. Transverse section through the distal part of the pyloric vestibule (antrum) and pyloric canal of the stomach of the pig eleven days of age with complete pyloric canal stenosis (pig 6, spring 1956).

a, Torus pyloricus longitudinalis; b, Gastric sphincter; c, Obliterated concave curvature; d, Duodenal sphincter; e', Pylorus greater curvature musculature; e'', Pylorus lesser curvature musculature; f, Proximal part of duodenum; g, Stretched wall of pyloric vestibule. Pyloric canal is stenosed. x 6.

Fig. 47. Transverse section through the pyloric canal of the stomach of a pig ten days of age (normal standard of comparison, Fig. 2).

a, Torus pyloricus longitudinalis; b, Gastric sphincter; c, Concave curvature; d, Duodenal sphincter. x 6.

Comment.

The pyloric canal (Fig. 46) is stretched. The gastric sphincter (b), the convave curvature (c), and the duodenal sphincter (d) are not distinguishable. Individual variations in the degree of occlusion of the canal lumen, thickening of the musculature, prominence of the longitudinal mucosal folds in the canal lumen, hyperplasia of the connective tissue, and hypertrophy of the mucosa seem to correspond with the age of the pig, the developmental stage of the stomach, and onset and the severity of the gastric lesion. No specific inflammatory changes were present in the mucosa. Muscle fiber hypertrophy and extensive hyperplasia of the connective tissue (Fig. 52, 53) were the most conspicuous lesions in all stenosed pyloric canals. The length of the pyloric canal was 11 mm. (normal range at same age 8-10 mm.), and the width from the greater to the lesser curvature was 11 mm. (normal range at same age 8-10 mm.).

Plate XXVIII

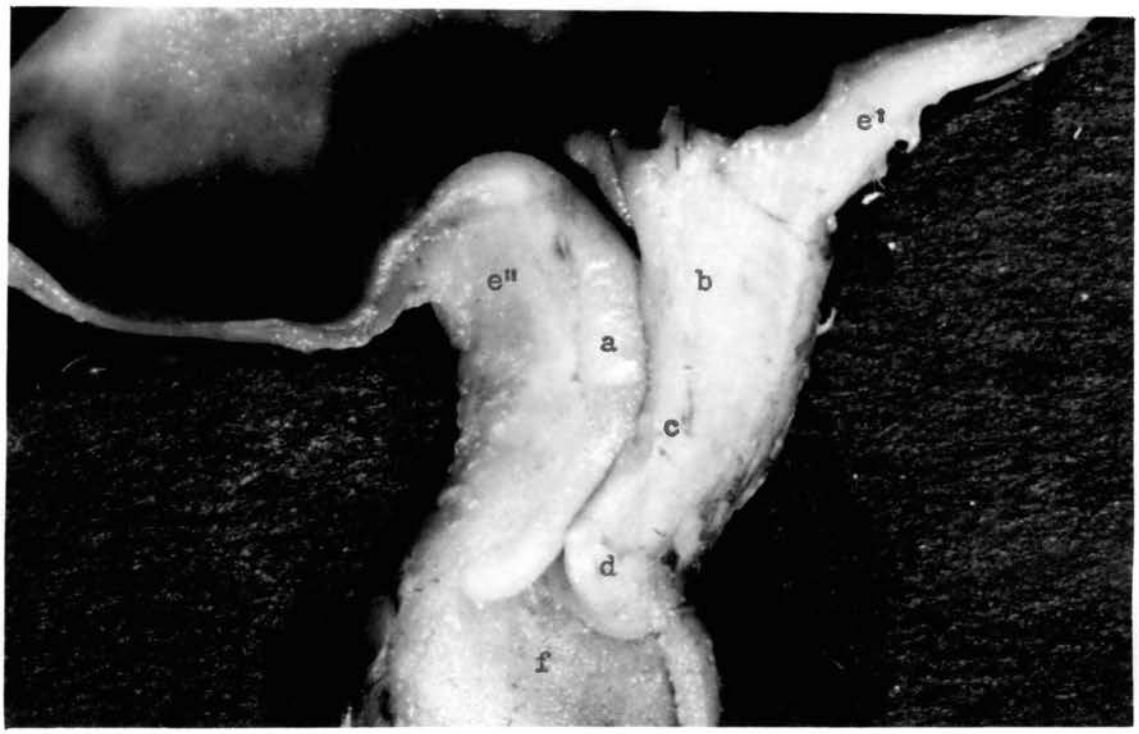


Fig.46

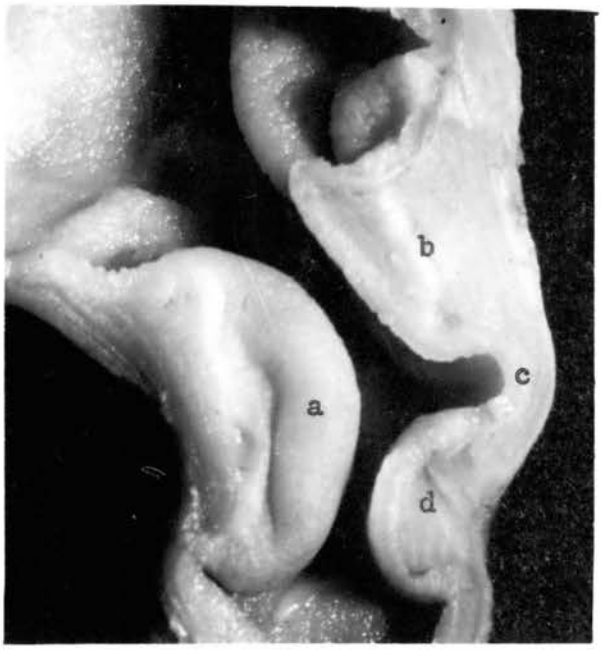


Fig.47

Microscopic Anatomy of the Stomach of the Newborn Pig with Hypertrophic Pyloric Canal Stenosis

Pylorus

In all pigs with pyloric canal stenosis the pylorus musculature showed an irregular hypertrophy of the fibers of the middle circular and inner oblique muscle coats. No specific lesion, however, was seen which could be considered the underlying etiologic factor responsible for the muscle fiber hypertrophy.

Pyloric Canal

The most striking histopathology of the components of the pyloric canal were (a) an irregular hypertrophy of the fibers of the middle circular and inner oblique muscle coats and (b) a conspicuous hyperplasia of the connective tissue, particularly of the collagenic fibers (Figs. 50, 52, 53). The increase of connective tissue makes it readily understandable why the pyloric canal has a fibromatous consistency. Further, if one accepts the supposition that coarse broad bands, cords, and sheets of dense connective tissue reflect immobility, then contraction of large masses of connective tissue may not only make the pyloric canal very rigid but may narrow the canal lumen to such a degree that none or very little chyme can pass into the intestine. Thus accumulation of ingested milk in the stomach will eventually lead to the formation of a cohesive milk mass too large to be expelled from the stomach by way of vomiting or through the pyloric canal lumen by accelerated peristalses.

The absence of the outer longitudinal muscle coat on the lesser curvature and patchy absence of the muscle coat on the greater curva-

ture is noteworthy. Torgersen (1942) found that absence of the outer longitudinal muscle coat at the lesser curvature is characteristic of the pig stomach, and Horton (1928) observed patchy absence of the muscle coat on the greater and lesser curvature in infants less than one year of age. A longitudinal muscle coat hiatus on the greater curvature of the stomach of the newborn pig is shown in Fig.23 d. Horton comments on the marked individual variation of the muscle coats making up the musculature of the pyloric canal in normal human stomachs but states that in general the middle circular muscle coat has a thickness of about twice that of the outer longitudinal muscle coat. A similar difference in thickness of the muscle coats may be seen in the pyloric canal of the newborn pig (Fig. 24). Thus the striking enlargement of the middle circular muscle coat in hypertrophic pyloric canal stenosis in the newborn pig and in the infant may be explained on the basis that the muscle coat has normally a greater thickness, which is enhanced by the hypertrophy of its fibers.

The seemingly simultaneous muscle and connective tissue fibrogenesis may well find its explanation in the fact that in the digestive tract of the pig smooth muscle arises in common with the interstitial connective tissue from mesenchymal syncytia (McGill, 1909). In this common syncytium soon after the muscle forms, collagenic fibers arise, and at a still later stage elastic fibers develop. It may be hypothesized that this generic sequence may well account for the variation of the fiber components of the connective tissue as well as the quantity of the latter in the components of the pyloric canal.

The mucosa revealed no specific lesion that could be interpreted as the cause of the pathologic musculo-connective tissue fibrogenesis. The hypertrophy of the pyloric canal glands (pig 1, spring 1956) shown in Fig. 49 is not clearly understood. It may be hypothesized that the consistent

absence of inflammatory changes in the mucosa may suggest disturbed inherited chemical mechanisms within the glands caused by an abnormal environment or a nutritional deficiency state yet to be clarified.

Plate XXIX

Fig. 48. Transverse section through the midportion of the head of the gastric sphincter of the pyloric canal of the stomach of a pig one day of age with pyloric canal stenosis (pig 1, spring 1956).

a, Normal middle circular and inner oblique muscle coat; b, Hypertrophic middle circular muscle coat; c, Normal outer longitudinal muscle coat. Arrows point at morphologically normal Auerbach plexus ganglia. x 110.
Schleicher's connective tissue stain.

Fig. 49. Longitudinal section through the mucosa (mucous membrane) of the pyloric canal of the stomach of a pig one day of age with pyloric canal stenosis (pig 1, spring 1956).

The glands are distinctly hypertrophic. (Compare with normal pyloric canal glands (Fig. 26). x 250.
Schleicher's connective tissue stain.

Comment.

Because of the age of the pig and the absence of a gross and microscopic inflammatory lesion in the pyloric vestibule and pyloric canal we believe that pig 1, spring 1956, is an example of in utero pyloric canal stenosis. The gross anatomy of the pyloric canal is presented in Fig. 40.

Plate XXIX

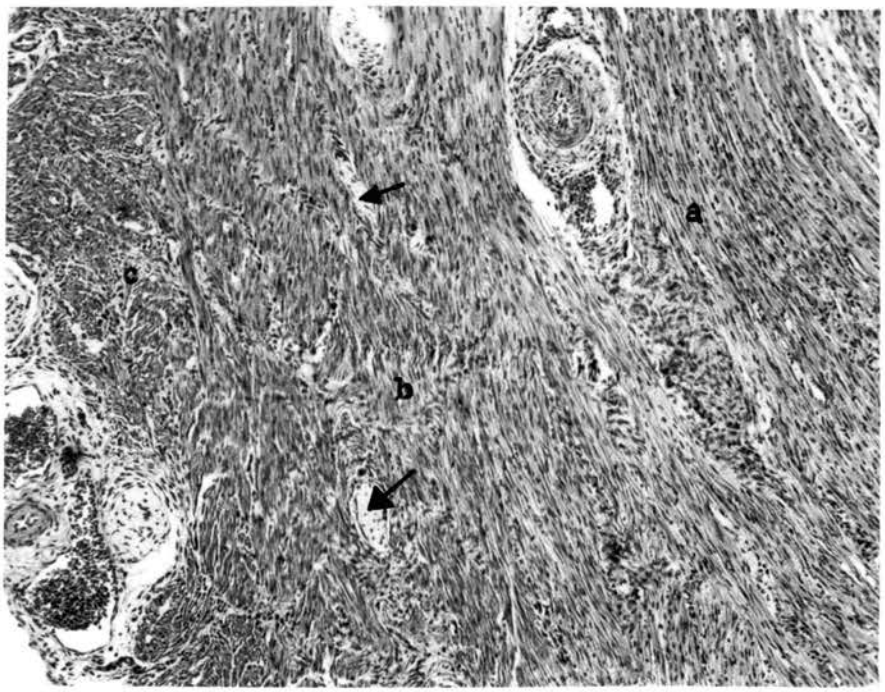


Fig.48

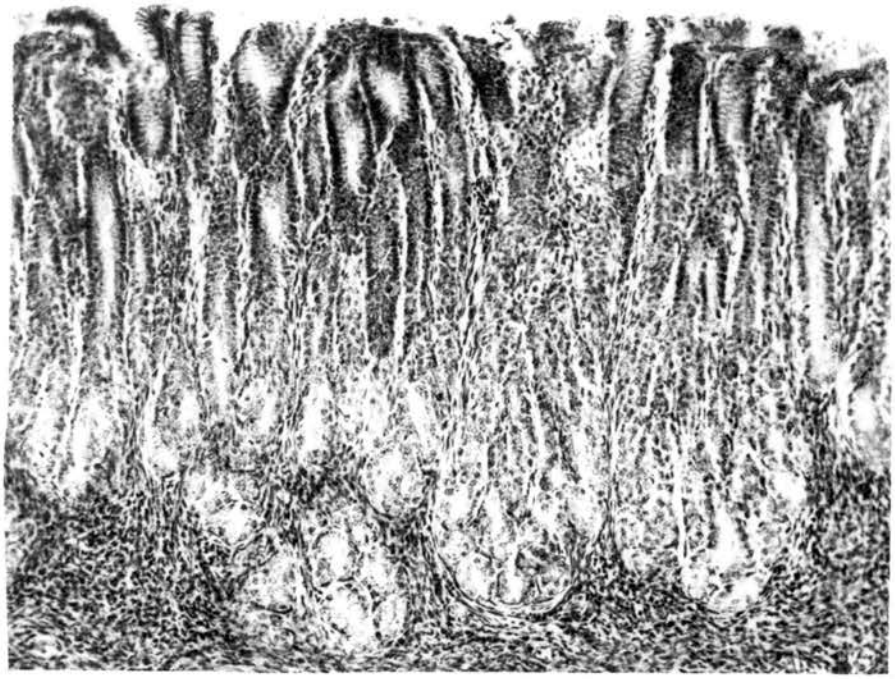


Fig.49

Plate XXX

Fig. 50. Collagenic fibers. Longitudinal section through the mucosa (mucous membrane) of the greater curvature of the pyloric canal at the level of the concave curvature of the pig eleven days of age with pyloric canal stenosis (pig 6, spring 1956).

Note the orientation, marked waviness and variation of thickness of the collagenic fibers. Very fine strands of reticular fibers are interwoven with the collagenic fibers. The latter are quantitatively increased. x 500.
Dublin's silver stain.

Fig. 51. Elastic fibers. Longitudinal section through the mucosa (mucous membrane) of the greater curvature of the pyloric canal at the level of the concave curvature of the pig eleven days of age with pyloric canal stenosis (pig 6, spring 1956).

Quantitatively the elastic fibers are within the normal range, but are thicker than normal and oriented in all directions. See normal elastic fiber pattern in Fig.35.

Comment.

Niero (1947) is of the opinion that in pyloric canal stenosis in the human an increase of elastic fibers contributes to the over-all muscular hypertrophy. We were not able to confirm Niero's observation in our series of newborn pigs with pyloric canal stenosis.

It has been pointed out in the text of this thesis that the degree of abundance of collagenic, elastic, and reticulum fibers determine in part local mobility and immobility.

Randall and Jackson (1953) state that collagenic and reticular fibers are derived from the extra-cellular protein matrix and that no distinct chemical difference exists between the two fibers.

Plate XXX

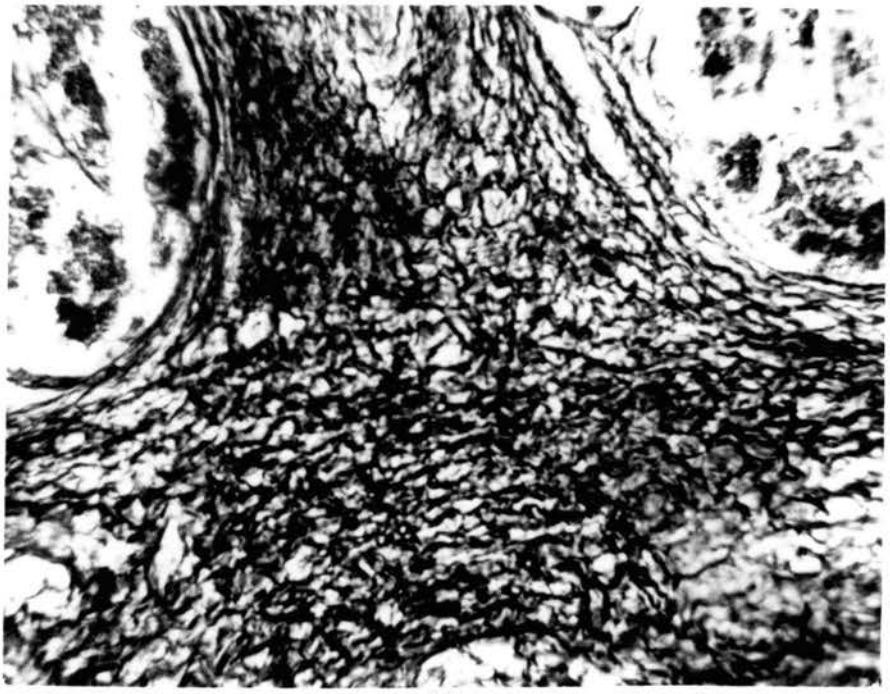


Fig.50

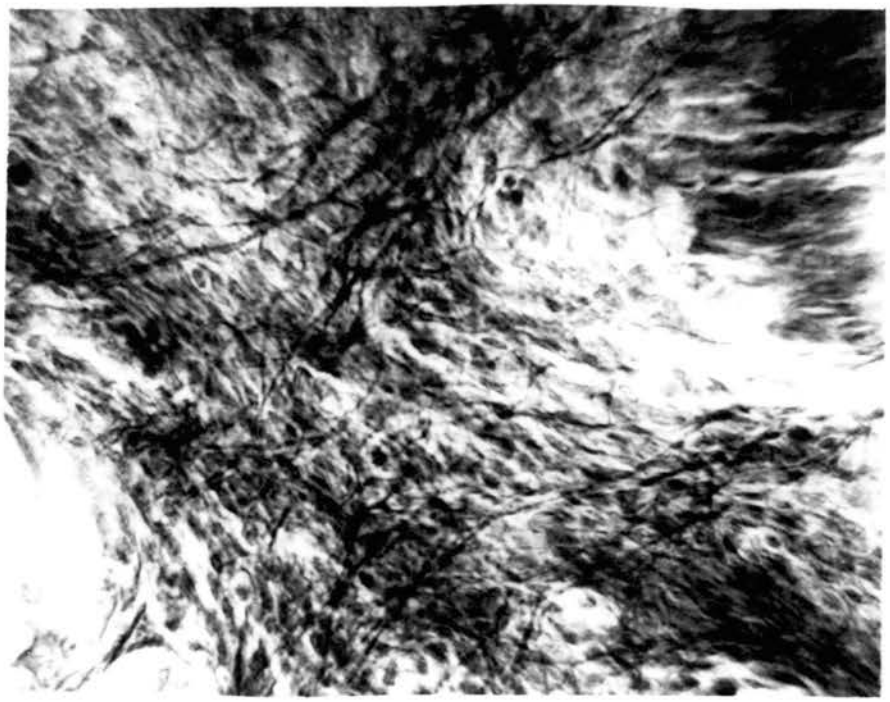


Fig.51

Plate XXXI

Fig. 52. Average connective tissue septal pattern in the greater curvature of the pyloric canal at the level of the concave curvature in newborn pigs with pyloric canal stenosis

Distinct increase in the quantity and thickness of the connective tissue septa and hypertrophy of the muscle fibers of the middle circular coat. x 360. Schleicher's connective tissue stain.

Fig. 53. Connective tissue sheets and septa in the body of the torus pyloricus longitudinalis in newborn pigs with pyloric canal stenosis.

The musculature is partitioned into irregularly sized bundles. It is however, difficult in many areas in the torus pyloricus longitudinalis to differentiate between normal and abnormal fibrogenesis because of the great variability of connective tissue growth present at birth and rate of development of fibrous tissue in postnatal life. x 360.
Schleicher's connective tissue stain.

Comment.

Randall and Jackson (1953) state that fibrogenesis is controlled by chemical processes in the protein matrix which is an extracellular substance of great complexity. Fibrogenesis is as yet not clearly understood. It may thus be hypothesized that, if these chemical processes become altered to such a degree that a "chemical lesion" results, the latter may be the causative factor responsible for the pathological connective tissue hyperplasia and hypertrophy of the muscle fibers. This appears to be a logical sequence of events since connective tissue and muscle fibers have a common matrix.

Plate XXXI

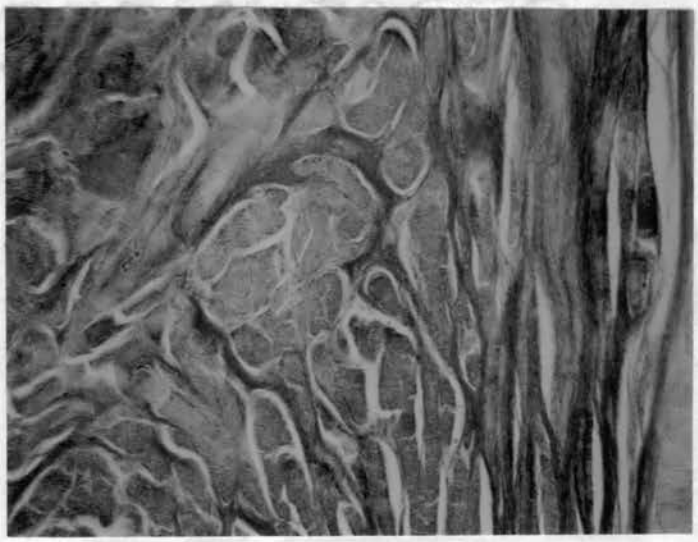


Fig. 52

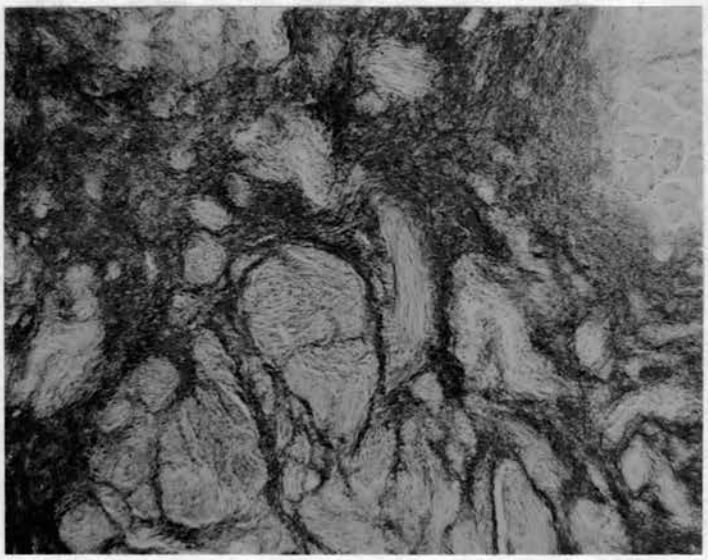


Fig. 53

Plate XXXII

Fig. 54. Section of liver of the pig eleven days of age with pyloric canal stenosis (pig 5, spring 1956).

The liver section is representative of the parenchymatous disease that may result from malnutrition caused by pyloric canal stenosis. In the photomicrograph may be seen cloudy swelling, intracellular edema (hydropic degeneration), and severe fatty metamorphosis. A few normal liver cells are present. The Bauer-Feulgen stain revealed severe glycogen deficiency. x 90.
Schleicher's hematoxylin-eosin stain.

Comment.

Cloudy swelling is the mildest form of cell degeneration resulting from the presence of very fine precipitated proteins (granules), and intracellular edema is the sequel of increase of water. These two parenchymatous disturbances are considered early stages of fatty metamorphosis. Hypoglycemia may result from extensive fat retention. The latter prevents the liver from sustaining the blood sugar because of inability to handle carbohydrates produced by the process known as neoglycogenesis.

Lichtman (1949) states that liver fat is not readily altered. Thus conversion of the liver fat into carbohydrates is not likely to occur at a beneficial rate.

Plate XXXII

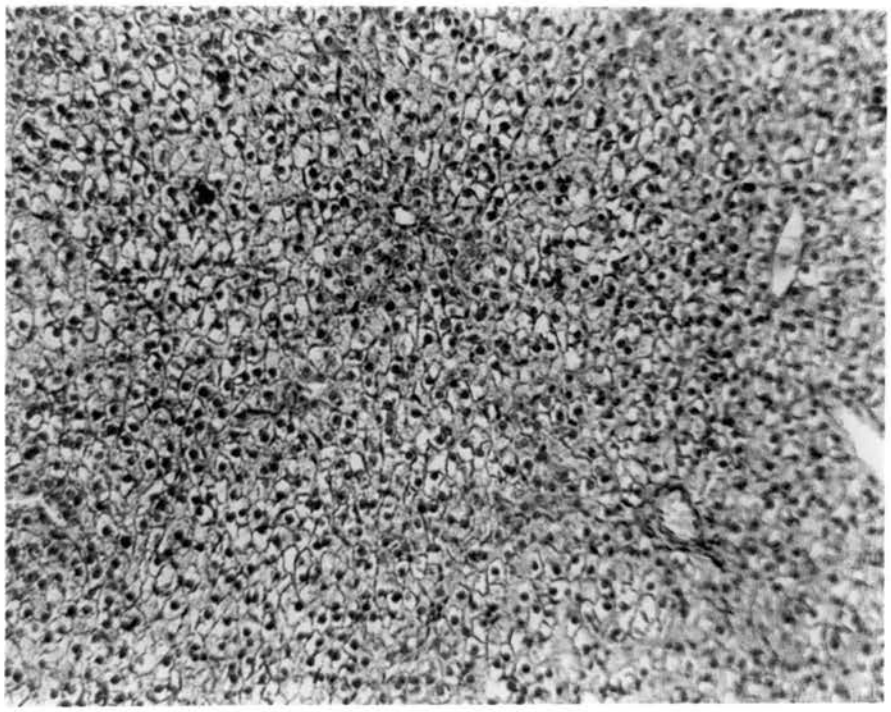


Fig.54

Physiology of the Stomach of the Newborn Pig with Hypertrophic Pyloric Canal Stenosis

Gastric Emptying Time

The gastric emptying time was ascertained in three pigs (pig 3, fall 1955; pigs 5 and 6, spring 1956). The study was carried out with the aid of radio-opaque media and radiographs made every hour for six consecutive hours and twenty-four hours after the ingestion of the media. The radiographic study revealed that practically all of the media was still present in the stomach at the twenty-four hour check. The stomach was balloon-shaped (Fig. 44), which is considered by Buckstein (1948) to be characteristic of pyloric stenosis in the human subject. Pressure applied to the abdominal region to cause flow of media into the intestine was met with failure. The procedure apparently caused the pigs great discomfort as evidenced from their violent struggle to free themselves and the high pitch of their squeal.

Free Hydrochloric Acid

No free hydrochloric acid could be demonstrated in the small amounts of gastric juice obtained by the needle aspiration technique from the stomach of the four pigs with pyloric canal stenosis diagnosed during life (Table 4).

pH of Gastric Juice

The pH of the gastric juice of the specimens used for the preceding test ranged from 3.0 to 5.5.

It is interesting to note that Marriot and Davidson (1923) found the gastric content in the newborn infant at the height of breast-

milk digestion to range from 3.0 to 4.0.

Pepsin and Rennin

Examination of the gastric juice samples used for the preceding tests gave positive evidence for rennin only.

Physical Character of the Milk Curd

The milk curd was removed from the stomach after complete fixation of this organ in situ during life and before fixation of the stomach in the case of natural death of the pig. The weight of the milk masses ranged from 10 to 180 grams (Table 4). The somewhat cohesive masses were made up of a hard core which was covered by thick and tenacious mucus. One to several loosely packed layers of milk curd surrounded the core. In only three instances was bile present in the core, but no bile was detectable in the layers, suggesting that regurgitation of bile ceased at the time the hard core formed. Whether pylorospasms preceded the stenotic state is conjectural. Should pylorospasms have occurred, however, the amount of bile entering the pyloric vestibule was so small that the methods used for detection of bile were not sufficiently sensitive or the bile was completely destroyed at the time the milk mass was examined.

It is interesting to note that according to Ladd, Ware, and Pickett (1946) absence of bile in the vomitus of newborn infants is indicative of pyloric canal stenosis.

Blood Sugar Level

The blood sugar level before death, either by euthanasia or natural

death of the ten pigs (Table 4) ranged from less than 50 mg. to 80 mg. percent of whole blood. It is interesting to note that only half of the pigs, one to eleven days of age, had a blood sugar above 50 mg. percent. The latter concentration was considered the upper glycemia range.

Liver Glycogen and Fat

Glycogen

The liver glycogen was determined by the same optical and histochemical method used for the estimate of the glycogen quantity in the liver of the normal newborn pig. In practically all pigs with pyloric canal stenosis the liver glycogen was reduced (Table 4). It is, however, understood that the visual estimate of the glycogen quantity is subject to personal error and thus can not be considered a reliable method for the determination of the glycogen concentration in the whole liver, though the procedure was the only way open to us at the time this phase of investigation was conducted.

Fat

The liver fat was determined by the same optical method used for the determination of the liver glycogen in normal newborn pigs. The non-fixed liver sections 100 micra thick were stained with Sudan III and IV. The data presented in Table 4 revealed that fatty metamorphosis of the liver cells does not necessarily follow glycogen depletion or that fatty metamorphosis is proportional to the degree of glycogen deficiency. For example, the glycogen was severely reduced in the liver

of pig 3, fall 1955, yet there was no fatty metamorphosis. The latter, however, was severe in the liver of pig 5, spring 1956 (Fig. 54), which had a low liver glycogen concentration.

Sebrell and Harris (1954) point out that ascorbic acid (vitamin C) deficiency tends to favor fatty metamorphosis. Thus the extraordinary richness of this vitamin in sow's colostrum may be a biological necessity during the early life of the newborn pig. It is interesting to note that Braude, Kon, and Thompson (1946) and Braude et al., (1947) who reported the high concentration of ascorbic acid in sow's colostrum, remark that the reason for the presence of this vitamin ranging in quantity from 12 to 36 mg. with an average of 23.8 mg. per 100 ml. of colostrum is not clear.

Ladd, Ware, and Pickett (1946) found that ascorbic acid deficiency occurs in hypertrophic pyloric stenosis in the human.

Behaviorism of the Newborn Pig with Hypertrophic pyloric canal stenosis

To recognize hypertrophic pyloric canal stenosis in the field the observer must become acquainted with the behavior pattern of the normal newborn pig. Thus a brief discussion of the behaviorism of the normal newborn pig is presented here to allow the novitiate to identify the sick pig and make clinical and laboratory studies along the line presented in this thesis.

It is generally agreed that the newborn pig has a poorly developed fat layer and body temperature-regulating mechanism (Newland et al., 1952). This lack of protective insulation and thermal control is expressed by

the tendency of the newborn pig to seek warmth wherever it can find it. Thus normal newborn pigs huddle and compete with each other for a warm place even when in an electrically-heated brooder. The normal pig does not particularly mind being laid over by littermates and, therefore, seldom leaves the huddle because of this action.

In general, the newborn pig is alert and playful. Its haircoat is soft, smooth, and lustrous (Bailey, 1955). It is readily aroused by the call of the sow and moves quickly toward the source of food. It fights vigorously for the teat, his by virtue of priority, should an aggressive littermate dare claim the teat. It suckles with little interruption, and its suckling response may range from greedy to normal within a minute's time simulating very much the suckling response of the newborn infant (Norval, 1946). When the milk flow ceases, the pig either leaves the udder abruptly, keeps on suckling, or tries other free teats. It has a tendency to urinate when leaving the sow. The act completed, it looks for a warm place to rest, which generally amounts to huddling with littermates.

The pig afflicted with hypertrophic pyloric canal stenosis may display a normal behavior pattern during the first few hours or days of its life. The first visible sign of illness is its objection to be laid over by littermates. The pig leaves the huddle rather than engage in a struggle for comfort. It may either stand around, drowsily and shivering, or wander aimlessly about the pen. It eventually joins or settles down away from its littermates. It is not readily aroused by the sow's call. Thus the good teats are occupied by littermates before it gets to the sow. It may fight for a teat or suckle on any free teat, generally the last pair of rear teats, which give less milk and thus are not

particularly challenged by the stronger members of the litter. It may suckle greedily or after a few short and feeble suckling movements may fall asleep with the teat in its mouth. Tenacious saliva may exude from its lips. The pig gives the impression that it has spent its strength. It makes no attempt to suckle even when the udder is hand massaged and the teat is moved about in its mouth, but it will swallow milk squirted in the back of its mouth. It misses nursing periods because of sleeping a great deal. While sleeping, it shivers continuously even when in an electrically heated brooder. It does not play spontaneously or take part in coarse frolicking of its littermates. It may or may not become potbellied. Urine excretion may amount to only a few drops. The feces consist chiefly of mucus. The haircoat becomes rough and lusterless. Apathy, extreme drowsiness, hypothermia, and complete loss of consciousness precede death. The terminal state may or may not be accompanied by a severe hypoglycemia (Table 4).

The terminal symptoms just mentioned correspond to those of baby pig disease. Thus it is readily understandable why a pig with hypertrophic pyloric canal stenosis may elude recognition and is placed in the nonspecific category of baby pig disease.

Two methods, however, aid in the isolation of the pig with hypertrophic pyloric canal stenosis during life from pigs with baby pig disease, (a) giving milk mixed with charcoal and checking the feces for the presence of this marker and (b) ascertaining the gastric emptying time with the aid of radiography. Finally the stenotic state of the pyloric canal lumen can be clearly demonstrated by transverse or longitudinal sectioning of the pyloric canal at autopsy.

In Table 4 are presented the physical, biochemical and histochemical data of ten newborn pigs with hypertrophic pyloric canal stenosis.

TABLE 4

Physical, Biochemical, and Histochemical Data of Ten Newborn Pigs with Hypertrophic Pyloric Canal Stenosis

Pig No.	Breed	Sex	Age at Death (Days)	Weight at Birth (lbs.)	Weight at Autopsy (lbs.)	Size of Litter	Litter and Boar Identification	Milk Mass in Stomach (gm.)	State of Pyloric Canal at Autopsy	Rectal Temp. °F. before Death	Blood Sugar mg./100 ml.	Liver Glycogen*	Liver Fat #
							Fall 1955						
1	Dur.	F	5	1.6	3.2	12	20-21 (L-185)	30	Stenosed	95.6	75	++	+
2	B-I	F	5	2.0	3.3	15	40-42 (82)	11	Stenosed	94.8	-50	+	+++
3&	B-I	M	6	2.3	4.2	15	40-43 (82)	86	Stenosed	94.0	-50	+	-
4&	X-B	F	8	2.9	2.5	10	4-10 (9-X)	80	Stenosed	95.7	75	++	++
							Spring 1956						
1	Hamp.	M	1	2.0	2.5	13	5-11 (Long.)	30	Stenosed	94.7	75	++	-
2	Hamp.	F	3	1.5	1.0	17	24-14 (Mac)	10	Stenosed	94.0	-50	+	+++
3	Pol.	F	4	1.8	2.8	11	14-8 (Clim.)	15	Stenosed	94.0	-50	++	+++
4	Hamp.	F	6	2.0	2.8	11	8-9 (Long.)	55	Stenosed	94.0	75	++	+++
5&	Chest.	M	7	1.9	2.0	13	8-10 (Whitel.)	56	Stenosed	94.0	-50	+	++++
6&	Hamp.	M	11	2.6	2.0	17	24-11 (Mac)	80	Stenosed	94.0	80	+++	+

*+ Indicates quantity of glycogen estimated from ten liver sections (fixed) 5 micra thick.
#+ Indicates quantity of fat estimated from ten liver sections (unfixed) 100 micra thick.
& Pyloric canal stenosis diagnosed during life.

V. OTHER PATHOLOGICAL CONDITIONS INVOLVING THE STOMACH AND PYLORIC CANAL IN NEWBORN PIGS

Atony of the Stomach and Obstruction of the Pyloric Canal by a Tenacious Plug of Mucus in a Full-term Stillborn Pig

The pig examined was a male pig weighing 3.2 pounds at birth. With the exception of a prominent potbelly the animal was well developed. When the abdomen was opened the potbelly was noted to be due to a huge thin-walled stomach filled with fluid. With the aid of a needle aspiration method, 123 ml. of a turbid fluid were obtained. The fluid, which had a pH of 2.5, contained no free hydrochloric acid, pepsin, or bile, but the presence of rennin was demonstrated. From the gross appearance of the skin and other vital organs it seemed that death occurred shortly before or during birth. The intestines contained dark mucilaginous material (meconium) and air.

The stomach was fixed in situ and opened along the greater curvature. The wall of the stomach was so thin that the millimeter divisions of a ruler could be seen through the stomach wall. Tenacious mucus covered the orifice of the gastric sphincter and filled the lumen of the pyloric canal and part of the duodenum. It has been pointed out in this thesis (see legend to Fig. 12) that normally in the fetal pig the gastric sphincter is covered by tenacious mucus which seals the pyloric canal lumen and that this mucus seal seems to liquefy sometime before birth. Thus it may be presumed that the mucus plug has a definite

function during the early development of the gastro-intestinal tract. Failure of the plug to undergo resolution, therefore, leads to the accumulation of gastric secretions and swallowed amniotic fluid.

It is interesting to note that in the newborn infant a condition known as "congenital atonic motor insufficiency of the stomach" may manifest itself. This clinical entity is believed caused by a dearrangement of the intrinsic Auerbach-Meissner's plexuses.

The distal part of the pyloric vestibule and the longitudinal section of the pyloric canal with the mucus obstruction removed are presented in Figs. 55, 56.

Plate XXXIII

Fig. 55. Transverse section through the distal part of the pyloric vestibule of the stomach of a full-term stillborn pig with atony of the stomach.

a, Head of torus pyloricus longitudinalis; b, Aberrant semicircular mucosal fold; c', Greater curvature; c", Lesser curvature. Arrow points at plug of mucus sealing the orifice of the gastric sphincter and pyloric canal lumen. x 3.

Fig. 56. Longitudinal section through the pyloric canal of the stomach of a full-term stillborn pig with atony of stomach (Fig. 55).

a, Torus pyloricus longitudinalis; b, Gastric sphincter; c, Concave curvature; d, Duodenal sphincter; e, Semicircular mucosal fold; f, Duodenum; g, Duodenal papilla; h', Greater curvature musculature; h", Lesser curvature musculature. x 6.

Comment.

This full-term stillborn male pig weighed 3.2 pounds at birth. It was the only casualty in a litter of twelve. It had a prominent potbelly caused by 123 ml. of fluid in the stomach resulting from a tenacious plug of mucous occluding the pyloric canal lumen. After fixation of the stomach in situ the plug was removed. The pyloric canal had a well formed lumen. No specific aberrancies of the microscopic anatomy of the components of the pyloric canal were found.

Plate XXXIII

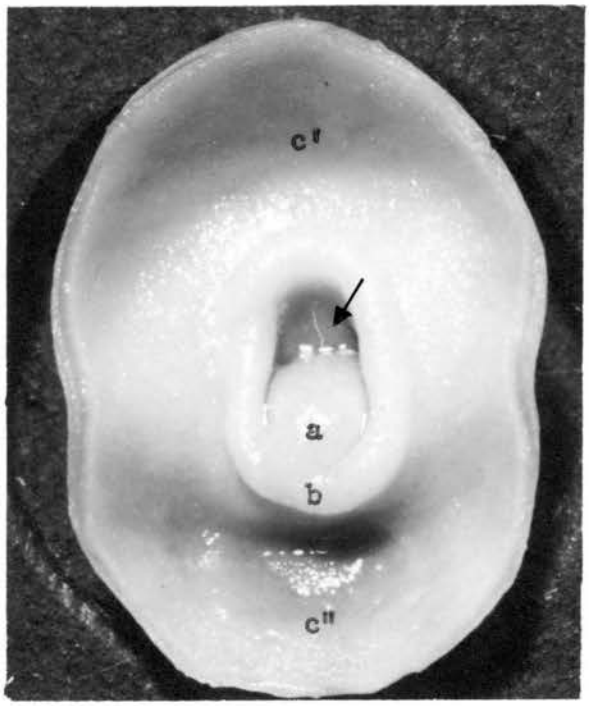


Fig.55

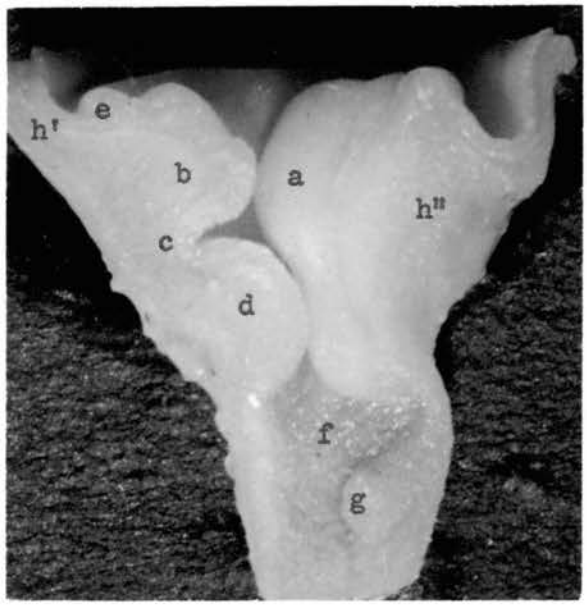


Fig.56

Atrophy of the Greater Curvature Musculature of the Pyloric Canal in a Pig Three Days of Age

The pig examined was a female weighing 2.3 pounds at birth. It scoured continuously beginning shortly after it suckled for the first time. Because it failed to respond to corrective measures, the pig was eliminated from the herd. Autopsy revealed an anomalous condition of the pyloric canal. The lumen of the latter was patent and distended. In place of the gastric sphincter there was only a slight thickening of the greater curvature musculature, a mere suggestion of the concave curvature, which was demarcated by a dwarfed duodenal sphincter.

Assuming that the pyloric canal was nonfunctional, that is, continuously open, the scouring may have resulted, in part at least, from colostrum entering the intestines without having been physically and chemically altered in the stomach. Thus the colostrum acted as a cathartic and caused a continuous scouring.

The gross anatomy of the pyloric canal of this case is shown in Fig. 57 A.

Increased Thickness of the Greater Curvature Musculature of the Pylorus and Pyloric Canal in a Full-term Stillborn Pig

The pig examined was a male weighing 3.4 pounds at birth. The thickness of the greater curvature musculature averaged 2.2 mm. (normal 1.6 mm.). This increase of thickness was caused by an irregular hyperplasia and hypertrophy of the muscle fibers of the outer and the middle circular muscle coats. From the comparative point of view the amount of connective tissue in the torus pyloricus longitudinalis was more abundant than in normal tori. Thus the concavity of the duodenal

sphincter (letter d in Fig. 57 B) may be the result of a rigid torus pyloricus longitudinalis pressing against a still pliable muscle coat. Only 8 ml. of a slightly yellowish fluid were obtained with the needle aspiration method. The fluid had a pH of 3.0, and contained bile but no free hydrochloric acid. The fluid was not tested for pepsin and rennin activity. It seems superfluous to debate whether the closed duodenal sphincter may represent one of three conditions: (a) an aberrant form of stenosis, (b) a pylorospasm, which manifested in utero or during birth, or (c) a normal closure of the duodenal sphincter. Further, it seems highly speculative whether the partial occlusion of the pyloric canal would have resolved itself following ingestion of colostrum.

The gross anatomy of the pyloric canal of this case is shown in Fig. 57 B.

Malformed "Intra-gastric" Pyloric Canal in a Full-term Stillborn Pig (Cross-bred)

This pig examined was a male weighing 1.0 pound at birth. When the abdomen was opened, a fluid-filled stomach was noted. With the aid of a needle aspiration method, 23 ml. of a turbid slightly yellow fluid were obtained. The fluid had a pH of 2.0 and contained bile but no free hydrochloric acid. Because of accidental loss of the fluid, pepsin and rennin activity tests could not be carried out.

The stomach was fixed in situ and opened on the greater curvature. In the distal portion of the pyloric vestibule a tumor-like mass protruded into the chamber. The mass revealed to be an anomalous pyloric canal communicating via a very narrow channel with a distorted duodenum (Fig. 58 f). The location of the malformed pyloric canal was such that

the pyloric glands were present below the sphincter. The term "intragastric" is thus derived from the position of the pyloric canal in the pyloric vestibule.

The gross anatomy of the pyloric canal of this case is shown in Fig. 58.

The statement of Bell (1947) is noteworthy that atresia of the pylorus is a rare anomaly found in the newborn infant.

In summary, it may be said that the anomalies presented in Figs. 57, 58 seem not only to strengthen the hypothesis of Torgersen (1952) that the pyloric canal seem to have arisen from two different parts of the gastro-intestinal tract uniting at the point known as the pyloric canal in the human stomach but also to support our assumption that the pyloric canal of the pig stomach may be a "Zwischendarm" subject to selective disease. Should this hypothesis be born out by future studies, then the pig and the human stomach may be phylogenetically more closely related than is apparent at this stage of our knowledge. There can be little doubt that so far much has to be learned pertaining to the anatomy, physiology, and phylogeny of the pyloric canal in both the simple stomach animal and man.

It is interesting to note that Meshan and Farrer-Meshan (1956) express the opinion that the pylorus, the antrum (pyloric vestibule), and duodenal bulb tend to act in a single unit.

Plate XXXIV

Fig. 57-A. Longitudinal section through the pyloric canal of the stomach of a pig three days of age with atrophy of the greater curvature musculature.

a, Torus pyloricus longitudinalis; b, Underdeveloped gastric sphincter; c, Underdeveloped concave curvature; d, Dwarfed duodenal sphincter. x 6.

Fig. 57-B. Longitudinal section through the pyloric canal of the stomach of a full-term stillborn pig with increased thickness of the greater curvature musculature.

a, Torus pyloricus longitudinalis; b, Gastric sphincter; c, Concave curvature; d, Duodenal sphincter. x 6.

Fig. 58. Longitudinal section through the pyloric canal bulging into the pyloric vestibule of the stomach of a full-term stillborn pig.

a, Torus pyloricus longitudinalis; b, Gastric sphincter; c, Concave curvature with prominent transverse mucosal folds; d, Duodenal sphincter; e, Thickened longitudinal mucosal folds, f, Tail of torus pyloricus longitudinalis; g', Greater curvature musculature of the pyloric vestibule; g", Lesser curvature musculature of the pyloric vestibule. x 6.

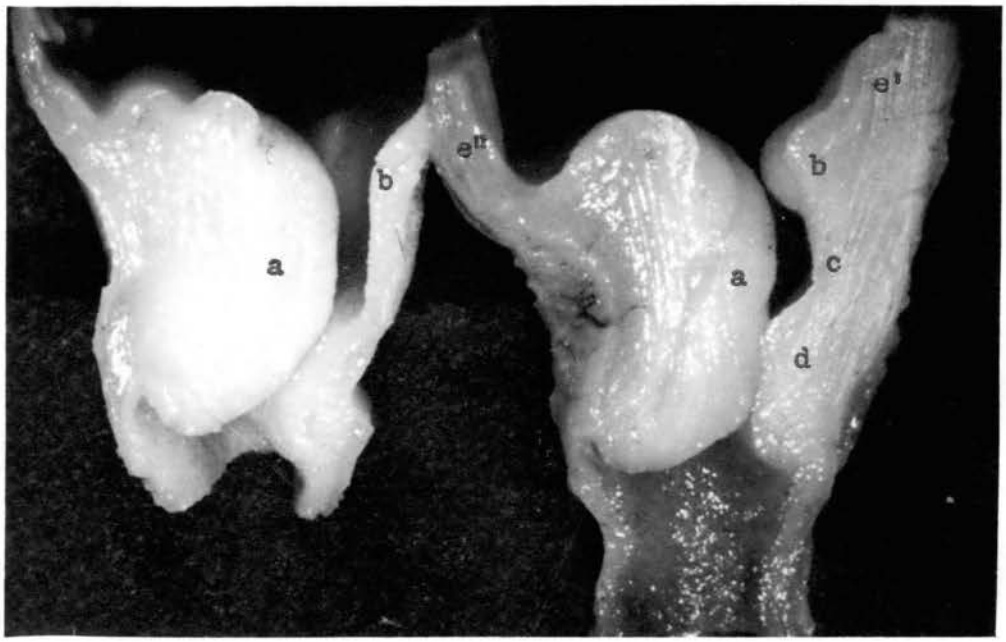
Comment.

The anomalies presented in Figs. 57-A, 57-B, 58 suggest that in the pig the pyloric canal may be a "Zwischendarm" subject to selective disease.

Torgersen (1942) postulates that the duodenal cap is a "Zwischendarm" and that the pyloric canal has arisen from two different parts of the intestinal tract which united at the point known as pyloric canal. According to him the pyloric sphincter is part of the duodenal cap (bulb).

Bell (1947) states that atresia of the pylorus is a rare anomaly found in the newborn infant.

Plate XXXIV



A B

Fig.57

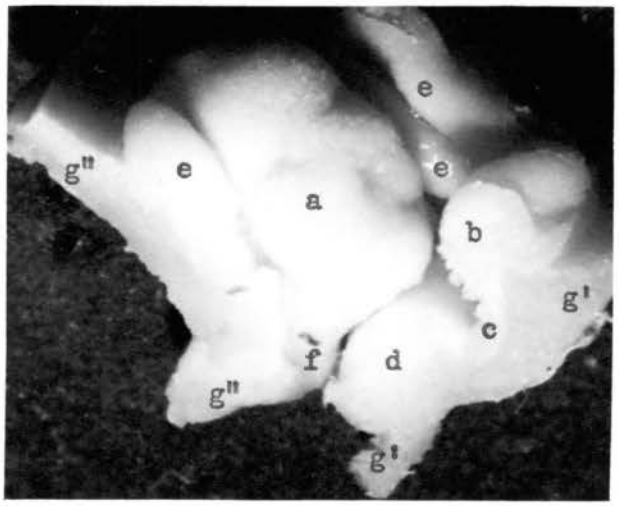


Fig.58

Pyloric Ulcer and Pyloric Canal Stenosis in a Newborn Pig, Three Days of Age

The pig examined was a male weighing 3.0 pounds at birth. Because it missed nursing periods on the second postnatal day, failed to suckle on the third day, displayed symptoms of apathy and was unable to stand on its legs, the pig was eliminated from the herd. The animal was anesthetized; the stomach was fixed in situ during life and opened at the greater curvature. A slightly bile-stained milk mass was present in the stomach. The curd weighed 56 grams. The gastric mucosa was characterized by a superficial nonspecific gastritis associated with patchy petechial hemorrhage and edema. On the lesser curvature of the pyloric vestibule was a raised ulcer, the long axis of which measured 8 mm. and the short axis 6 mm. The crater was somewhat terraced, and its floor was covered with necrotic material. The ulcer was grossly and microscopically similar to peptic ulcer as seen in man. The lesion fulfilled the definition by Ivy, Grossman, and Bachrach (1950) of an alimentary canal ulcer, namely, "a circumscribed defect in the mucosa extending through the muscularis mucosae."

The concomitant pyloric canal stenosis is noteworthy since the closure of the canal lumen resulted from edema of the mucosa and interstitial edema rather than hyperplasia, hypertrophy, or both, of the muscle fibers of the middle circular muscle coat. Thus it may be assumed that the gastric stasis was induced by the ulcer, which caused inflammatory edema in the mucosa and musculature. The pathogenesis of the ulcer is problematical, for in the newborn pig the hydrochloric acid-pepsin mechanism is not sufficiently developed to enter into the production of an ulcer.

Although such factors as local malnutrition resulting from a poor blood supply or ingested straw, sand, urine, feces or vaginal discharge from the sow may be causative agents, we could not establish the primary cause of the ulcer. The lesion suggests, however, that in the newborn pig the mucosa of the gastro-intestinal tract may be vulnerable to mechanical or chemical injury judging from the size of the ulcer, and the fact that among the seventy-six pigs autopsied four pigs ranging in age from three to eight days had a single pyloric ulcer. Although only the pig cited above had concomitant pyloric canal stenosis.

It is a well known fact that an ulcer can develop within a few hours to days depending on the injurious agent and degree of bacterial action of the gastric juice and mucous membrane. The location of the ulcer within the ration of the "Magenstrasse" is particularly interesting since the vast majority of peptic ulcer occurs in this area in the human.

Texter et al., (1953) studied 55 cases with pyloric ulcer in the human. They found that among these subjects 49 had the ulcer on the lesser curvature. Sodeman (1950) noted that local muscular spasms occurring in the adult human evidenced by persistent closure of the pyloric sphincter is generally associated with ulcers on the lesser curvature and duodenum.

Plate XXXV

Fig. 59. Raised pyloric ulcer on the lesser curvature of the pyloric vestibule of the stomach of a pig three days of age.

Arrow points at the border of the ulcer the long axis of which measured 8 mm. and the short axis 6 mm. A superficial nonspecific gastritis associated with patchy petechial hemorrhage characterized the gastric mucosa. x 6.

Fig. 60. Longitudinal section through the pyloric canal of the stomach of the pig three days of age with pyloric ulcer (Fig. 59).

a, Torus pyloricus longitudinalis; b, Edematous mucosa (mucous membrane) pressing tightly against the torus; c, Thickened musculature of the duodenum caused by interstitial edema; d, Thickened greater curvature musculature caused by interstitial edema. x 6.

Comment.

The ulcer penetrated deep into the muscularis externa; thus was fulfilled the definition of an alimentary canal ulcer formulated by Ivy, Grossman, and Bachrach (1950). According to them an ulcer of the gastro-intestinal tract is a "circumscribed defect in the mucosa extending through the muscularis mucosae." The ulcer was grossly and microscopically similar to peptic ulcer as seen in man.

Kerkamp (1945) found gastric ulcers in 18 out of 754 consecutive autopsies of swine less than one year of age. The youngest pig in his series was a male about 6-7 weeks old.

Plate XXXV

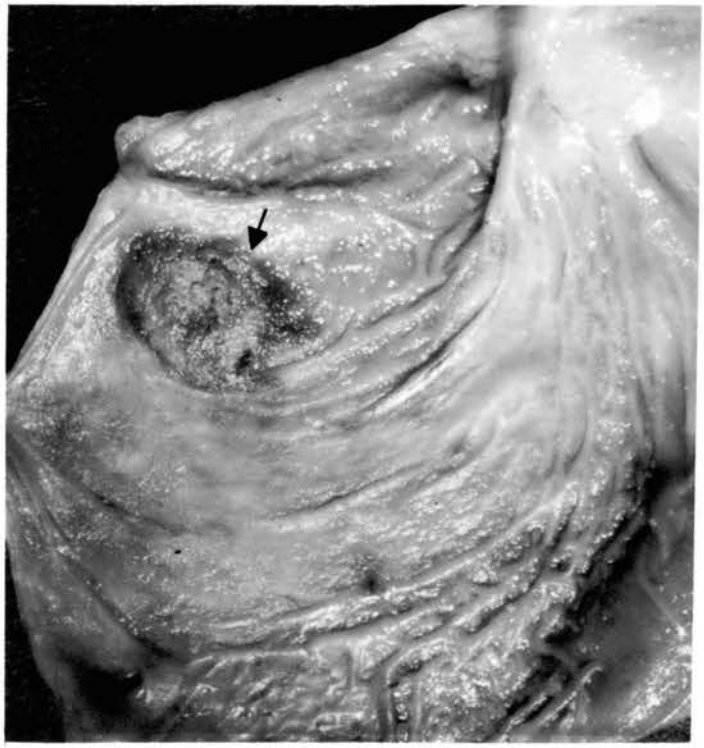


Fig.59

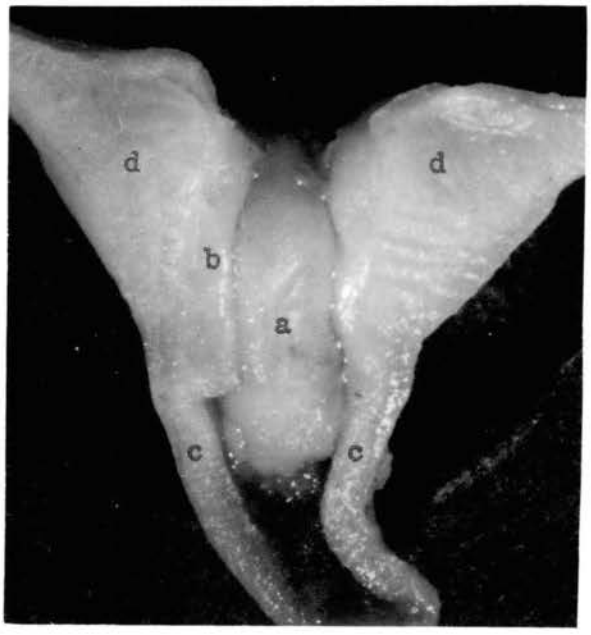


Fig.60

VI. DISCUSSION AND SUMMARY

It has long been known that the gastro-intestinal tract of the pig is anatomically and physiologically similar to that of man; therefore, diseases of this system parallel to those in the human should occur. Our observation of "hypertrophic pyloric canal stenosis" in the newborn pig, which in many respects is analogous to "congenital infantile hypertrophic pyloric stenosis" in the human, supports this presumption. The stenotic state of the pyloric canal in both the pig and the human is caused chiefly by hyperplasia, hypertrophy, or both of the fibers of the middle circular muscle coat and concomitant hyperplasia of the connective tissue. The latter gives the stenosed pyloric canal the fibromatous consistency. So far the pathogenesis of pyloric canal stenosis in the newborn pig and the human is obscure. Evidence is available, however, of a hereditary background in the human. Such data are not available for the pig. In the latter, as in the human, the disease is not sex-linked. The definite predilection of the malady for the human male has not been observed in the male pig in our small series of cases with hypertrophic pyloric canal stenosis. It is noteworthy that in the pig the disease was present as early as the first day of life, which also has been observed in the newborn infant. It may be readily seen in Table 4 that the malady is not limited to a single breed or farrowing season. One may also note that only one or two pigs of a litter were afflicted and that these pigs were constituents of large litters. Neither birth weight nor parents could be

established as causally related to the disease.

Because the pig is multiparous, it is difficult indeed to ascertain the birth rank of the individual pig afflicted with hypertrophic pyloric canal stenosis, for there is no way of knowing in which horn gestation began and whether the position occupied by the fetus in the horn denotes "Anlage" rank. We are cognizant of the fact that two farrowing seasons are not sufficient to place any significance of the occurrence of the disease in large litters only; we are, nevertheless, inclined to speculate that a parallelism exists in the environment of the pig and the human during gestation. To align this presumption with biological phenomena one must draw upon an ecological principle, namely, the mutual relation of an organism to its immediate environment. Thus hypertrophic pyloric canal stenosis may simply be the expression of an unfavorable environment. Should this be the case, one may then postulate that the decrease of the birth rank incidence in the human (McKeown, MacMahon, and Record, 1951) is the sequel of an adjustment of the environment in subsequent pregnancies. By the same token, the occurrence of hypertrophic pyloric canal stenosis in one or two pigs in a large litter may be analogous. Rationalizing upon the fact that each pig fetus has its own amnion and that all have a primiparous character, it is biologically possible that among the amnions one or two may contain an aberrant fluid. Individual susceptibility of the fetus coupled with a specifically altered amniotic fluid may initiate the development and also determine the degree of hyperplasia, hypertrophy, or both of the muscle fibers as well as abnormal proliferation of connective tissue. The sequel of these processes may, in part at least, be responsible for the degree of stenosis of the pyloric canal lumen in utero and/or early postnatal life.

There is adequate evidence in the literature that the fetus ingests amniotic fluid. The most convincing proof has been obtained by injecting colloidal thorium dioxide into the amnion and demonstrating the presence of the radio-opaque substance in the gastro-intestinal tract with the aid of radiography. Although some physical and chemical studies of normal amniotic fluid have been made (Makepeace et al., 1931 and Cantarow, Stuckert, and Davis, 1933), it must be kept in mind that the origin of the fluid is so far not wholly solved (Arey, 1946), and the normal chemical composition of the amniotic fluid is only partly known (Dukes, 1955).

It is interesting to note that Ford, Brown, and McCrary (1941) state that both identical twins have the disease, but that only one of fraternal twins is afflicted. Sheldon (1938), however, found hypertrophic pyloric canal stenosis in only one of identical twins. It seems that these human data support the hypothesis that an aberrant environment (amniotic fluid) coupled with individual susceptibility of the fetus may be the etiological factor underlying the disease. Although it may be questioned that the amniotic fluid is the stressor, it seems that, until evidence is available showing that the malady is caused primarily by an imbalance of inherited interwoven chemical mechanisms in the pyloric canal proper, the environmental stress hypothesis seems worthy of consideration. The latter is believed supported by Trasler, Walker, and Fraser (1956), who were able to show that even a very small mechanical loss of amniotic fluid tends to produce teratalogical abnormalities in the fetus. Further evidence is available which leaves no doubt that physical and biotic environments may impinge upon organisms as a whole or parts thereof and that these environments are not only capable of

duplicating what is produced by gene systems but that aberrancies in these environments can disarrange genetically controlled systems. One is thus logically compelled to postulate that the pyloric canal is an independent anatomical structure (Zwischendarm), in which resides primordial matter that has inherited physical-chemical dynamics of its own. It may thus well be that the pyloric canal of the pig stomach has a phylogenetic background similar to that presumed of man. Thus the problem of hypertrophic pyloric canal stenosis as an anatomical entity seems destined to repose here and the pathogenesis of the disease in both the animal and man of necessity has to be unraveled in the field of biochemistry and histochemistry; that is, by chemical analysis of the components of the pyloric canal, the gastric secretions, and the amniotic fluid. Though the directions for future research are thus clearly indicated the data presented in this thesis should prove of value not only to the swine producer, but also to those working in the field of comparative anatomy, physiology, and pathology because (a) the gross and microscopic anatomy of the stomach and pyloric canal in the pig from birth to the tenth post-natal day are described and illustrated for the first time, (b) new data are presented pertaining to the secretory activity and emptying time of the stomach of the newborn pig including the physical character of the curds of milk from various sources, and (c) the pathological states presented in this thesis of the stomach, pylorus, and the pyloric canal of the newborn pig are strikingly anologous to clinical entities in man.

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VITA

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