The diseases of the genital organs of domestic animals / by W. L. Williams.

Contributors

Williams, W. L. 1856-1945.

Publication/Creation

Ithaca : W. L. Williams ; London : Baillière, Tindall and Cox, 1943.

Persistent URL

https://wellcomecollection.org/works/f8mdhh3m

License and attribution

Conditions of use: it is possible this item is protected by copyright and/or related rights. You are free to use this item in any way that is permitted by the copyright and related rights legislation that applies to your use. For other uses you need to obtain permission from the rights-holder(s).

wellcome collection

Wellcome Collection
183 Euston Road
London NW1 2BE UK
T +44 (0)20 7611 8722
E library@wellcomecollection.org
https://wellcomecollection.org

MINISTRY OF AGRICULTURE
AND FISHERIES,
WEYBRIDGE.

1943.

Diseases of the
Genital Organs of
Domestic Animals.
(3RD. Edition)

W.L. WILLIAMS.

(106558) Wt 43574/1789 Im 9/54 FJP 694

22500588995

Med
K53783

Norman Combes.

1. VII. 44.

Presented by the Librarian,
Royal College of Veterinary
Surgeons

DEG
6.9.55

RETURN TO
VETERINARY LABORATORY
NEW HAW, WEYBRIDGE, SURREY.

The

DISEASES OF THE GENITAL ORGANS OF DOMESTIC ANIMALS

Digitized by the Internet Archive
in 2017 with funding from
Wellcome Library

https://archive.org/details/b29820492

The Diseases of the Genital Organs of Domestic Animals

BY

W. L. WILLIAMS

PROFESSOR EMERITUS, CORNELL UNIVERSITY

THIRD EDITION

PUBLISHED BY W. L. WILLIAMS

209 WHITE PARK ROAD, ITHACA, N.Y

1943

LONDON

BAILLIÈRE, TINDALL AND COX

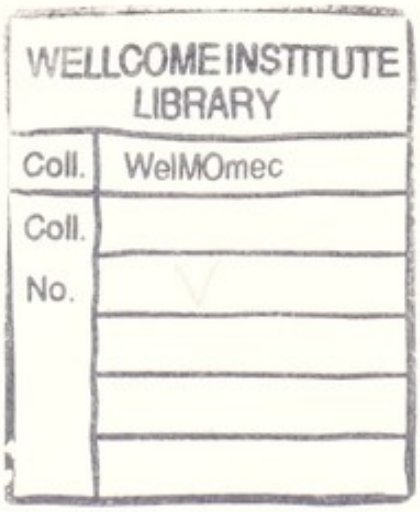
7 & 8, HENRIETTA STREET, COVENT GARDEN, W.C.2

1943

COPYRIGHT 1943 BY

W. L. WILLIAMS

Ministry of Agriculture
Fisheries and Food
Veterinary Laboratory
Library
Class No. US. P. XJ
Author Mk WIL
Access No. L 56/141
Demand No.



PRINTED IN THE UNITED STATES BY
GEORGE BANTA PUBLISHING COMPANY, MENASHA, WISCONSIN

PREFACE

THE present volume is the outgrowth of a series of publications covering a period of thirty-four years. The first of these, *Veterinary Obstetrics; Including the Diseases of Breeding Animals and of the New-Born,* appeared in 1909. That publication was later divided into two treatises, *Veterinary Obstetrics,* appearing in 1917 and the *Diseases of the Genital Organs of Domestic Animals* in 1921. In the second edition of *Veterinary Obstetrics,* 1931, and in the third edition in 1940, an effort was made to escape from the tradition that dystocia is based upon a whim of chance, which causes the fetus to assume an abnormal presentation, position or attitude, or to acquire abnormal form or dimensions, and to search for more fundamental factors in breeding and handling, which might explain the abnormalities leading to difficult labor. This change in concept aroused considerable interest. Chiefly because of this, Professor Dina Nai was instrumental in causing the treatise to appear in the Italian language in 1936. Due to the same reasons, Salvat Editores, Barcelona-Buenos Aires, have recently published both treatises in the Spanish language, rendering them available throughout Latin America, as well as in Spain.

Veterinary obstetrics, which largely revolves about dystocia, is inseparably bound up with the various pathological phenomena of the reproductive system. The attempt is now made, not only to link dystocia with genital diseases, but to study the relationship and interdependence of the various interferences with physiological reproduction. The value of separate studies of such phenomena as sterility, abortion and retained afterbirth is not denied, but it is desired to learn the fundamental causes of these and to weave them into a harmonious mosaic. The task has proven exceedingly difficult and the result has fallen far below the ideal. If, however, the effort may encourage a modest number of clinical veterinarians and breeders to study earnestly the animals under their immediate supervision, the chief aim will have been attained.

During the past years I have published numerous articles, chiefly in the *Cornell Veterinarian,* dealing with the pathology and teratology of reproduction, at a length inadmissable here. These may be studied at greater length with the aid of the bibliographic index. The materials upon which these studies have been based have been largely supplied by the clinics of the Veterinary College. Professors

Udall, Frost, Milks, Fincher, Gibbons, Danks, Stephenson and others have placed me under profound obligation by their many courtesies in providing the materials and opportunity for the study. W. W. Williams, D.V.M., M.D., has contributed generously, especially regarding the studies of spermatozoa, in which he has conducted important initial studies. Esteemed colleagues in various lands have extended courteous aid, including permission to copy instructive illustrations. It has been aimed to acknowledge these in the text.

W. L. WILLIAMS

Ithaca, N.Y.

CONTENTS

THE PROBLEM OF GENITAL DISEASE 3

SECTION I

THE BASIC CAUSES OF INTERFERENCES WITH REPRODUCTION
HEREDITARY AND CONGENITAL DEFECTS

CHAPTER I. HYBRIDIZATION 11
Extreme Outcrosses 11
Bulldog Calves 11
Tux-Zillertaler Cattle 13

CHAPTER II. INBREEDING 14
Muscle Contracture Monsters 15
Atresia of the Colon. Hydrocephalus. Cerebral Glioma 17
Excessive Ratio of Males 18
Cleft Palate 18
Persistence of the Right, instead of the Left Primitive Aorta 18
Color and Fertility 18
Cerebral Hernia 19
Umbilical Hernia 20
Teratological and Congenital Defects of the Reproductive System .. 20
Inguinal Hernia in the Male 21
Inguinal Hernia in the Female 22
Penial Defects 23
Cryptorchidy 23
Imperfect Descent of the Testicles with Gigantic Teats in the Male, and with Abnormal Uterine Caruncles in the Female 24
Arrested Development of Mueller's Ducts. Uterus Unicornis. Uterus Didelphys 26
Persistent Hymen 33
Abnormal Wolffian (Gartner's) Ducts 33
The Prevention and Handling of Monsters and Congenital Defectives 34

CHAPTER III. TERATOLOGICAL AND PATHOLOGICAL OVULATION AND SPERMATOGENESIS 37
Twin and Multiple Ovulation in Unipara 49
Equine Twins 49
Bovine Twins 53
Asexual (Freemartin), Acardiac and Amorphous Twins or Multiples 61
Pathological Spermatogenesis 76
Pathological Duration of Gestation 86
Abbreviated Gestation 87
Prolonged Gestation 88
Defective Nutrition of the Fetus 92

CHAPTER IV. THE INFLUENCE OF THE HEALTH OF THE NEW-BORN UPON ITS FERTILITY AS AN ADULT 96

CHAPTER V. THE BREEDING SEASON 107

CHAPTER VI. THE AGE AT WHICH YOUNG ANIMALS SHOULD FIRST BE BRED 110

CHAPTER VII. THE BREEDING OF DECREPIT AND OLD ANIMALS 117

CHAPTER VIII. THE RAPIDITY OF BREEDING 125
The Frequency of Coitus 125
The Post-Partum Rest Interval of the Female Before Re-breeding .. 129
The Sex Ratio and Breeding Methods 142

CHAPTER IX. FOODS AND FEEDING 145
The Essential Mineral Constituents of Food 146
Vitamins 148

CHAPTER X. LABOR, HOUSING AND CONFINEMENT 151
Labor 151
Confinement 152

SECTION II
ESTRUM, COITUS AND PREGNANCY

CHAPTER XI. ESTRUM, OR "HEAT" 157

CHAPTER XII. THE ARTIFICIAL INDUCTION OF ESTRUM 162
The Dislodgment of the Corpus Luteum 162

CHAPTER XIII. COPULATION, COITUS 167
The Dangers of Coitus 169
False Entry, Rectal Injuries 171
Laceration and Rupture of the Vagina 172
The Alleged Rupture of the Bovine Uterus and Bladder by the Penis of the Bull 173
Sodomy and Sadism 173

CHAPTER XIV. ARTIFICIAL INSEMINATION 176

CHAPTER XV. PREGNANCY 189
The Clinical Diagnosis of Pregnancy 190
The Uterine Seal 191
The Rectal Palpation of the Reproductive System 194
The Diagnosis of Twin Pregnancy 200
The Laboratory Diagnosis of Pregnancy 201

SECTION III
TUMORS OF THE REPRODUCTIVE SYSTEM

CHAPTER XVI. MALIGNANT TUMORS 205
Malignant Tumors of the Testicles 205
Malignant Ovarian Tumors 205
Malignant Tumors of the Uterus, Cervix and Vagina 207
Malignant Tumors of the Penis 208

CHAPTER XVII. BENIGN TUMORS 209
Benign Tumors of the Ovaries and Testicles, Par-ovarian Cysts ... 209

Benign Tumors of the Oviducts, Uterus, and Cervix 216
Benign Tumors of the Penis, Prepuce and Sheath 219

SECTION IV

SYSTEMIC AND ORGANIC INFECTIONS WHICH SOMETIMES INVADE THE GENITAL SYSTEM AND INTERFERE WITH REPRODUCTION

CHAPTER XVIII. ANIMAL PLAGUES 225

CHAPTER XIX. GENITAL TUBERCULOSIS 227
Genital Actinomycosis 249
Genital Bursattee 251

SECTION V

INFECTIONS BELIEVED TO HAVE MARKED AFFINITY FOR THE REPRODUCTIVE SYSTEM, CONTAGIOUS ABORTION, CONTAGIOUS STERILITY

CHAPTER XX. THE SPECIAL INFECTIONS OF THE REPRODUCTIVE SYSTEM IN PERSPECTIVE 255

CHAPTER XXI. BACTERIAL INVADERS 280
Bacillus abortus (Bang) 280
Bacillus abortivo-equinus (Good) 280
Vibrionic or Spirilla Abortion 286
The Symptoms of Bacterial Abortion 286
The Lesions of Bacterial Abortion 287
The Diagnosis of Bacterial Abortion 294
The Avenue and Date of Invasion 296
The Pathogenicity of Bacteria Associated with Abortion and Related Phenomena 307
The Control of Abortion, Sterility and Allied Phenomena Ascribed to Bacteria 310
Endocrines 313
Vitamins 313
Minerals 313
Disinfectants 314
Immunization, Vaccination 315
Quarantine and Slaughter 326

CHAPTER XXII. MOLDS ASSOCIATED WITH INTERFERENCES WITH REPRODUCTION 340

CHAPTER XXIII. PROTOZOAN DISEASES INTERFERING WITH REPRODUCTION 341
Dourine 341
Trichomonad disease 352

CHAPTER XXIV. VIRUS DISEASES OF THE REPRODUCTIVE SYSTEM 360
The Vesicular Venereal Disease, Coital Exanthem 360
Virus Abortion 363

CHAPTER XXV. UNCLASSIFIED GENITAL DISEASES 365
Infectious Vaginitis, Nodular or Granular Venereal Disease 365

Venereal Tumors of Dogs 375
Venereal Granulomata. Lymphosarcoma 375

SECTION VI

THE DISEASES OF VARIOUS GENITAL ORGANS

CHAPTER XXVI. THE DISEASES OF THE MALE REPRODUCTIVE ORGANS.... 383
The Diseases of the Testicles 388
Testicular Hypoplasia 388
Orchitis 394
Acute Orchitis 395
Chronic Orchitis 404
Testicular Degeneration 408
Degeneration of the Tubuli Seminiferi 408
The Pathology of the Semen and of the Spermatozoa 411
Epididymitis 420
The Diseases of the Accessory Male Organs 421
Diseases of the Vasa Defferentia and Seminal Vesicles 421
Prostatitis 436
Diseases of the Glans, Prepuce and Sheath 436
Balanitis 436
Post-Coital Urethral Hemorrhage 438

CHAPTER XXVII. THE DISEASES OF THE FEMALE GENITAL ORGANS.... 439
Atretic Graffian Follicles 439
Ovaritis. Abscess of Ovaries 441
Cystic Degeneration of the Ovisacs. Nymphomania 441
Nymphomania Due to Central Ovarian Cyst 451
Equine Nymphomania 451
Nymphomania in Small Animals 455
Intra-Follicular Hemorrhage 455
Post-Ovulation Hemorrhage into the Follicular Crater..... 456
Diseases and Abnormalities of the Corpus Luteum 456
Persistent, Retained, or Embedded Corpus Luteum 456
Cystic Degeneration of the Corpus Luteum 459
Hypertrophy of the Corpus Luteum 465
Corpora Nigra 466
The Diseases of the Oviducts 466
Salpingitis. Pavilionitis 467
Hydrosalpinx 470
Pyosalpinx 471

CHAPTER XXVIII. CHRONIC DISEASES OF THE UTERUS, CERVIX, VAGINA AND VULVA OF THE NON-PREGNANT FEMALE 473
Chronic Metritis and Cervicitis 473
Chronic Vaginitis and Vulvitis 485

CHAPTER XXIX. DISPLACEMENTS OF THE GRAVID UTERUS AND VAGINA.. 487
Uterine Torsion 488
Hysterocele. Uterine Herniae 500
Rupture of the Prepubian Tendon 500
Strangulation of the Colon by the Gravid Uterus 502
Vaginal Prolapse 502

CHAPTER XXX. THE DISEASES OF THE GRAVID UTERUS AND OF THE CONTAINED OVUM 505
The Early Death of the Ovum Due to the Union of Defective Sex Cells 505
The Necrotic Tip of the Chorion 507
Placentitis. Metritis. Chorionitis 513
Equine Placentitis 513
Placentitis and Chorionitis of Ruminants 514
Adventitious Placentae 521
Profuse Caruncular Hemorrhage in the Cow 523
Bovine Hematic Mummification 523
"Uterine Sand" 530
Edema of the Chorion 530
Dropsy of the Fetal Membranes 531
Hydramnios and Hydrallantois 531
Fetal Anasarca 537
Torsion of the Umbilical Cord 537
Amputation of Fetal Extremities by the Umbilical Cord 538
Utero-Fetal Disease and Dystocia 539
Fetal Death 540
Fetal Death with Papyraceous Mummification 541
Putrid Decomposition of the Fetal Cadaver 543

CHAPTER XXXI. THE PATHOLOGICAL PHENOMENA SURROUNDING THE BIRTH PERIOD 546
The Metritis of Advanced Pregnancy 546
Retained Afterbirth. Retained Placenta 549
Retained Afterbirth in Mare 552
Parturient Laminitis 553
Retained Placenta in Cow 555

CHAPTER XXXII. PARTURIENT INJURIES 569
Rupture of the Uterus 569
Rupture of the Perineum 569
Lacerations and Contusions of the Vagina 570

CHAPTER XXXIII. PUERPERAL DSPLACEMENTS OF THE URINO-GENITAL ORGANS 571

CHAPTER XXXIV. PUERPERAL DISEASES 573
Spontaneous Intrauterine Hemorrhage 573
Puerperal Tetanus 575
Pyometra 576
Uterine Abscess 577
Puerperal Pyaemia. Pyaemic Arthritis. Pyaemic Abscesses. Puerperal Meningitis. Pyelo-Nephritis 577

SECTION VII

THE DEFECTS AND THE DISEASES OF THE NEW-BORN

CHAPTER XXXV. ASPHYXIATION OF THE NEW-BORN 582

CHAPTER XXXVI. SEPSIS OF THE NEW-BORN 584

CHAPTER XXXVII. ANTENATAL AND EARLY POSTNATAL RUPTURES OF TENDONS AND LIGAMENTS 585

CHAPTER XXXVIII. THE IMPACTION OF THE LARGE INTESTINES WITH MECONIUM 590

CHAPTER XXXIX. THE PHYSIOLOGY, PATHOLOGY AND HYGIENE OF THE DIGESTIVE SYSTEM OF THE NEW-BORN 593

The Artificial Feeding and Handling of the New-Born 593

Diarrhea of the New-Born. White Scours. Calf Scours 593

Calf Pneumonia 604

Omphalophlebitis. Navel Ill 607

BIBLIOGRAPHY 616

SUBJECT INDEX 627

ILLUSTRATIONS

COLORED PLATE I. Gravid uterus of cow at term, showing fetal membranes and fetus ..... Facing page 1
COLORED PLATE II. Gravid uterus of cow at about four months, showing "abortion exudate" in non-gravid horn ..... Opposite page 290
COLORED PLATE III. Cervicitis in cow ..... Opposite page 482
CHART I. The rainfall in the area occupied by herd shown in Table X. ..... Page 98
TABLE I. Breeding record of eighteen heifers bred to calve first in 1912 ... 56
TABLE II. Breeding records of a diseased, compared with a healthy bull ... 79
TABLE III. Breeding records in a stud during 1920-1921 80
TABLE IV. Breeding records of three Hereford bulls during ten years 81
TABLE V. Breeding behavior of two Jersey bulls in a herd 87
TABLE VI. The duration of gestation and the mortality of bovine twins according to sex 89
TABLE VII. The breeding efficiency of mares according to the duration of time they were carried in utero as fetuses 90
TABLE VIII. Breeding record of female progeny of bull X (Wagner) 91
TABLE IX. Changes in the fertility of heifers following improvements in the feeding as young calves 97
TABLE X. Breeding records of a Holstein-Friesian herd from 1920-1933 inclusive, showing with Chart I the influence of rainfall upon fertility 99
TABLE XI. The duration of the estrual cycles in a group of twenty-four consecutive heifers from a Bang-negative purebred dairy herd 132
TABLE XII. Breeding records of nine cows from Table X showing the effects of rapid, compared with conservative breeding 136
TABLE XIII. The persistence of the positive reaction to the agglutination test for *Bacillus abortus*, from calfhood to breeding age 303

TEXT FIGURES

FIG. 1. Bulldog Calf 12
FIG. 2. Cerebro-Spinal Axis of bovine *Muscle Contracture* Monster 15
FIG. 3. Heads of *Muscle Contracture* Monsters Illustrating the Absence of Palate 16
FIG. 4. Cerebral Hernia of Pig 19
FIG. 5. Teratological Reproductive Organs of the Female Progeny of an Inbred Bull 25
FIG. 6. Defective Reproductive Organs from three of a Group of twenty-three Daughters from Sire-Daughter Matings 27
FIG. 7. Equine Uterus Unicornis 28
FIG. 8. Bovine Uterus Unicornis with Deformed Cervix 29
FIG. 9. Double Bovine Uterus 30
FIG. 10. Double Bovine Uterus from Aborting Heifer 31
FIG. 11. Double Os Uteri Externum. Cow 32
FIG. 12. Internal Generative Organs of Woman 39
FIG. 13. Internal Generative Organs of Mare 40

FIG. 14. Adult Bovine Uterus and Ovaries 41
FIG. 15. The Uterine Caruncles of Veal Heifers 42
FIG. 16. The Non-Gravid Uterus of Bitch 43
FIG. 17. Pregnant Canine Uterus 44
FIG. 18. Gravid Swine Uterus Containing 13 Fetuses 45
FIG. 19. The Placentation of Equine Twins 48
FIG. 19*a*. Allantochorions from full term, living equine twins 50
FIG. 20. Fused Chorions of Unicornual Bovine Twins 52
FIG. 21. Fused Chorions of Ovine Triplets 54
FIG. 22. Double Monster showing almost identical Color Markings 62
FIG. 23. *Amorphus Globosus*. Bovine 64
FIG. 24. Sagittal Section of Fig. 23 65
FIG. 25. Bovine Acardiac Monster, Twin to a viable Bull 66
FIG. 26. Sections of Gonads from a Single Mixed-sex Monster 70
FIG. 27. The Urino-Genital Exit of a Bovine Monster, Twin to a Bull ... 71
FIG. 28. Spermatozoa from Stallion 1 in Table III 80
FIG. 29. Healthy Stallion Spermatozoa 82
FIG. 30. Pathological Equine Placenta, after 387 Days Pregnancy 94
FIG. 31. Soiled Vulvar Tuft of Heifer Calf 102
FIG. 32. Clean Vulvar Tuft of Heifer Calf 103
FIG. 33. Soiled Preputial Tuft of Bull Calf 104
FIG. 34. Clean Preputial Tuft of Bull Calf 105
FIG. 35. A Group of Healthy Calves 106
FIG. 36. Healthy Allantochorion of Cow 118
FIG. 37. Diseased Allantochorion from Aborting Cow 119
FIG. 38. Diseased Allantochorion from Cow 120
FIG. 39. Healthy and Diseased Bovine Cotyledons 121
FIG. 40. Fetal Membranes from a Wrecked Bovine Uterus 122
FIG. 41. Multiple Ovarian Cysts in New-Born Calves 158
FIG. 42. Fatal Hemorrhage following dislodgment of the Corpus Luteum 164
FIG. 43. Ovarian Scalpel 165
FIG. 44. Deformed Equine Pelvis 168
FIG. 45. Ventral Hernia. Bull 181
FIG. 46. Chorionic Tufts of Villi, Magnified. Cow 188
FIG. 47. Uterine or Cervical Seal. Cow 192
FIG. 48. Cystic Mole. Cow 193
FIG. 49. Sagittal Section through the Cervix of a Pregnant Cow 195
FIG. 50. The Equine Vascular Allantois 197
FIG. 51. Ovarian Angio-Sarcoma. Heifer 206
FIG. 52. Ovarian Cyst-Adenoma. Sow 207
FIG. 53. Sarcoma of Genital Tract. Bitch 208
FIG. 54. Dermoid Cyst of Testicle. Horse 209
FIG. 55. Dermoid Cyst of Ovary. Mare 210
FIG. 56. Ovarian Cyst. Mare 211
FIG. 57. Par-Ovarian Cyst. Mare 212
FIG. 58. Par-Ovarian Cyst. Mare 213
FIG. 59. Par-Ovarian Cyst. Mare 214
FIG. 60. Large Ovarian Cyst. Pregnant Cow 215
FIG. 61. Uterine Fibroma. Cow 217
FIG. 62. Uterine Fibroma. Mare 217
FIG. 63. Amputation of Penis. Horse 219

FIG. 64. Amputation of Penis. Horse 220
FIG. 65. Amputation of Penis. Horse 221
FIG. 66. Penis of Bull Calf before Opening of Preputial Sac 230
FIG. 67. Penis of Bull Calf after Opening of Preputial Sac 230
FIG. 68. Coital Rupture of Prepuce. Bull 230
FIG. 69. Tuberculous Orchitis. Boar 231
FIG. 70. Tuberculosis of Glans and Prepuce. Bull 232
FIG. 71. Primary Penial Tuberculosis. Bull 233
FIG. 72. Tuberculosis of Sheath and Prepuce 234
FIG. 73. Tuberculosis of Glans and Prepuce. Bull 235
FIG. 74. Tuberculosis of Peripenial Lymph Glands 236
FIG. 75. Sagittal Section of Fig. 74 236
FIG. 76. Tuberculosis of Ovary and Oviduct. Cow 237
FIG. 77. Tuberculous Ovary in Serial Sections 238
FIG. 78. Advanced Tuberculosis of Oviducts and Cornua 239
FIG. 79. Advanced Tuberculosis of Ovary and Oviducts 240
FIG. 80. Generalized Tuberculosis of the Reproductive System 241
FIG. 81. Horizontal Section of Fig. 80 242
FIG. 82. Advanced Tuberculosis of Reproductive System of Cow Negative to Tuberculin Test 243
FIG. 83. Advanced Genital Tuberculosis without Peritoneal Adhesions 244
FIG. 84. Sagittal Section of Fig. 83 245
FIG. 85. Advanced Tuberculosis with Profuse Pyometra 246
FIG. 86. Tuberculosis of the Cervix 247
FIG. 87. Sagittal Section of Fig. 86 248
FIG. 88. Avian Tuberculosis of Bovine Genital System 249
FIG. 90. Genital Actinomycosis. Cow 250
FIG. 91. Necrotic Swine Embryos 284
FIG. 92. Necrotic and Healthy Swine Embryos 285
FIG. 93. Diseased Allantois. Mare 291
FIG. 94. Normal Vascular Allantois. Mare 292
FIG. 95. The Equine Vascular Allantois from a Nine Months' Abort 293
FIG. 96. Necrotic Tips of Bovine Fetal Sacs 298
FIG. 97. Bovine Fetal Sacs from Uteri in which the Endometrium Has Been Destroyed 299
FIG. 98. Dourine. Stallion 343
FIG. 99. Dourine. Mare 344
FIG. 100. Mild Granular Venereal Disease. Heifer 366
FIG. 101. Advanced Granular Venereal Disease. Cow 367
FIG. 102. Granular Venereal Disease of Glans and Prepuce. Bull 368
FIG. 103. Histological Section of Nodule of Vaginitis 369
FIG. 104. Histological Section of a Series of Nodules 371
FIG. 105. Histological Section of a Series of Nodules 372
FIG. 106. Histological Section of a Mass of Nodules 373
FIG. 107. Venereal Tumors. Dog 375
FIG. 108. Venereal Tumors. Bitch 376
FIG. 109. Venereal Tumors. Bitch 376
FIG. 110. Semen from Healthy and Diseased Bulls 389
FIG. 111. Spermatozoa from three Diseased Bulls 390
FIG. 112. Histological Section of Testicular Hypoplasia. Young Bull 391
FIG. 113. Sperm blocking of Vasa Efferentia. Goat Testicle 394

FIG. 114. *Bacillus abortus* Orchitis. Bull 396
FIG. 115. Congenital Purulent Infection of Umbilicus. Calf 398
FIG. 116. Section through the Pelvis of Calf Shown in Fig. 115 399
FIG. 117. Same as 116, with most organs in sagittal section 400
FIG. 118. Orchitis and Epididymitis. Horse 401
FIG. 119. Orchitis and Epididymitis. Horse 402
FIG. 120. Degenerative Orchitis. Bull 403
FIG. 121. Diseased Genital Organs. Horse 405
FIG. 122. Healthy Genital Tract. Horse 406
FIG. 123. Chronic Orchitis. Bull 407
FIG. 124. Calcareous Degeneration of Testicle. Goat 408
FIG. 125. Defective Spermatozoa after Experimental Degeneration. Bull .. 410
FIG. 126. Spermatozoa from two bulls of low fertility and excessive ratio of abortion of pregnancies 414
FIG. 127. Normal and Immature Spermatozoa. Bull 416
FIG. 128. Spermatozoa from Healthy Stallion and from one of low Fertility 417
FIG. 129. Bovine Spermatozoa with Tails Detached 418
FIG. 130. Elongated, narrow Spermatozoa heads 419
FIG. 131. Diseased Ampulla of vas deferens. Horse 422
FIG. 132. Abscess of Seminal Vesicle and Ampulla. Bull 422
FIG. 133. Intra-pelvic Urino-genital Organs of an Adult Bull with Abscess of left Seminal Vesicle 424
FIG. 134. Testicles of Bull, some of whose Organs are shown in Fig. 133 .. 427
FIG. 135. Old-standing Semino-vesiculitis in a mature Jersey Bull 428
FIG. 136. Same as 135 with Ampullae of the Vasa Defferentia removed to expose the bases of the vesicles 429
FIG. 137. Semino-Vesiculitis and Disease of Ampullae. Bull 430
FIG. 138. Abscess of Seminal Vesicles. Bull 432
FIG. 139. Diseased Testicles, Vasa Defferentia and Vesiculae Seminales. Bull 435
FIG. 140. Adherent Ovaries. Cow 442
FIG. 141. Nymphomania. Cow 444
FIG. 142. The Pelvic Ligaments. Cow 446
FIG. 143. Nymphomaniac cysts of cow 447
FIG. 144. Cystic degeneration of swine ovaries 453
FIG. 145. Cystic and Normal Ovaries. Bitch 454
FIG. 146. Intra-Follicular Hemorrhage in the Ovary. Heifer 455
FIG. 147. Cystic Corpora Lutea. Cow 460
FIG. 148. Cystic Corpus Luteum. Cow 462
FIG. 149. Cystic Corpus Luteum with Adhesion of the Ovary in the Pavilion. Cow 463
FIG. 150. Cystic Corpus Luteum with Adherent Cystic Oviducts. Cow 464
FIG. 151. Salpingitis. Pregnant Sow 465
FIG. 152. Corpus Nigrum. Cow 466
FIG. 153. Hydrosalpinx and Uterine Abscess. Heifer 468
FIG. 154. Cystic Corpus Luteum, Hydrosalpinx and Pelvic Adhesions 470
FIG. 155. Cystic Pavilion and Hydrosalpinx. Cow 471
FIG. 156. Tubo-Ovarian Abscess. Cow 472
FIG. 157. Pyometra in Bitch 474
FIG. 158. Instruments for Douching Uterus of Cow 478

FIG. 159. Sclerosis of Cervix and Uterus. Cow 479
FIG. 160. Vaginal Ends of Cervices. Cow 480
FIG. 161. Rupture of Uterus with Catheter. Cow 481
FIG. 162. The Allantochorion of the Mare 490
FIG. 163. Pregnant Uterus of Bitch in Situ 491
FIG. 164. Transverse Rupture of Uterus due to Torsion. Ewe 496
FIG. 165. Unicornual Torsion. Cat 497
FIG. 166. Extra-Uterine Fetuses. Swine 498
FIG. 167. Rupture of Prepubian Tendon. Mare 501
FIG. 168. Sutures for Uterine and Vaginal Prolapse 504
FIG. 169. Invaginated Necrotic Tip of Chorion 508
FIG. 170. Allantochorion of *Schistosomus reflexus*. Cow 510
FIG. 171. Penetrant Necrosis of Chorion. Cow 515
FIG. 172. Healthy and Diseased Cotyledons. Cow 517
FIG. 173. Allantochorion subsequent to Destruction of the Uterine Caruncles. Cow 519
FIG. 174. Chorion indicating mild Cervicitis 520
FIG. 175. Membranes after Abortion at 116 Days. Cow 522
FIG. 176. Inter-placental Hemorrhage associated with Dystocia and Stillbirth. Cow 524
FIG. 177. Interplacental Hemorrhage leading to Hematic Mumification .. 525
FIG. 178. Sagittal Section of Uterus with Interplacental Hemorrhage. Cow 526
FIG. 179. Completed Mummification. Cow 526
FIG. 180. Bent Mummified Fetus. Cow 527
FIG. 181. Amnio-Allantoic Dropsy. Cow 533
FIG. 182. Opened Uterus from Fig. 181 534
FIG. 183. Fetal Anasarca. Twin Lambs 537
FIG. 184. Twin Aborts. Mare 540
FIG. 185. Maceration of Fetus with Retention of Skeleton in the Uterus. Cow 543
FIG. 186. Macerated Fetus sloughing through Abdominal Floor. Cow ... 544
FIG. 187. Horizontal Section of Genital Tract of Cow to show Didelphic Type 572
FIG. 188. Tesselated Intrauterine Hematoma. Cow 574
FIG. 189. Musculo-tendinous Rupture of the Extensor Pedis Longus. Foal 586
FIG. 190. Antenatal Rupture of Extensor Pedis Longus Muscles and Detachment of Flexor Pedis Perforans. Foal 587
FIG. 191. Rupture of Extensor Pedis Longus Muscles and Dislocation of Patellae 588
FIG. 192. Umbilical Cord. Foal 608
FIG. 193. Abscess of Umbilical Vein and Artery. Foal 609
FIG. 194. Carpal Arthritis following Navel Infection. Foal 613
FIG. 195. Abscess in third Phalanx following Navel Infection. Foal 614
FIG. 196. Abscess in Spinal Canal Following Navel Infection. Calf 615

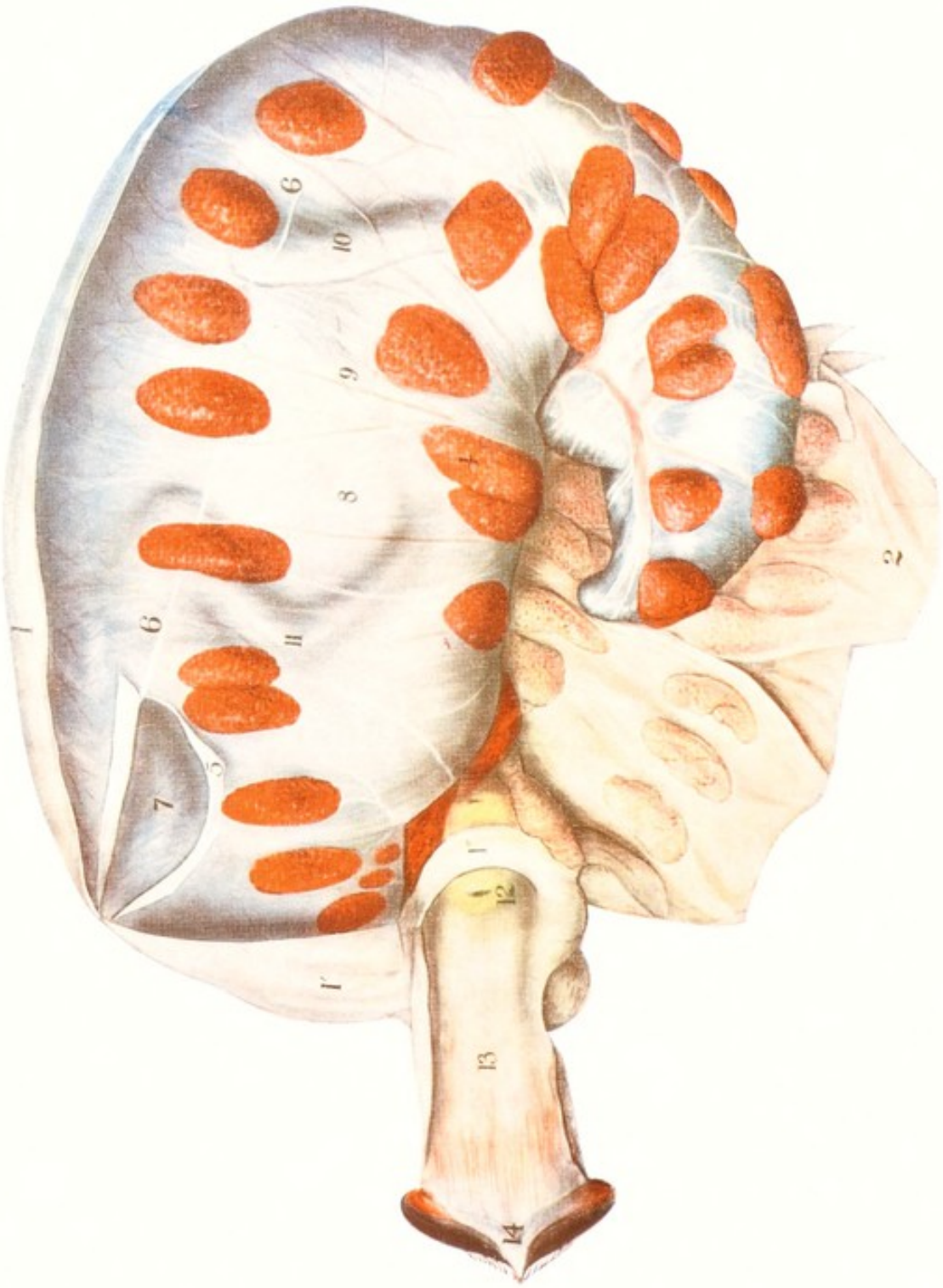

1
6
10
9
8
4
11
6
7
3
1'
12
13
14
2'

Plate I

Gravid Uterus of Cow at full term laid open to show Fetal Sac with faint outline of Fetus

1, dorsal or greater curvature of uterus; 1′, supra-vaginal pouch of uterus; 1″, section through uterus at cervix; 2, right wall of uterine horn detached from chorion and turned down; 4, ventral row of chorionic (fetal) cotyledons. The first two are partly hidden beneath the fetal sac against the cervix. The apex of the fetal sac is rolled outwards showing the median raphe to the left of the last four cotyledons; 5, dorsal margin of the allantoic sac incised and turned down, exposing 7, the amniotic sac; 6, 6, dorsal line of allantoic sac, above which only the vascular layer of the allantois extends over the amnion, closely adhering to it; 9, umbilical cord; 10, left fetal tibia; 11, left fetal carpus; 12, os uteri externum and cervix, showing the cervical canal directed along the uterine floor; 13, vagina; 14, vulva.

The

DISEASES OF THE GENITAL ORGANS OF DOMESTIC ANIMALS

THE PROBLEM OF GENITAL DISEASE

REPRODUCTION is not essential to the life or health of the individual. On the contrary it constitutes an important burden upon the parent, which may imperil its health or life. Ideal mammalian reproduction occurs only in those individuals having at their command, over and above their needs for the maintenance of physical life and growth, ample reserves of energy for fertilization and for the adequate nurture of the young until it shall have reached a stage of development which will enable it to lead an independent existence.

Unicellular organisms multiply by the division of the single cell to constitute two or more similar beings, without sex. Higher forms of life become divided into two sexes, and reproduction has its beginning in the union of a male and a female cell. Some fishes, such as salmon, do not breed until their growth has been completed: they then leave the ocean, ascend a river, the female ovulates, the male discharges semen in a manner to best secure the union of the spermatozoa and ova, and both male and female die. The species is perpetuated by the enormous numbers of young following the single ovulation, largely compensating for the great mortality of the progeny.

Ideal mammalian reproduction does not terminate the lives, nor materially imperil the health of the parent animals. It is the slowest type of animal reproduction, the ratio of the mortality of the young is the lowest, and the longevity of the individual the highest. Horses and cattle of both sexes sometimes remain fertile for over 25 years.

Although the rate of mortality among fertilized mammalian ova is the lowest in animal life, reproduction in domestic animals falls far below the ideal. Vast numbers of fertilized ova, embryos and new-born perish. The rate of mortality per unit of time is inverse, so far as can now be determined, to the age of the new individual. In unipara there are commonly two or more copulations per identified pregnancy; once pregnancy has been recognized, abortion occurs in perhaps 10 to 15 per cent. The losses among domestic mammals are greatest in dairy cattle, most of which die from, or are discarded because of genital diseases, including mammary affections, without having produced the number of healthy young which should be anticipated.

Authentic data regarding the lifetime reproductive efficiency of any species or breed of domestic animals are not available. Among

the most informative data found recorded are those of Wing[271] concerning the Holstein-Friesian herd at Cornell University. During the forty years covered by Wing, the Holstein-Friesian herd had averaged 3.35 pregnancies, with an average of 2.4 viable calves per cow. The data are not complete. Some fertile cows were sold. Wing states that 232 heifer calves were raised, that 192 were pregnant at least once, and that, along with 34 which had been pregnant, 20 unbred heifers remained in the herd in 1928. This leaves 20 heifers missing, which presumably failed, for some unrecorded reason, to breed. No documentary trace of the Wing herd since 1928 is available: it disappeared without leaving a trace. Wing reports 463 viable calves from the 232 females raised, or barely less than two per female raised.

Henderson[86] studied the reproductive efficiency of an anonymous herd consisting of six breeds of dairy cattle, the multiplicity of breeds indicating that it was the property of an agricultural college. A prior herd had been eliminated because of *Bacillus abortus* infection and a new Bang-negative group assembled by purchase. The number of individual cows studied, and the average number of viable calves per cow are not stated. The average copulations per recognized pregnancy was placed at 2.1. The abortion rate for all breeds was 13.9 per cent, while for the Holsteins the ratio was 17.8. Wing's bulletin included only the Holstein cows of the Cornell University herd and the average abortion rate, including "dead at birth" was 18.6 per cent, or 0.8 per cent higher than in the Henderson herd. The females described by Wing were all grown, and bred as heifers within the herd. The heifers in first pregnancy supplied 50 per cent of the total number of abortions. In the Henderson herd, assembled by extensive purchase, many of the cows had already calved one or more times. Consequently Henderson's data show that fewer abortions occurred at two, than at three or four years. So far as may be inferred from the Henderson data, the average number of viable calves per cow was certainly no better than in those recorded by Wing. No herd with an abortion rate of 17.8 per cent can produce a high average of viable calves.

A common ideal of reproduction advocated for dairy cows is that heifers shall first calve at about two years, and annually thereafter. Had such ideal been attained in the Wing herd, without abortions or dead at birth, the average duration of life of the cows, for the production of 2.4 calves would have been 3.4 years. Unofficial information indicates that the Cornell University dairy herd is fairly representative for large dairy herds of the northeastern United States

subject to high-pressure dairying. Although there are many thousands of dairy cattle maintained in the 48 state agricultural colleges and experiment stations, no authentic reports are available showing the lifetime production of viable young during the existence of any one of these herds. At least one other state agricultural college herd, in addition to that reported by Wing, contains no descendants of the original foundation animals.

The rate of reproduction in private herds devoted to the breeding of fashionable purebreds is usually discouragingly low, so low that the majority of such herds fade out and disappear. The breeding efficiency of such herds is not authentically recorded, for quite obvious reasons.

It has been my privilege to report clinical observations in a large Holstein-Friesian herd under unusually favorable environment for study.[257] In a herd of 55 Holstein-Friesians bred in the northern United States and exported as heifers to the tropics, one group of 17, from a highly fertile herd, averaged 8.4 viable calves per cow while the other 38 females, from 8 separate herds of varying health, averaged 5.8 viable calves. The 17 heifers, coming from an unusually fertile herd, were logically expected to breed well, but the other 38 animals were from herds not then believed greatly superior in genital health to the Wing herd. In their new environment the cows were constantly at pasture, day and night, which doubtless contributed much to their excellent breeding record.

Until late in the nineteenth century, veterinarians, animal husbandmen and governments were primarily concerned with the great animal plagues: rinderpest, foot-and-mouth disease, contagious pleuro-pneumonia and tuberculosis of cattle, which swept relentlessly over state and nation, causing appalling losses and imperilling the food supply of the inhabitants. Finally these have been, or are being brought under control. Meanwhile grave threats to physiological reproduction were being insidiously evolved.

During the past few decades there has arisen great anxiety regarding the diseases associated with *Bacillus abortus*. In 1928 Mohler[144] stated that in the decade "tuberculosis losses have been cut in half while losses from abortion disease have been doubled." Influential veterinary writers have so expressed themselves as to justify the inference that abortion and allied phenomena menace the reproduction of adequate numbers of cattle. More government-employed veterinarians are engaged, at a greater cost per annum, in researches upon contagious abortion of cattle, than have hitherto been devoted to any one disease in the history of veterinary medicine.

Except in areas devastated by war or famine, or overrun by some great animal plague destroying breeding and non-breeding animals alike, there has not been recorded a time when the reproduction of a sufficient number of domestic animals has been in recognizable peril, and there is no reason to believe that such time will come. At present, in the northeastern United States, where *Bacillus abortus* is probably as prevalent and virulent as in any area in the world, many heifer calves are destroyed soon after birth for supposed economic reasons, and the requisite number of adult dairy cows for replacements are imported from other states. Vast numbers of excellent, highly fertile beef heifers are slaughtered annually in order to keep the numbers within economic bounds. Were all the fertile females of any species of domestic mammals mated with fertile sires, far more young would be produced than mankind could use or the world feed.

Modern civilization demands the constant advancement of the individual efficiency of domestic animals and seeks this end through selection of breeding animals, along with improved feeding and care. Conflicting ambitions or alleged economic reasons arise to oppose or wreck the desired progress. This may be best illustrated by dairy cattle, in which genital diseases attain their maximum incidence and virulence. For alleged reasons of economy the dairyman grows his calves artificially in a penurious manner. During the critical period when their reproductive systems are undergoing their final stages of evolution, they are in a low state of health; excessive numbers are crowded together in restricted areas; they are first bred when very immature and quite unprepared to carry safely the unnatural burden; they are forced to yield an abnormal quantity of milk; and while still lactating heavily they are rebred and during a period of six or eight months are required to carry the double sexual load of lactation and pregnancy. Admittedly each of the foregoing practices may be beneficial when prudently applied.

The commercial dairyman turns for his sires and foundation cows to the breeder of purebreds, who assembles large numbers of the best individuals of the breed. These are forced to the utmost in feeding and breeding, and produce young of illustrious pedigree, not infrequently perilously vulnerable to disease. Many such fashionable herds become wrecked from intrinsic causes and the adventurous breeder, bankrupt in so far as his success as a breeder goes, disperses his dangerously diseased stock at public auction and distributes widely the perilous weaknesses and diseases which he has assiduously cultivated through error.

There occurs with regrettable frequency the picture of the steady advancement of dairy efficiency in a family, generation after generation, until a climax is reached in a famous cow, when regression sets in and the illustrious line fades out. The breeder needs then go back to the original level, pick up another family and patiently attempt to rebuild high efficiency. As a rule, for reasons readily comprehended, the advancement of great families is graphically described, but their decline and disappearance omitted. Wing[271] has rendered one outstanding example publicly available in the record of Glista Ernestine, which he rated as the fourth greatest dairy cow of history, as measured by the lifetime production of milk and butter fat. She was of the sixth generation in her line, with definite advancement in milk and fat production in each generation, culminating in her lifetime production of 202,006 lbs. milk and 7,342 lbs. fat. She lived to 16 years and produced 13 calves, of which seven were heifers and were bred. No one of her daughters or granddaughters equalled, or nearly approached her record for life in calves, milk or butter; the line rapidly faded out and is virtually extinct. The example is by no means unique, but there are few instances where authentic records are available for illustration.

The principles involved in the pathology of reproduction in dairy cattle are generally applicable in all species and breeds of domestic animals. Attractive theories have been evolved, apparently logical official regulations for the control of genital diseases have been promulgated, and many wonderful remedies have been announced, but the diseases continue to advance in their destructiveness.

A clinical experience of 64 years, initiated by plunging, without chart or compass, into a stormy sea of equine genital diseases, including the first recorded outbreak of dourine in an English-speaking country, followed by rich opportunities for the clinical study of the pathology of reproduction in horses and cattle of diverse types and located in widely differing climates, and under varying methods of breeding, feeding and handling, has served as a basis for some important conclusions. No one of these appears to me of greater significance than that, ere the ever-increasing incidence and virulence in the genital diseases of domestic animals are to be controlled or mitigated, there is need for broader and deeper study of these, not as they flash before the observer for the moment, but continuous for generations. Without ignoring the immediate welfare of the individual animal and the financial interests of its owner, the destiny of the family or breed should be placed in the foreground. A fleeting vision of cure may follow the dislodgment of a corpus luteum, mas-

sage of the uterus, disinfection, the administration of minerals, vitamins, hormones, vaccines or other spectacular panacea; or genitally diseased animals may be isolated or slaughtered. The urgent demand, however, is that adequate measures be applied which may advance the vigor, longevity, efficiency, and reproductive power of the most desirable families.

SECTION I

THE BASIC CAUSES OF INTERFERENCES WITH REPRODUCTION. THE TERATOLOGY AND PATHOLOGY OF THE REPRODUCTIVE CELLS. HEREDITARY AND CONGENITAL DEFECTS

CHAPTER I

HYBRIDIZATION

THE foundation for physiological mammalian reproduction is the union of a mature, healthy spermatozoon with a perfect ovum, both cells emanating from the same species. In nature, the union of the male with the female cell is dependent upon coitus. The contact with the cells may be brought about by artificial insemination.

If a domestic mare (*Equus caballus*) be mated with a male ass (*Equus asinus*) conception, gestation and parturition commonly follow the same as when the two individuals are of the same species. The coitus between the mare and ass is unnatural, the mare being forcibly restrained. The resultant mule is noted for its strength, endurance and longevity. All males, and most females are unfertile.

When a domestic cow (*Bos taurus*) is mated with a bison bull (*Bison Americanus*), conception occurs, followed by amnio-allantoic dropsy, which seriously imperils the lives of both the fetus and mother. At parturition dystocia is the rule and almost all male fetuses perish. The few male hybrids which survive are sterile. The female hybrids are moderately fertile when mated with a male of either parent species. Boyd,[17] who bred some 30 hybrids, attributed the dystocia to the large chest and prominent withers of the hybrid fetus. Dystocia is essentially constant, however, when amnio-allantoic dropsy is present, whether the fetus is large or small. There are several groups of teratological and pathological fetuses associated with such dropsy.

Extreme Outcrosses

BULLDOG CALVES

When breeds or families of domestic animals are crossed, the progeny are often superior in size, quality, and fertility to either of the parent stocks, but this is not constant. The genealogy of domestic cattle is lost in antiquity. There exist two fairly distinct types —the longhorns, with long legs and slender bodies, and the shorthorns, which have short legs and compact bodies. The two types cross readily but not always satisfactorily.

Dexter cattle have been largely derived from the Kerry breed. It

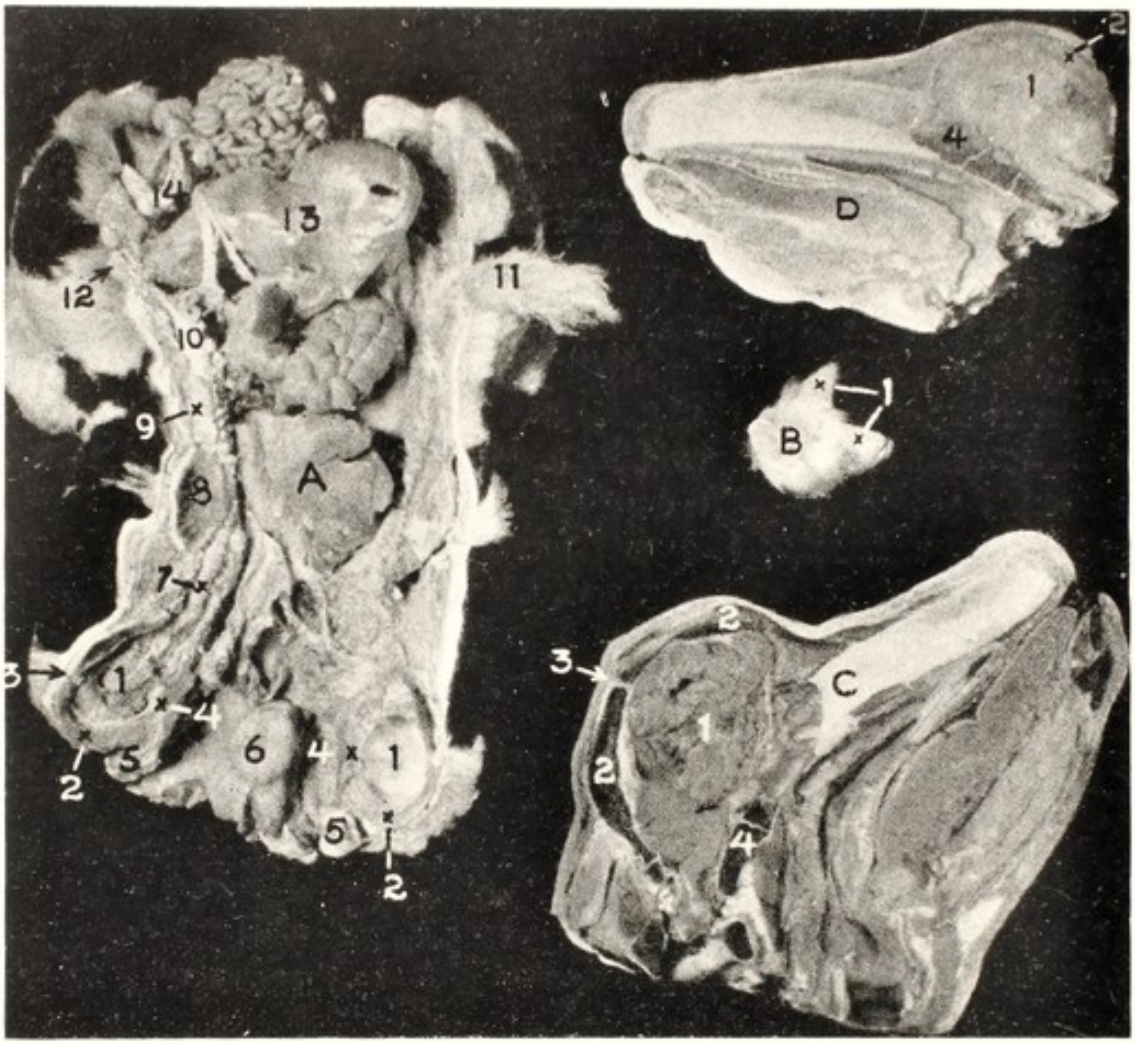


FIG. 1. Bulldog Calf.

A, A non-viable bulldog fetus incised longitudinally along the mid-dorsal line. 1, 1, The cranial cavity; 2, 2, the greatly thickened, ivory-like cranial vault; 3, the parieto-frontal fontanelle; 4, the cranial base, very short and devoid of inter-vertebral cartilages or divisions; 5, 5, the nose; 6, tongue; 7, cervical area of spinal cord; 8, superior spinous processes of the thoracic vertebrae, forming a single fluted plate of ivory-like bone; 9, superior spinous processes of the lumbar vertebrae; 10, sacral spinal cord; 11, tail; 12, anus; 13, large colon; 14, urinary bladder and distended vagina. *B*, A pair of toes from *A*. *C*, A viable bulldog calf. 1, Cranial cavity; 2, 2, grossly thickened, cancellated cranial walls; 3, parieto-frontal fontanelle; 4, cranial base with indistinct inter-vertebral cartilages. *D*, Sagittal section of a normal calf cranium for comparison. Note the thinness of the cranial walls, 2, and the prominent inter-vertebral cartilages, 4. *(Cornell Veterinarian)*

is believed by some that there was an outcross with the Devon cattle. However brought about, the Dexter is an excessively compact animal. It was sought to fix the Dexter type by mating Dexter with Dexter, but according to Crewe[36] this leads to the production of so large a ratio of *bulldog calves* that the practice is rendered economically impossible. It is found, however, that typical Dexters may

be successfully produced by mating Dexter with Kerry. As in the American bison-domestic cow cross, so in the Dexter-Dexter mating associated with bulldog calves, there is amnio-allantoic dropsy which seriously imperils the lives of both fetus and dam. Carmichael[25] studied 13 bulldog calves in Nganda cattle, said to have been developed by crossing Eastern Province with Angola cattle. The former are zebus with short horns and legs, and a hump; the latter have horns with a spread as great as 6 feet. All three breeds are maintained upon the one estate, with all bulldog calves in the Ngandas. Eleven of the thirteen were firstlings. Amnio-allantoic dropsy is not recorded.

TUX-ZILLERTALER CATTLE

Adametz and Schulze[3] state that the Tux-Zillertaler cattle of the Austrian Tyrol were developed by crossing the compact Simmental with the slender Pinzgau breed. The animals of the new breed were semi-monstrously compact and amnio-allantoic dropsy, with dystocia, was rampant. In one small commune, twelve lay obstetrists were required for aiding the cows at calving. Dropsy of the amnion and allantois were constant, so that the owners reported that when the waters broke the stable was flooded. The commune changed to Fleckvieh, and the lay obstetrists were no longer needed.

Solitary bulldog calves occur in all countries and breeds. When they appear in notable groups they seem to have the common historical background of an extreme outcross, probably followed by inbreeding in order to fix the new type. Almost all bulldog fetuses are dead when expelled or extracted, or are non-viable. An individual like *A* in Fig. 1 is absolutely non-viable after interruption of the umbilical circulation, because, in the absence of ribs and sternum, respiration is impossible.

The conclusion becomes justifiable, if not inevitable, that bulldog calves arise from the union of two genetically inharmonious sex cells, either of which, uniting with a genetically compatible cell, would probably produce physiological young, not associated with amnio-allantoic dropsy or dystocia.

Chapter II

INBREEDING

ONE of the chief tenets in the advancement of the quality of purebred animals is the mating of individuals having some degree of consanguinity. During the initial stages in the founding of a new breed or family, inbreeding constitutes the shortest path to the stabilization of type. Purebreds inevitably trace back to a common ancestry. The older the breed, the greater the consanguinity of its individual members. Once the character of a breed or family has become fixed, many influential breeders advocate inbreeding, or the lesser consanguinity of line-breeding. Wing[271] practised a limited degree of inbreeding, largely as a matter of convenience. The pregnancies following inbreeding, so far as readily followed, show 60.9 per cent of viable calves, compared with 73.1 per cent in those not inbred. The interpretation is rendered uncertain, because the inbreeding was largely among primipara, in which, among dairy cattle, the ante-natal and early post-natal mortality is far higher than among pluripara. The progeny of the inbreds apparently show a male-female ratio of 100:60. Of 14 inbred heifers, 7 show one pregnancy each and the other 7 an average of 3.9. The ratio of twin gestation in the herd was 1:45 and for the inbreds 1:17. A more impressive example is provided by the once famous Duchess family of shorthorns as related by Sanders.[175] That family, under unparalleled inbreeding, forged rapidly to the front and monopolized for a time the prizes at the leading cattle shows in Great Britain and the United States, only to fade out completely a few years later, leaving the perpetuation of the breed to other less famous families, in which inbreeding had not been extensively practised.

The discussion of the valuable results due to prudent inbreeding is left to geneticists and animal husbandmen, considering here only those sinister consequences which frequently appear.

Quite recently, attention has been given to teratological studies of an important group of monsters or defectives under the designation of *inherited lethal characters,* occurring chiefly in connection with incestuous mating. In this group the teratological defect renders the individual non-viable outside the uterus. The parent animals are not recognizably imperfect; they apparently produce physiological spermatozoa and ova, which when united with cells from unrelated

animals tend to produce perfect young, but when incestuously mated, e.g., sire-daughter, the product of the union is a non-viable monster. The phenomenon may be regarded as the antithesis of the American bison-domestic cow hybridization. The examples are numerous.

Muscle Contracture Monsters

Important group losses, associated with inbreeding, are recorded in dairy cattle and in sheep, due to a type of monstrosity commonly designated *muscle contracture* monsters. Solitary cases, not always recognized as having been linked with inbreeding, have long been known to veterinary obstetrists as a cause of severe dystocia in cows. The monstrosity has also been designated as contraction of the tendons, anchylosis and mummification. Careful study fails to reveal the presence of any one of the abnormalities named. The available evidence indicates that, excepting those muscles supplied by the cranial nerves, none have at any time been endowed with contractile power. All muscles supplied by motor nerves from the spinal cord

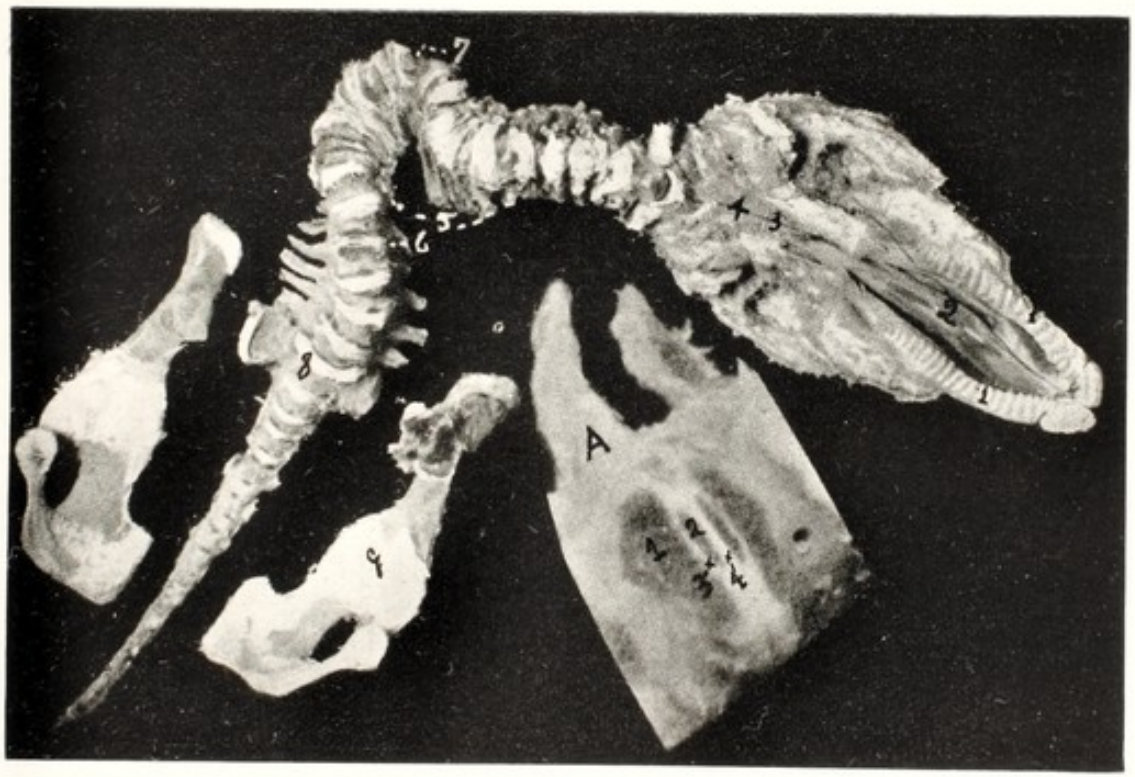


FIG. 2. Cerebro-spinal Axis of Bovine *Muscle Contracture* Monster, with Extreme Flexure of the Thoracic Spine.

Monster devoid of intervertebral articulations. 1, 1, Palatine borders of the maxillae; 2, turbinated bones; 3, basi ethmoid; 4, basi sphenoid; 5, ribs I-XIII; 6, superficial section from the ventral surface of lumbar vertebrae II and III, which is enlarged to constitute inset *A*; 7, traction fracture of the spinal column during delivery; 8, sacrum; 9, os innominatum. *A*, Inset from lumbar vertebrae II-III, enlarged. 1, Diaphysis of II; 2, epiphysis of II; 3, line of demarcation between diaphysis and epiphysis, showing traces of cartilage; 4, cartilaginous line between vertebrae. *(Cornell Veterinarian)*

are identical in appearance with the muscles of the pelvis and pelvic limbs of that type of monster in dairy cattle, known as *Perososomus elumbis*. In this monster the spinal cord terminates in the posterior dorsal, or anterior lumbar region, wholly depriving all muscles posterior thereto of motor nerves. The designation of *anchylosis* is equally inept, there being no visible evidence that any developed articulation has been destroyed. The patella, one of the last skeletal bones formed, remains an integral part of the femur. The spinal column remains as a single bone throughout most of, or its entire length, as indicated in *A*, Fig. 2. In all cases of this monster coming under my observation, there has been an absence of the palate, as shown in Fig. 3, which justifies the conclusion that the arrest in the muscle development occurred in an era of embryonic life prior to the formation of the palatine bone.

The duration of gestation is about normal and the skeleton attains approximately its usual length, but the gross arrest of the muscular tissue renders the monster subnormal in weight and volume. The

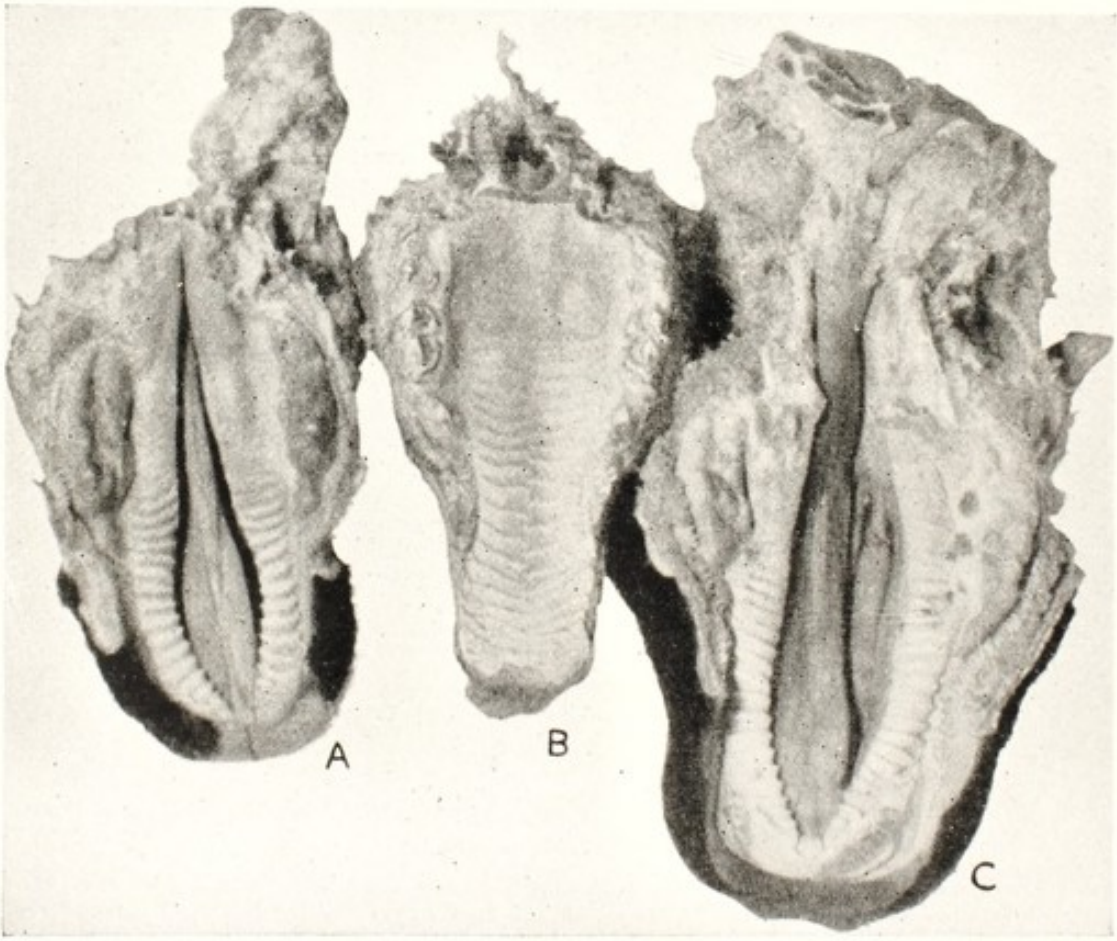


FIG. 3. Heads of *Muscle Contracture* Monsters Illustrating the Absence of Palate in the Monsters, *A*, *C*, as compared with the normal, *B*. *Note that the dentition is backward in the monsters. (Cornell Veterinarian)*

deformities of the spinal column and of the extremities cause severe dystocia, which necessitates fracture or other division of bones in delivery. Copious fresh hemorrhages at the point of fracture of bones, in all cases I have studied, testify that the fetus was alive when extraction was begun. The absolute rigidity of the chest walls, in almost all cases, renders respiration impossible when the umbilical circulation is interrupted. All cases of this monster observed by me or found recorded in cattle have occurred in the purebred dairy breeds and have been definitely linked with incestuous breeding, largely sire-daughter mating. They have largely occurred in primipara, perhaps because in many cases the breeder uses his sire upon the daughters as a matter of convenience, pending the securing of a new sire. Furthermore, immature primipara are more prone than pluripara to produce teratological and pathological young.

Löje[123] records 10 cases by one red Danish dairy bull, three of which followed sire-daughter mating. Amnio-allantoic dropsy was constant. Hutt[97] records five cases, three of sire-daughter mating and all inbred. Amnio-allantoic dropsy was apparently present. Two cows perished from the dystocia. The case illustrated in Fig. 2 followed sire-daughter mating.

Roberts[171] observed over 60 cases in a flock of purebred sheep in which extensive inbreeding was practiced. Amnio-allantoic dropsy prevailed, with dystocia and a high mortality of ewes.

Atresia of the Colon. Hydrocephalus. Cerebral Glioma

Atresia of the colon has been recorded only in the horse and has been best described by Yamane.[276] Less extensive observations have been made by Nusshag.[153] Yamane's studies were based upon a high-class Percheron stallion purchased in Ohio and exported to the island of Hokkaido, Japan. His progeny were excellent, and inbreeding was practiced among them. All went well until the third generation, when astresia coli appeared among the inbreds, and in the next generation glioma and hydrocephalus were noted among the animals affected with atresia. Yamane studied 25 cases, all of which traced back on both sides to the imported stallion. Aside from the defects cited, the foals were well developed, and gestation and parturition normal. As a rule the foals failed to stand up after birth and soon showed symptoms of obstruction colic. The defect was uniformly in the ascending colon, with a complete hiatus in both gut and mesentery.

Excessive Ratio of Males

CLEFT PALATE

McPhee, Russell and Zeller[132] found that the inbreeding of swine led to a prompt decline in the average litter size, and in the average birth weight of pigs, associated with a gross increase of the ratio of males and numerous cases of cleft palate and cryptorchidy. In marked contrast, Winters, Kiser, Jordan, and Peters[272] found that by cross- instead of inbreeding, they increased the litter size, the average weight of pigs, and their growth per unit of food.

Woodward and Graves[274] have reported that in researches in inbreeding of cattle, 57 pregnancies in their foundation cows yielded 10.5 per cent abortions and 89.5 per cent viable calves. Twenty-two of the heifer calves mated with their sires, with a total of 48 pregnancies, yielded 39.6 per cent abortions, 2.0 per cent dead at term, 12.5 per cent non-viable monsters and 45.8 per cent viable calves. The inbred calves were subnormal in birth weight and showed a high mortality in calfhood.

Persistence of the *Right* Instead of the *Left* Primitive Aorta

Milks and Williams[138a] studied five dogs in which the right, instead of the left primitive aorta persisted, to constitute the permanent aorta. This made it necessary that the *Ductus arteriosus* pass over the trachea and esophagus in order to reach the aorta. When the pups began to consume solid food, deglutition was barred and the monsters succumbed to starvation. Three of the five cases came from one small kennel of Boston bulldogs and at least two of them were by one sire. The same kennel has supplied three cases of hypospadias.

Color and Fertility

Clinicians well know that grey horses—those born black which later turn white—frequently develop benign melanotic tumors which may mechanically imperil health and fertility. Later the melanoma may become the seat of a malignant neoplasm and terminate the reproductive and physical life. Thus, step by step, the hereditary color of the hair, its loss of black pigment with age, with the advent of melanotic tumors of benign character, may lead eventually to sterility and death.

In some portions of the Rocky Mountains, the Cascade Range, and the Hawaiian Islands, important losses of cattle occur from palpebral carcinoma. In these areas there occur prolonged periods of drought, with high winds which carry much dust, especially sharp

dust from lava soils, irritating the eyes and causing profuse and prolonged lachrymation. The cancers appear mostly in those individuals having white eyelids. The albinotic eyelids, distinctly hereditary, are of no significance in most areas, but in certain localities lead to disease which may prematurely terminate the breeding, and the physical life of the individual.

An attempt was made to found an albino stud at Fredriksborg, Prussia. The breeding records have been carefully studied by Wriedt[275] who reports that when pure albino mares were bred to partial albino stallions, the reproduction was satisfactory: when the pure albinos were mated, the fertility promptly dropped. Near the close of the eighteenth century the stud was in peril of extinction owing to sterility. Thirteen albino mares mated with albino stallions produced one foal per annum; mated with imperfect albino stallions they dropped 10 foals. Tortoiseshell male cats are usually infertile.

Cerebral Hernia

In 1909 I described briefly a destructive incidence of cerebral herniae in pigs[231] in the swine herd of the Agricultural College at Cornell University. The brain was herniated through the fontanelle, as indicated in Fig. 4, in some cases covered with hairy skin, in others hairless. Convulsions were common and the mortality high. Inbreeding was not apparent. The defect grew in frequency, until it was found necessary to discard the family. Nordby[151] studied the defect in five purebred herds, comprising 200. It was generally lethal and was associated with epilepsy. It was distinctly hereditary. Hughes and Hart[94] studied four cases in the University of California herd, three of which followed sire-daughter mating and the other the mating of two closely related progeny of the herd boar.

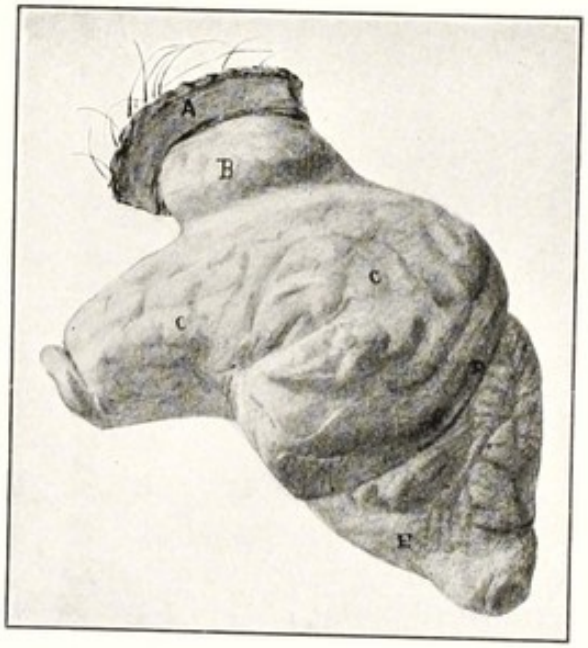


FIG. 4. Cerebral Hernia of Pig, Left Lateral View.

A, Segment of skin, covering the herniated portion of the cerebrum, *B*; *C*, *C*, cerebrum; *B*, cerebellum; *E*, medulla oblongata.

Umbilical Hernia

During early embryonic life portions of the abdominal viscera, especially the liver, normally lie outside the abdominal cavity within the umbilical cord. As gestation advances they move into the abdominal cavity and the umbilical ring closes intimately about the navel vessels. Teratologically the ring may fail to close. Not infrequently portions of the small intestines or of the omentum protrude through the umbilical ring, covered only by the skin, to constitute ordinary umbilical or navel hernia. The size of the ring may vary widely. In the foal and calf it is ordinarily 1 to 3 or 4 inches in diameter. Exceptionally the hernial ring may have an antero-posterior diameter of 12 inches or more and the hernial sac may contain intestines, liver or stomach.

The defect is unsightly and the herniated organs may become strangulated and undergo necrosis. When the hernia is very large, especially in the bull, copulation is barred, the protruded penis being deflected from its normal course. In rare cases the herniated organs are not covered by skin (Schistocormus) and the organs—intestines, liver, etc.—are naked. Such defects, according to my observations, are largely accompanied by malformation of the urino-genital system.

Teratological Defects of the Reproductive System

So far as may be determined, more domestic animals are born with important defects of the genital, than of any other system of organs. They are conspicuously hereditary. Some lower fertility; others bar reproduction. The anlagen for the genital organs are among the last, if not the last to be laid down in the fertilized ovum. I have elsewhere discussed the fact[252] that the reproductive systems of most monsters are conspicuously abnormal. In these the basic monstrosity excludes the possibility of reproduction in most instances. Of far greater economic importance is the vast array of primary genital aberrations in otherwise normal animals.

The reproductive organs provide unequalled opportunity for an infinite list of abnormalities. The system is divisible into three zones: 1, the gonads (testicles and ovaries) which produce the spermatozoa and ova; 2, the tubal zone comprising the derivatives from the Wolffian ducts (epididymes, vasa defferentia, vesiculae seminales) and from the Muellerian ducts (Fallopian tubes, uterus, cervix); 3, the copulatory area (penis and clitoris, scrotum and vulva). Physiologically, only the organs of one sex (except for unimportant vestiges

of those of the opposite sex) may be present in a given area and the sexual character of the organs is alike in all three zones.

Teratologically the gonads may be absent, or neuter, as is regularly observed in freemartins; one may be male and the other female, or a gonad may contain both tubuli seminiferi and Graffian follicles. In the latter instance neither normal spermatozoa nor ova form, so far as known. In the presence of both testicles and ovaries, a few instances are recorded in which the ovaries have functioned, followed by conception and birth.

In the second zone there may be absence of the derivatives of both Wolffian and Muellerian ducts, or both vas deferens and uterus may be present in variable degree of arrest, but only one opening to the exterior. The organs of the copulatory zone are inevitably unisexual. The genital tubercle can form only a penis or clitoris, or a compromise between the two incapable of performing the function of either; the genital folds form either scrotum or vulva (labial majora), or a compromise between the two. In swine and the dog, if the inguinal canal remains open, the ovary may migrate to the supramammary region or even to the vulva.

The gonad zone may be male, the tubal area female, and the copulatory organs a compromise between the two sexes. Thus in the highly illustrative example of the equine hermaphrodite there are two definite, though imperfect, testicles located in the normal ovarian area, or imperfectly descended into the inguinal canal; in the tubal region a normal uterus, cervix and vagina; and in the copulatory zone a vulva of ordinary size and form, while from between the two labiae there projects a compromise erectile organ which can not properly be designated as either clitoris or penis. The behavior of the monster is erratic, like that of the cryptorchid stallion. No scrotum forms.

The dual character of the reproductive system invites a riot of teratological defects, associated with highly interesting problems in genetics, animal husbandry, embryology and clinical veterinary medicine.

Inguinal or Scrotal Hernia in the Male

The inguinal ligament, or gubernaculum testis, of the male embryo extends from the testicle through the inguinal canal to the fundus of the scrotum. Early during gestation, in most species of domestic animals, the testicles descend into the scrotum preceded by the processus vaginalis of the peritoneum. The walls of the inguinal canal so

closely invest the spermatic cord that intestines and other viscera are prevented from passing through the internal inguinal ring. Normally, unlike in man, the parietal peritoneum does not fuse with the contiguous visceral layer of the cord, but remains open throughout life, the scrotal sac being continuous with the peritoneal cavity. If the internal inguinal ring be abnormally open, viscera protrude and, frequently becoming strangulated, imperil the life of the defective animal. Inguinal hernia is especially perilous to breeding males at the time of coitus: the posture of the body and the exalted intra-abdominal pressure tend to force an intestinal loop through the open inguinal ring, followed promptly by strangulation.

Inguinal Hernia in the Female

The round ligament of the ovary normally atrophies and disappears as a distinct structure at the internal inguinal ring and a patent inguinal canal does not develop. Teratologically the round ligament may extend through the inguinal canal to the supramammary region (ruminants) or to the vulvar zone (sow and bitch) accompanied by the processus vaginalis from the peritoneum as in the male. Into this open canal the ovary and oviduct necessarily follow, accompanied by the ovarian end of the uterus. Possibly in some cases intestine or omentum also become herniated. The ovary and oviduct become halted in the supramammary region in ruminants but may pass to the vulvar zone in the sow, where they may be mistaken for a testicle.

If the malformation escapes identification or its significance is ignored, and the female becomes pregnant, a fetus may be located in the inguinal canal. When parturition approaches, the inguinal fetus is incarcerated and the life of the female imperilled. The dystocia may be overcome by timely hysterotomy. If the female survives, she and any living young should be discarded as breeders.

Umbilical and inguinal herniae are among the commonest and economically most important hereditary defects of domestic animals. Their incidence is greatly advanced by inbreeding. They occur most frequently in horses and swine, but are not at all rare in other species. In a high-class Percheron stud, in which the defect had previously been unknown, I observed the occurrence of about 90 per cent of umbilical herniae following the introduction of a new sire without visible signs of the defect.

After a careful study, Warwick[219] found the incidence of herniae in swine enormously increased by inbreeding. He emphatically condemns the use for breeding of any animal of either sex which is or

has been affected with hernia, or is closely related to such individual.

The peril to the health or life or the individual from hernia may be removed by surgical operation, but the danger of hereditary transmission cannot be overcome except by the discard of the defective animal as a breeder.

PENIAL DEFECTS

In rare cases aberrations in the development of the penis incapacitates the animal for copulation. I attended one young bull in which the penis was somewhat chisel-shaped and directed upward when protruded, instead of being acutely conical and directed slightly downward. A loose, baggy fold of skin about the umbilicus, caught by the penis, formed a pocket in which the glans lodged, preventing copulation.

Richter observed defective development in the penis of male goats, especially an arrest in the development of the sigmoid flexure, rendering the male incapable of protruding the penis.

As a rule the impotence of the male to copulate cannot be overcome by known surgery. If it were possible thus to remedy the defect, it would be highly undesirable owing to the danger of the transmission of the defect to progeny.

CRYPTORCHIDY

The testicles of domestic herbivora migrate from the abdomen into the inguinal canal or scrotum before birth; the testicles of carnivora after birth. Teratologically they may remain permanently in the inguinal canal or abdomen to constitute cryptorchidy, one of the most frequent and economically important arrests in development in domestic animals. Like scrotal hernia, it is most common in horses and swine. It is interesting to note that cryptorchidy is extremely rare in the mule. I have not observed a case and several colleagues have had the same experience. Professor Frick states that he has not observed a case during twenty years in the clinic of the veterinary school at Kansas State College, in which many mules are entered. Other veterinarians have recollections of single cases. Yet the testicles of this hybrid are uniformly arrested in development to such degree that the mule is sterile.

Cryptorchidy renders castration more difficult, dangerous and expensive. The uncastrated cryptorchid horse largely becomes mentally deranged, is difficult to handle, and may become an outlaw. The flesh of the cryptorchid boar is so repulsively flavored that it is not edible.

I have observed 75 per cent of cryptorchidy in the progeny of a monorchid ram. Clinical observation dictates the conclusion that it is definitely and dangerously hereditary and should, as a rule, be attributed to genetic defects in the spermatozoa or ova—not to some pathogenic force acting upon the embryo during pregnancy. There may be exceptions. Most male monsters are cryptorchid. Individual cases occur in which a monorchid sire is said to have produced no cryptorchid young. Even so, the absence of the cryptorchidy may well be attributed to the vigor of the dam. When both testicles are abdominal, no spermatozoa are formed and the male is infertile. There occur, chiefly in horses, dermoid cysts of the testicles, containing hair and dental tissue; the tumors are regularly abdominal.

In man, cryptorchid testicles exhibit a marked tendency toward the development of malignant growths in the undescended glands. This is almost unknown in domestic animals. I have met with one case of cryptorchidy in a pig in which malignant new growth was suggested by clinical appearances.

Imperfect Descent of Testicles Associated with Gigantic Teats and with Defective Uterine Caruncles in Female Progeny

I have recorded[259] an example of a highly bred Guernsey bull of normal fertility, with testicles incompletely descended, apparently because of an abnormally high fixation of the gubernaculum testis, which led the upper end of the testicle to tilt forward, so that the scrotum hangs obliquely downward and backward. His teats are gigantic, approximately the size of those of a mature heifer. Thus far, all his get, of both sexes, have gigantic teats, as illustrated in Fig. 5, and all male progeny have tilted testicles. Two of his female progeny—one a fetus expelled dead at term, the other a calf a few days old—have been autopsied, and both showed definitely abnormal uterine caruncles. The fetal membranes of one heifer by the defective bull have been available for study. Her afterbirth was virtually free from pathological changes. Teratologically, the membranes clearly indicated gross abnormalities in the uterine caruncles. They were irregular in number and arrangement and extremely variable in size. Some caruncles near the apices of the cornua, where they should be small, were gigantic instead, while many of those near the bases of the cornua, where they should be of maximum size, were dwarfed.

The indications point to pernicious hereditary transmission of the defects. It is reliably reported that the giant teats constitute a definite family peculiarity and that several of the bulls have lactated.

The associated defective uterine caruncles are of special interest. The caruncles constitute the organs of nutrition to the intra-uterine young; the mammae furnish milk for the new-born. In these defective animals the arrests in development were essentially parallel in uterine caruncles and mammary glands. This suggests that, when the

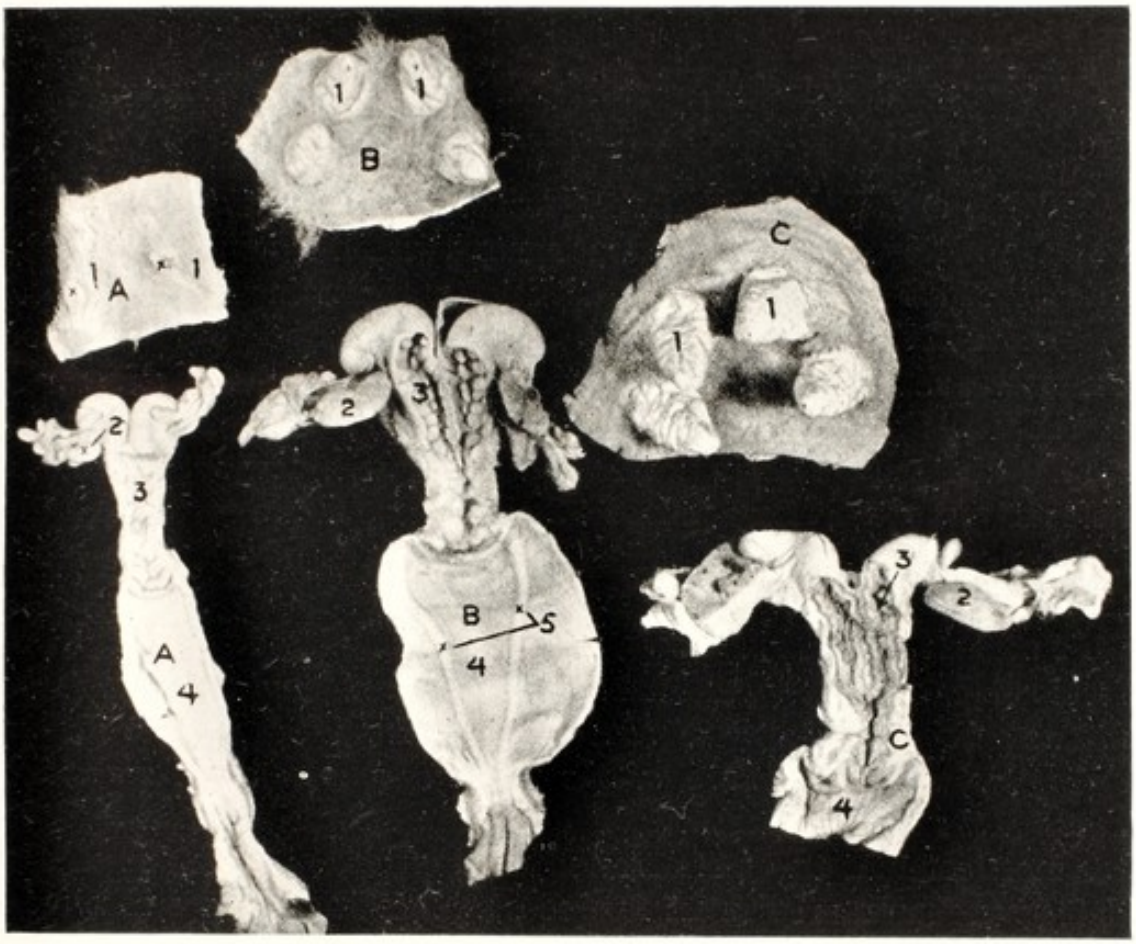


FIG. 5. Teratological Reproductive Organs of the Female Progeny of an Inbred Bull, with Those of a Normal Calf, *A*, for Comparison.

The teats of *A*, 1, 1, are inconspicuous, while those in *B* and *C* are gigantic. The ovaries, 2, are also gigantic in the defectives, containing hemorrhagic masses resembling corpora lutea in the fetus, *B*, expelled dead at term, and in the heifer calf, *C*, dead at 30 days. The uterine caruncles, 2, are gigantic in *B* and *C*. The vagina, 4, of *B* is grossly distended and Gartner's ducts, 5, are greatly enlarged. *(Cornell Veterinarian)*

mammae show teratological defects, the uterine caruncles may well be correspondingly abnormal, lowering the nutritive supply to the fetus and depressing the reproductive power of the cow. Hence, when such external defects are present, the breeder and veterinarian may well be on guard, lest important imperfections exist in other organs of the reproductive system. The ovaries in *C* of Fig. 5 are also teratological, showing in the left one hemorrhages in follicles similar to corpora lutea. Cysts are also present.

Such ovaries are not rare in sterile heifers. The uterus from an incurably sterile heifer, in a highly fertile herd, had similar, though larger, ovaries. There were many small cysts and corpora lutea. The surface of the ovaries was rough and pitted in a manner to suggest the rupture of innumerable small cysts followed by hemorrhages or by small corpora lutea.

Arrested Development of Mueller's Ducts. Uterus Unicornis. Uterus Didelphys

Fincher and Williams[53] have recorded an instructive example in which a highly regarded sire was mated with his daughters during three successive years. When the heifers of the incestuously bred progeny arrived at breeding age it was found that 56 per cent of them were absolutely sterile because of arrests in the development of the ducts of Mueller, an additional 13 per cent were virtually sterile, and the remaining animals were of very low fertility. The defects were mainly arrests in the development of the uterus, including the cervix. No defects of the organs of reproduction could be found in the sire or in any one of his unrelated females. No defects were seen in any of his progeny out of the unrelated cows. The defects were extremely variable. They were chiefly classifiable as *uterus unicornis, atresia of one or both cornua at the base, uterus didelphys, and persistence of the median walls of the Muellerian ducts at the os uteri externum*. The variations represent on the whole the most common types of arrested development in these ducts. The defective genital systems of five of the inbred heifers are shown in Figs. 5 and 6. Similar defects are possible in all species but are by far most frequent in dairy cattle. Uterus unicornis and uterus didelphys are among the most common defects in the derivatives of the Muellerian ducts. Uterus unicornis may apparently be of two distinct types. The most common type is that illustrated in Figs. 7 and 8, in which both ovaries are present in their normal position, while one cornu is represented by vestigial bands of connective tissue. Very rarely it appears there may occur a definite single ovary, uterus and vagina upon the median line. Thus Higgins[90] reports having spayed a bitch in which he found a single ovary and uterus along the median line. The occurrence is supported by the case illustrated in Fig. 7, in which there was a single kidney and ureter located upon the median line.

The cases offer the interesting suggestion that the defects did not have their origin in the urinary or genital organs but were in-

heritances from aberrant Wolffian systems. It is to be borne in mind that, according to the teachings of embryology, both the urinary and reproductive systems are largely derived from the Wolffian bodies and ducts. If these are morphologically abnormal it can not well be expected that the organs largely derived therefrom will be free from aberrations; if there be a single median Wolffian body, a single

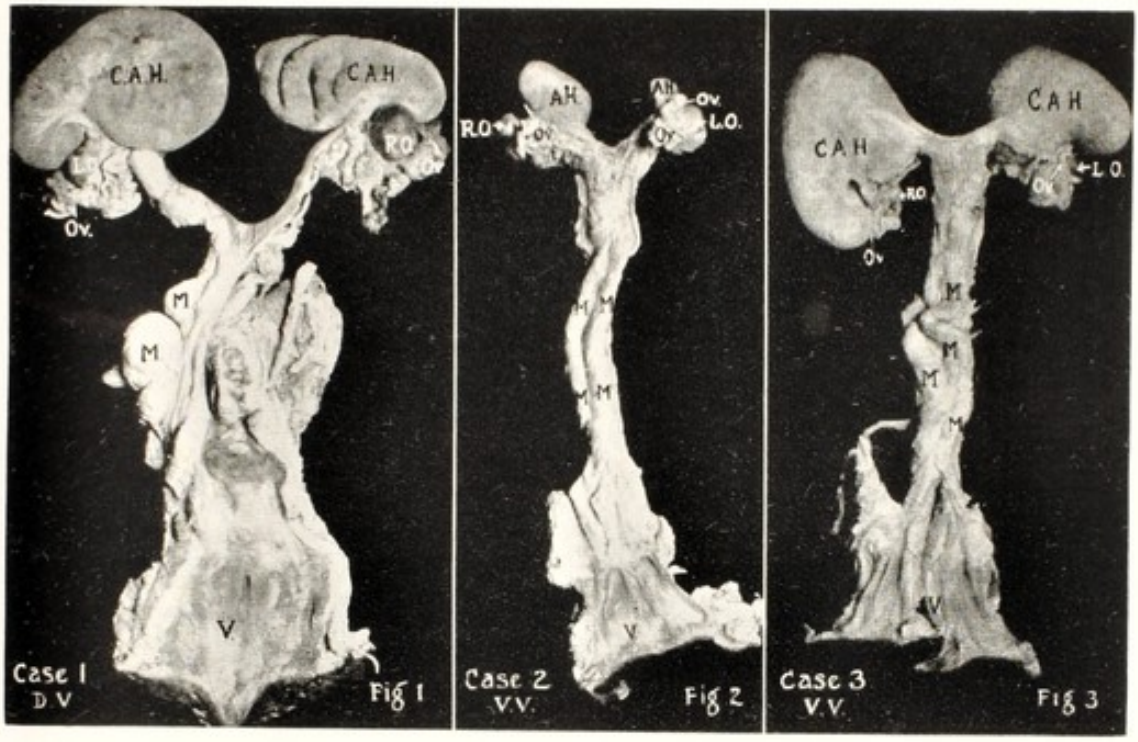


FIG. 6. Defective Reproductive Organs from Three of a Group of Twenty-three Daughters from Sire-Daughter Matings.

AH, Apex of uterine cornu; *CAH*, cystic apex of uterine cornu; *RO*, right ovary; *LO*, left ovary; *OV*, oviduct; *M*, arrested Muellerian duct represented by a fibrous cord; *V*, vagina. *(Cornell Veterinarian)*

median kidney or gonad with a single median duct may be anticipated. Taking this view, the single ovary and uterus are not the result of fusion of two ovaries and uteri, but like the cyclops monster are due to defects antedating the origin of the aberrant organs.

In the example of equine uterus unicornis described by Williams and Fincher[263] the abdomen was completely open, with all abdominal viscera naked. Only one kidney and ureter was present. I observed six examples of uterus unicornis in a series of 3000 abattoir cows, five of which had been pregnant. The breeding efficiency of such cows is unknown, but is probably very low. The defect should be regarded as hereditary and the involved individuals discarded. Williams and Fincher[258] describe a case of uterus unicornis with an abnormal cervix, the cranial end of which projected into the cornual cavity in



FIG. 7. Equine Uterus Unicornis.

1, 1, The ovaries; 2, 2, the pavilion of the oviduct; 3, right oviduct; 4, ovarian pocket; 5, 5, right uterine cornu; 6, fibrous band representing the left uterine cornu; 7, uterine body; 8, uterine wall without endometrium opposite the base of the vestigial left cornu; 9, cervix; 10, hymen; 11, meatus urinarius; 12, vagina; 13, clitoris; 14, urinary bladder laid open along the median ventral line; 15, the single ureter from the single kidney; 16, meatus of the ureter on the mid-dorsal line.

such a manner that it caused imprisonment of the estrual debris, as shown in Fig. 8. Each of the two defects occurs singly. I have handled

for sterility, without success, cases of the abnormal projection of the cervix with uterine distension.

Practically speaking, domestic ruminants have a double uterus



FIG. 8. Bovine Uterus Unicornis with Deformed Cervix, Causing Retention of Estrual Debris.

1, 1, False uterine seal, simulating pregnancy; 2, ovarian end of cervix projecting abnormally into the cornual cavity; 3, 3, estrual debris imprisoned within the left (single) cornu; 4, vestiges of arrested right cornu; 5, right ovary with enlarged, retained corpus luteum; 6, left ovary with a freshly ruptured ovisac. *(Cornell Veterinarian)*

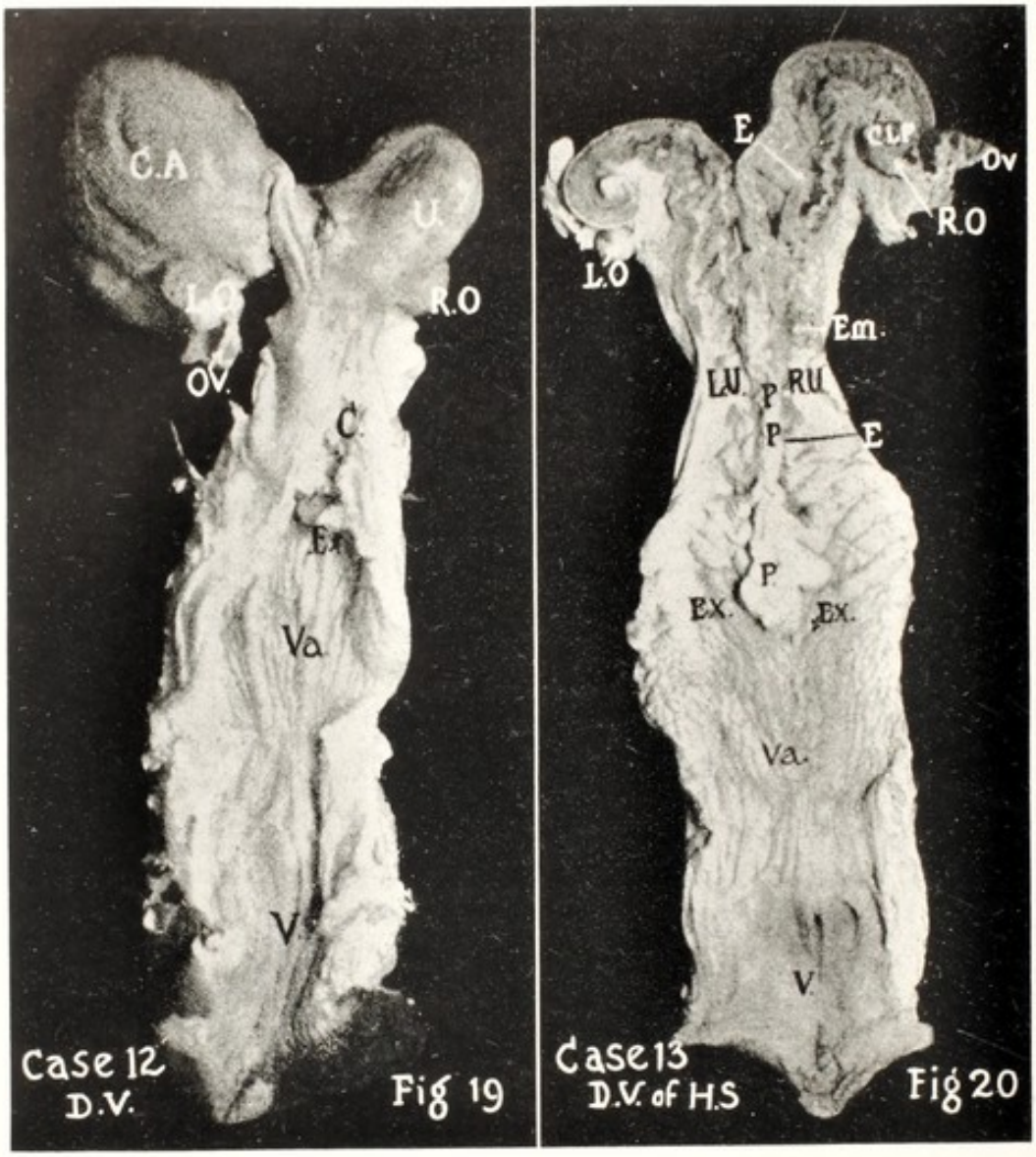


FIG. 9. Double Bovine Uterus, from Same Group of Heifers as Those Shown in Fig. 6.

RO, Right ovary; *LO*, left ovary; *U*, uterus; *OV*, ovary; *CLP*, corpus luteum of pregnancy; *LU*, cavity of left uterus; *RU*, cavity of right uterus containing embryo, *E*; *PPP*, fused median walls of Muellerian ducts; *EX*, *EX*, external openings of the two uteri.

with single cervix. Teratologically the double uterus of the cow is occasionally associated with a double cervix, due to the failure of the median walls of the Muellerian ducts to become resorbed in the cervical area, as shown in Fig. 9. The cow was pregnant when slaughtered. W. W. Williams personally relates having been called to attend a cow which had been bred on the previous day. He removed a dead fetus from the vagina. The uterus was didelphic with distinct cervices. The cow had been pregnant in the left uterus, and

as occasionally occurs, the corpus luteum atrophied with the death of the fetus, an ovisac ripened in the right ovary, and estrum, copulation and incomplete expulsion of the fetal cadaver followed. Both uteri were douched. Fertilization occurred in the right uterus

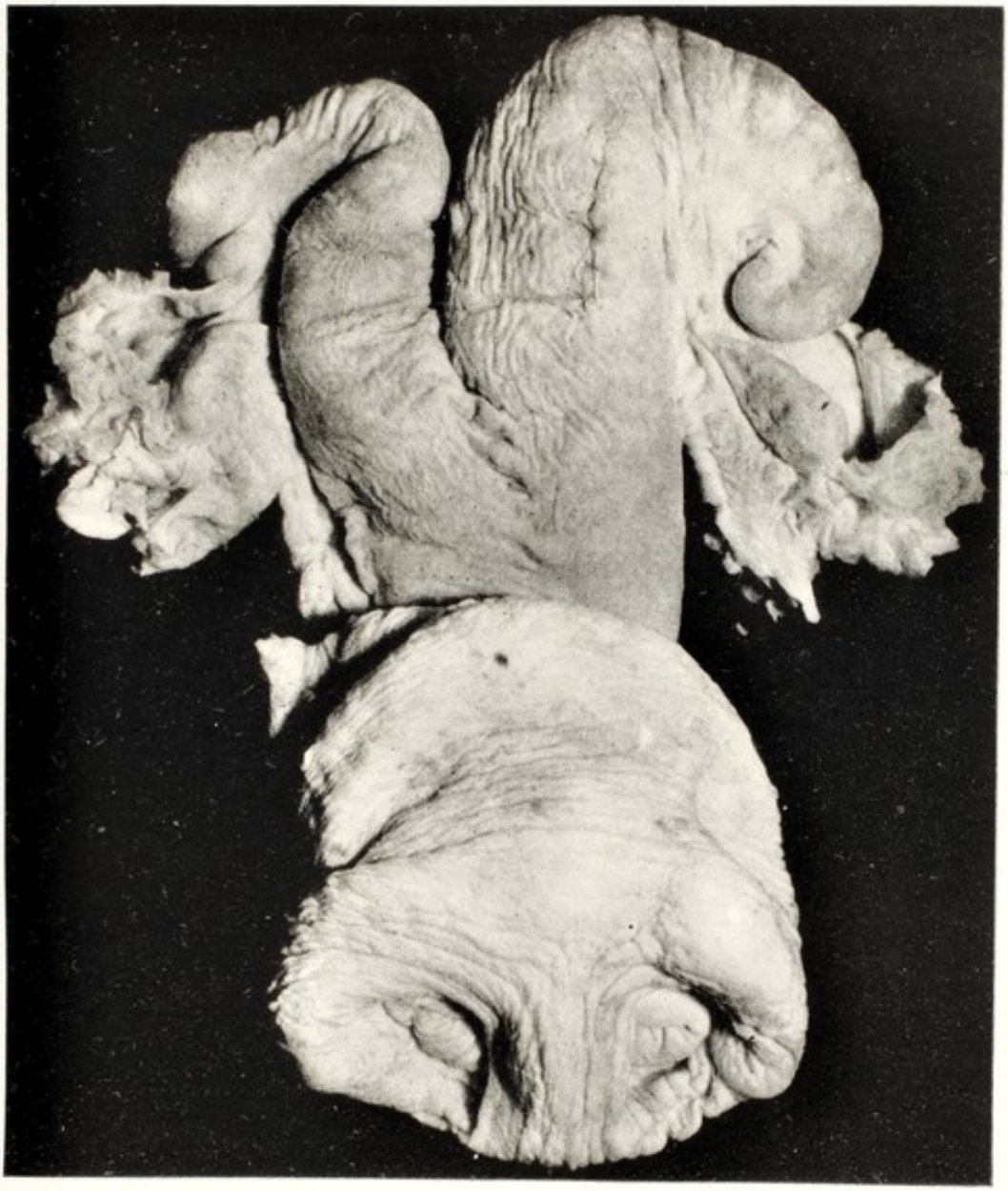


FIG. 10. Bovine Double Uterus from Heifer Which Aborted at Six Months. *Unopened. The same conditions were present as those shown in Fig. 9. (Cornell Veterinarian)*

and was not disturbed by the douching on the day following breeding. Fincher,[54] attending a 2-year-old heifer for retained afterbirth following abortion, found a double uterus as shown in Fig. 10. Member of a Bang-negative herd, the heifer reacted to agglutination test at

1:640, and was slaughtered. Four years later Fincher[56] attended, because of dystocia, a second heifer with double uterus and uterine torsion, which he overcame by cesarean section. Ignoring Fincher's advice, she was rebred, and Fincher was again called because of dystocia, which was relieved by dilating the cervix by incision. The cow died.

More frequently there is observed in dairy cows a less extensive persistence of the median walls of the Muellerian ducts at the vaginal end of the cervix, forming a perpendicular fleshy column, as indicated in Fig. 11. It is not authentically known how fertile such



FIG. 11. Double os Uteri Externum—Persistence of the Median Walls of the Muellerian Ducts at the External Opening of the Cervix, Anterior to Which There Is a Single Cervical Canal.
(Keller; in Halban und Seitz, Biologie u. Pathologie des Weibes)

cows may be. Neither the uterus unicornis, the double cervix nor the perpendicular fleshy column directly prevents conception or parturition. The cases of double uterus observed by Fincher ended in disaster. The instances of uterus unicornis observed have not been definitely sterile. I have observed the perpendicular fleshy column at the vaginal extremity of the cervix in sterile heifers, and have divided it, but the result was not learned. The animals are largely fertile. The column is sometimes so great that it constricts the cervix of primipara and must be divided in order to permit the expulsion or extraction of the fetus. If the fetus is expelled without

interference, a portion of the afterbirth may emerge upon either side of the column and hang fast.

Cows having such defects, if they breed and milk satisfactorily, may be retained for the production of milk, but their progeny of both sexes should be discarded. Such defects constitute a definite badge of inferiority with a marked tendency to become more and more exaggerated, generation after generation.

Persistent Hymen

Persistent hymen may occur in any species. It is most common in ruminants. It is sometimes imperforate, barring coitus and imprisoning utero-vaginal secretions, leading to the distension of these cavities. In the heifer and ewe, I have observed pyogenic infection of the contents so that, instead of mucus or estrual debris, large collections of pus filled the cavities. It is readily overcome by surgery in so far as the physical life and health of the individual goes. When the contents become purulent, it is scarcely possible to expect fertility. The use of such animals, when fertile, is unjustifiable because of the danger of hereditary transmission.

Abnormal Wolffian (Gartner's) Ducts

Teratology and pathology of the Wolffian, or Gartner's ducts occur not rarely in the vagina of the cow. Normally these ducts should disappear entirely in the female except for very faint traces without canalization. Occasionally there is present in the vulva, on either side of the meatus urinarius, a cul-de-sac, ending blindly forwards, sufficiently large in exceptional cases to admit three or four fingers for a distance of two or three inches, representing an abnormally large, open terminus of Gartner's duct. In one case the sac was so extensive that it prevented coitus until it had been divided surgically. A small sac of this type is seen in Fig. 101.

More commonly the teratological defect is in the floor of the vagina, where the remnants of the Wolffian ducts have become canalized, as shown at 5 in Fig. 5. If the meatus becomes occluded, the duct becomes distended with lymph, to form a tortuous, fluctuant cord, 1 or 2 cm. in diameter. Occasionally the contents become infected and purulent. In one instance I found the duct greatly distended with innumerable calculi, some of which were 1 cm. in diameter. Carcinoma developed at the site of my incision. Usually the ducts may be opened freely and the contents allowed to escape. The question of heredity should be considered.

The Prevention and Handling of Monsters

The constant increase in the incidence of monsters and lesser types of hereditary defects demands far greater consideration than has yet been accorded. Inbreeding and line-breeding have unquestionably done much to advance the quality of domestic animals; upon the other hand, serious losses have frequently followed the practice. The path of safety in the mating of consanguineous individuals is not yet authentically known. Breeders rivet their attention entirely too closely upon the immediate ability of the animals to produce meat or milk, or to win prizes or races, and overlook defects which progressively lower the vigor, longevity and fertility of the breed or family.

Some congenital defectives may be rendered useful for labor, meat or other valuable purposes by surgical interference. The not extremely rare hermaphroditic or mixed-sex equine monster, with abdominal or inguinal testicles, near-normal uterus, vagina and vulva, no scrotum, no penial sheath, an erectile organ offering a compromise between penis and clitoris, and the behavior of a cryptorchid stallion, may be castrated per vaginam, inguinally or through the flank and converted into a docile work horse. The penis-clitoris may be modified surgically so that the urine will escape within the vulva as in the mare, and the horizontal backward stream of urine eliminated.

Numerous supposedly unimportant hereditary defects are ignored, and, yet worse, are often disguised in order to render the animals eligible for show or sale. Umbilical hernia may be surgically disguised. Not long since, two prominent staff members of a veterinary school, maintained by the state for the purpose of guarding the health and efficiency of its domestic animals, were advocating a surgical technic for *curing* scrotal hernia without removing the testicles, thereby enabling the breeder to sell his faulty animal, to beget more scrotal herniae, and perhaps supply more opportunity to veterinary practitioners to operate upon the defective animals. Quite recently two staff members of an agricultural experiment station, maintained by the state for the advancement of the quality of livestock, having found that *kinky tail* had become hereditarily established in the station herd, devised and recommended a technic by which the unsightly defect could be surgically disguised so that the animals would not be excluded from the show ring and might be sold at high prices, thus perpetuating kinky tails. A favorite disguise, which is so frequently practiced that it is almost regarded as a virtue, is the amputa-

tion of the supernumerary teats from the mammae of dairy cows. Breeders know, or think, that a cow will more probably win a prize or sell at a higher figure if she shows four, instead of five or six teats. The fraud can probably be detected in most cases by careful search for the scar.

This type of surgery raises interesting questions in the field of professional ethics. The veterinarian may find himself in a position where a refusal to operate involves insubordination to a superior officer. I was asked to remove an umbilical hernia from a fashionably bred bull, because the hernial sac was so large that it barred coitus by deflecting the penis downward. It was desired to use the bull in a state agricultural college herd, for teaching agricultural students the basic principles of genetics and animal husbandry. I demurred, but the animal husbandman was virtually my superior officer and he commanded me to proceed. The operation proved a failure, and I regarded it as one of my most brilliant failures. Fortunately the artificial insemination comet had not yet flared in the veterinary firmament and I was not compelled to attempt that. When an influential client demands that the veterinarian remove surgically the evidence of an hereditary defect, the latter's position is not enviable.

Monsters and lesser defects are too largely regarded as isolated, meaningless curiosities, when a careful study would show that they have a definite background of heredity. The economic losses are not confined to the monstrous or defective individuals. Teratological defects constitute a definite badge of inferiority, not in the aberrant individual alone, but also in its relatives. Mention has been made of the studies of McPhee, Russell and Zeller,[132] showing that the continued inbreeding of swine not only increases the incidence of monsters, but more significantly decreases the number of pigs in the litter, and decreases the vigor, rapidity of growth, and fertility of the progeny. The litter-mates of monsters in multipara and the siblings of unipara are of less value because of their consanguinity with monsters.

Some minor abnormalities may possibly be diluted or bred out by intelligent mating. If the teats of a dairy cow project almost horizontally from the udder, when she walks, the leg strikes the teat and the udder is forcibly revolved to and fro upon its perpendicular axis until the gland becomes pendulous, causing inconvenience in milking and predisposing the animal to mastitis. The defect may be diluted by the prudent selection of sires, or concentrated by mating her with one of her bull calves.

Breeding out abnormalities is a tedious, and usually unfortunate

adventure. A client mated two high-class Percherons, and the mare dropped a filly too monstrously compact to have any value. When the latter was mated with a normal sire, the defect was definitely diluted, but the offspring, a filly, was still too compact to be of value. She also was mated with a normal sire, and further dilution ensued, but this foal of the third generation was undesirably compact. The abnormality was being bred out, but it was costing far more than the animals were worth.

A given teratological defect of an individual may fail to reappear in its progeny and be supplanted by aberrations of a wholly different type. Sometimes the inference may be drawn that the defect of the parent has destroyed its reproductive equilibrium and opened the way for the occurrence of unpredictable abnormalities. I found myself in possession of a highly bred trotting mare of good conformation and in apparently excellent health, after handling for an extensive odontome, a common teratological phenomenon in horses. She was mated with a good sire and the resultant foal, otherwise apparently normal, had such crooked legs that he was worthless. Her next foal by the same sire was so high at the withers as to suggest hybridization with a giraffe, and was so far from the usual conformation as to virtually destroy his value. The third and last foal did better by dying very young.

The breeder should learn, as early and emphatically as possible, that the most direct and safest path to the improvement of his animals lies in the mating of individuals which are anatomo-physiologically perfect in every visible detail, and free from perilous consanguinity, as well as from the opposite extreme of hybridization between distantly related species or breeds.

Chapter III

TERATOLOGICAL AND PATHOLOGICAL OVULATION AND SPERMATOGENESIS

THE preceding pages have been devoted to the consideration of teratological defects, largely following hybridization and inbreeding. It is not known that they are dependent upon any definite morphological defects in the sex cells.

It is here desired to discuss diseases or disturbances of the functions of the ovaries and testicles, which may lead to the production and discharge of immature, pathological or teratological sex cells. The field is open to much conflict of opinion. It is generally admitted that, after a spermatozoon and an ovum have united to constitute fertilization, the life and health of the resultant individual are menaced by disease, to the end of its existence. It is not so generally admitted that the immaturity or disease of a spermatozoon or ovum may, after their union, constitute a menace to the health or life of the new individual. It seems to be the general, although unexpressed, belief that fertilization can only occur when a perfect male cell unites with a perfect ovum.

This hypothesis, whether expressed or implied, leads directly to one of the most important errors occurring in animal husbandry. Until quite recently, if the semen of a male showed numerous motile spermatozoa, he was regarded as safely fertile. That hypothesis still largely prevails, despite the fact that a few workers in this field[114,245,250,266] have indisputably shown it to be erroneous and dangerous. The safety of any pregnancies attained, the reproductive life of the female involved and the value of any progeny born are regimented in harmony with the ratio of morphologically defective spermatozoa. If a male sold as a sire produces visible pregnancy in fifty per cent of his healthy females, he is technically fertile, although most of his pregnancies end in disaster. The sire having motile spermatozoa which are largely defective morphologically is a far greater menace to his females than if wholly unfertile. If a female becomes recognizably pregnant after five or six matings with a healthy sire, she is commonly regarded as fertile, even though she abort, or her pregnancy terminate in some other type of disaster.

The evidence regarding the significance of the morphology of the sex cells is most abundant and authentic for the male. Vast numbers

of spermatozoa are freely available for study, and the findings can be authentically checked by the results of his activity as a sire. Ova are not available for such study, but there is being accumulated an extensive and highly significant mass of clinical observations, which justify definite conclusions upon which the breeder may act with confidence. The conclusion is now inescapable that diseased, unstable ovaries release ova faulty in numbers and immature or otherwise defective, which tend to end in disaster after fertilization. Yet more abundantly it has been shown that diseased or overloaded testicles discharge immature and morphologically defective spermatozoa, any pregnancies from which largely terminate pathologically.

Each mammalian genus or species has a well established law of ovulation in harmony with the plan of the uterus and mammae. Disease or disturbance of the ovaries upsets the law of ovulation and causes the release of unripe, or defective ova, or an excessive or subnormal number. The ovulation in multipara may be unbalanced in the two glands, an excessive number being released from one, and a subnormal number from the other. Such pathological ovulation may lead to abnormal location of the fertilized ovum, defective placentation, intercornual migration, abortion, dystocia, and, in case of birth, an excessive number of young in conflict over nursing.

Domestic mammals include the definitely uniparous mare and cow, the equally characteristic multiparous swine and carnivora, and between these the goat and some breeds of sheep which tend conspicuously to produce twins.

From the Muellerian ducts of the early embryo are derived the vagina, cervix, uterine body, cornua, and oviducts. The two ducts fuse for varying distances, according to genus, from the caudal toward the cranial end. The fusion is normally complete, in all species, in the vaginal and cervical zones. Variations in fusion occur in the utero-cornual area, the degree of fusion corresponding with the uniparity or multiparity of the species. While all portions of the Muellerian ducts are essential to reproduction, the utero-cornual zone, in which the fertilized ovum resides, and is nourished up to the time of birth, is most important. Among domestic animals, so far as is known, placentation and the nourishment of the intra-uterine young are limited to that area extending from the os uteri internum of the cervix to the point at which the duct of Mueller is crossed by the round or inguinal ligament. Outside this zone it is not definitely known that placentation occurs. In contrast it is well known that in woman placentation occurs pathologically in the oviducts (tubal pregnancy). The Muellerian ducts of woman and other primates fuse

completely throughout the utero-cornual area, their median walls are resorbed, and a common cavity, the body of the uterus, results. Hence in primates the inguinal ligaments cross the Muellerian ducts immediately against the fundus of the uterus, as shown in Fig. 12.

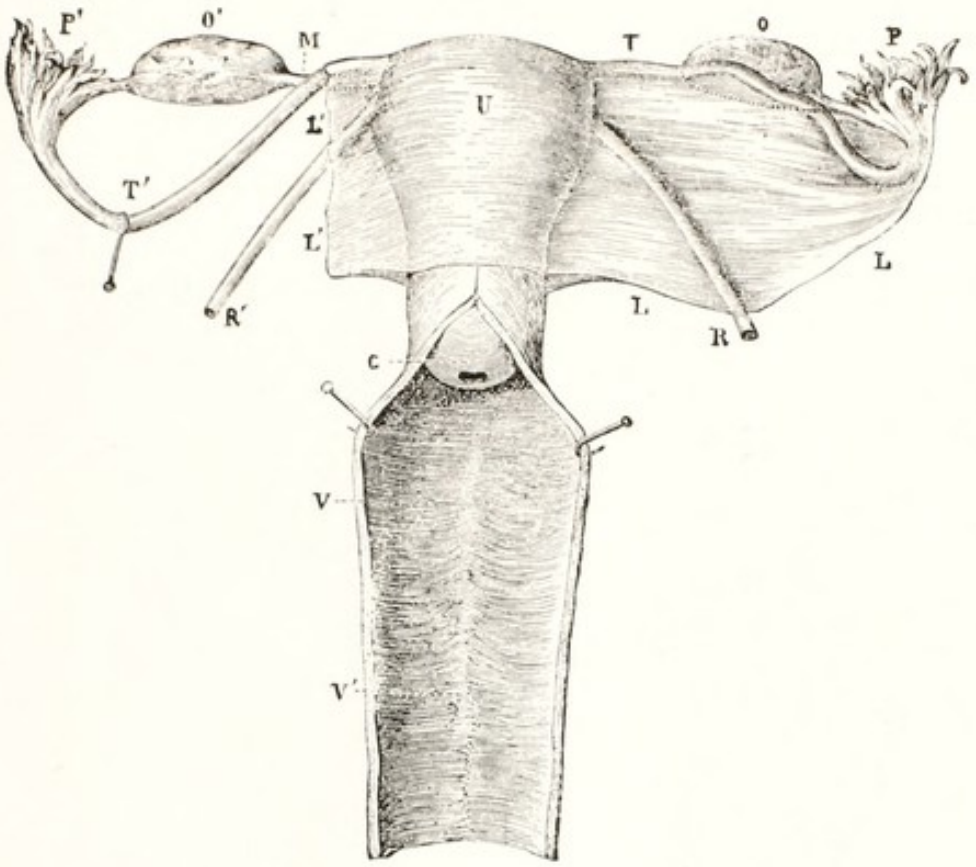


FIG. 12. Internal Generative Organs of Woman, Viewed from the Anterior (Ventral) Side.

L, broad ligament of left side; *L'*, broad ligament of right side, partly removed; *U*, uterus; *O*, *O'*, ovaries; *P*, *P'*, pavilion of oviducts; *T*, *T'*, oviduct; *R*, *R'*, round or inguinal ligaments; *C*, cervix; *V*, vagina; *V'*, posterior (dorsal) column of vagina. (Barnes after Tarnier)

The Muellerian ducts of the mare, Fig. 13, fuse completely to a point about midway between the cranial end of the cervix and the crossing of the round ligament, where the similarly capacious cornua leave abruptly at right angles, to form a cruciform uterus, the entire interior of which has functional endometrium with diffuse placentation. The fused area of the ducts is designated *body of the uterus*, but does not represent the corpus uteri of primates. The body of the uterus of woman equals, embryologically and functionally, both body and cornua of the mare.

In ruminants the Muellerian ducts fuse completely and their median walls are resorbed at their cervical ends to a point anterior

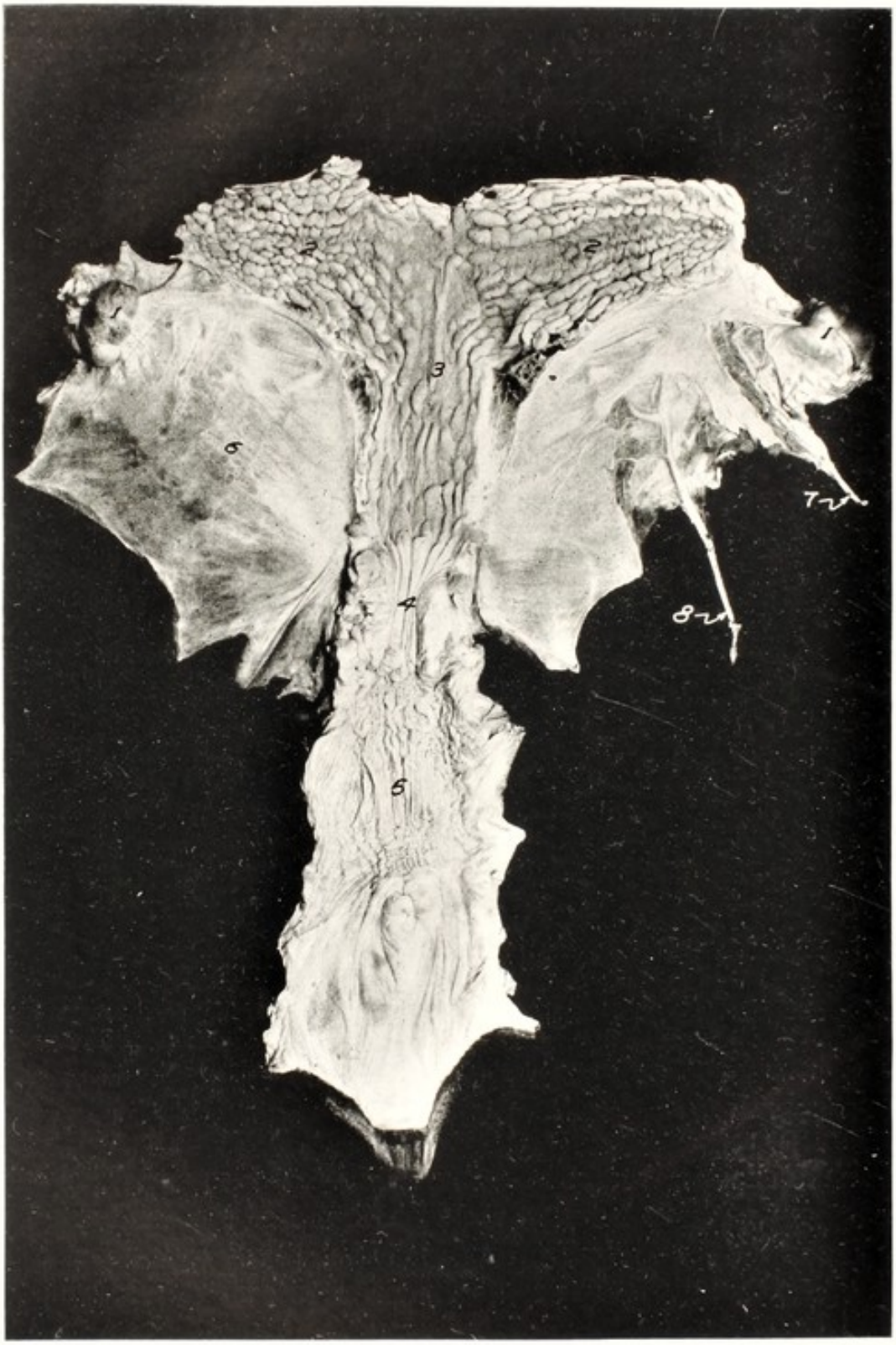


FIG. 13. Internal Generative Organs of Mare, Viewed Dorsally.

The upper or dorsal wall of the uterine body, 3, has been removed. The uterine cornua have been incised along the dorsal side of the attachments of the broad ligaments, and the dorsal wall of the cornua spread out forward.

1, 1, Ovaries; 2, 2, uterine cornua; 3, uterine body; 4, cervix; 5, vagina; 6, left broad ligament; 7, ovarian artery; 8, uterine artery.

to the os uteri internum of the cervix for a distance equal to the transverse diameter of the cornual cavity at its base. The uterus thereby becomes a uterus didelphys, with a single cervical canal. Functionally no true uterine body exists: the fusion between the bases of the uterine cornua, or twin uteri, serves merely to provide communication between the two organs and to permit the fetus at birth to enter the common cervix from either tubular organ. Cranial to this so-called uterine body, the two cornua are externally fused, the peritoneum resorbed, and the two slightly divergent organs firmly bound together by the inter-cornual ligament. The two uteri accordingly lie side by side almost parallel, as shown in Fig. 14.



FIG. 14. Adult Bovine Uterus and Ovaries.

1, Os uteri externum 2, right uterine cornu; 3, ovary; 4, ovarian ventricle; 5, ostium abdominale of the Fallopian tube, 6.

When the inter-cornual ligament is divided and the two tubes, opened along the convex, dorsal line, are spread out at right angles to the vagino-cervical axis, the hypothetical uterine body completely vanishes, as indicated in Fig. 15. Or, if the cervix and cornua are sec-



FIG. 15. The Uterine Caruncles of Veal Heifers.

The heifer calves are estimated at five to ten weeks old. The inter-cornual ligaments and muscles have been divided, separating the two cornua completely, after which they are spread out at right angles to the long axis of the cervix and vagina. The cornua have been opened along their greater curvature, opposite the attachment of the broad ligaments (compare Fig. 14). The two dorsal rows of caruncles are marginal, the two ventral rows are central.

1, Vagina; 2, cervix; 3, right cornu; 4, ovary.

tioned horizontally as shown in Fig. 187, it is seen that the canal of one cornu communicates with the other by a U-shaped bend so that the two cornua have one cavity from ovarian pole to pole.

The uteri of multipara differ from those of unipara. There are two, long, independent cornua which leave the technical body immediately at the anterior opening of the cervix. There is no inter-cornual liga-

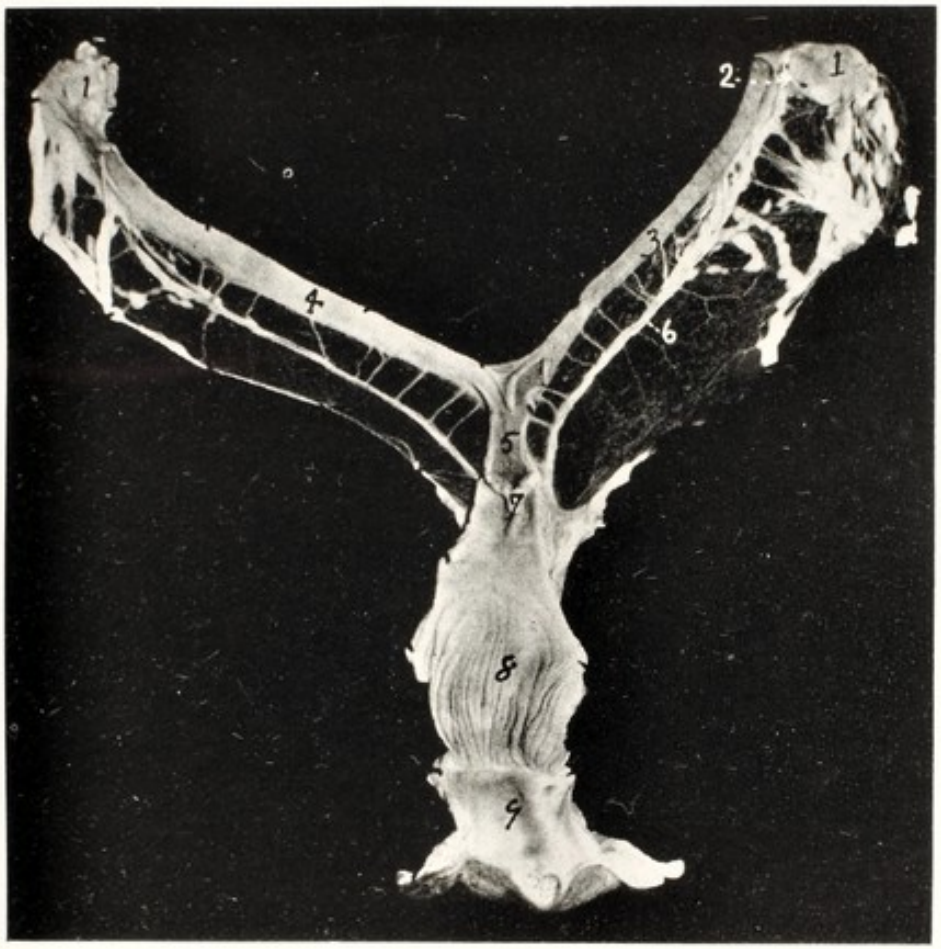


FIG. 16. Non-gravid Uterus of Bitch, Viewed Dorsally.

1, 1, Ovaries hidden within the ovarian ventricles; 2, the fenestrum or slit of communication between the ovarian ventricle and peritoneal cavity; 3, right cornu unopened; 4, left cornu laid open; 5, "uterine body"; 6, uterine artery in the broad ligament; 7, cervix; 8, vagina; 9, vulva.

ment. Normally no part of a fetus occupies the body of the uterus, but the allantochorion of the basal fetus of one horn crosses over and occupies the base of the opposite cornu for a variable distance. This tends to obliterate the sharpness of the angle of departure of the cornua from each other and causes them to leave the vagino-cervical axis almost perpendicularly, much like the cornua of the equine uterus, Fig. 13. The morphology of the multiparous uterus facilitates migration of the ova from one cornu to the other, and when

unequal ovulation occurs permits the balancing of the load between the two sides. Data are not available to indicate how frequently this may occur in carnivora, though its possibility may be seen from a study of Figs. 16, 17.

While excessive ovulation occurs as a pathological phenomenon in

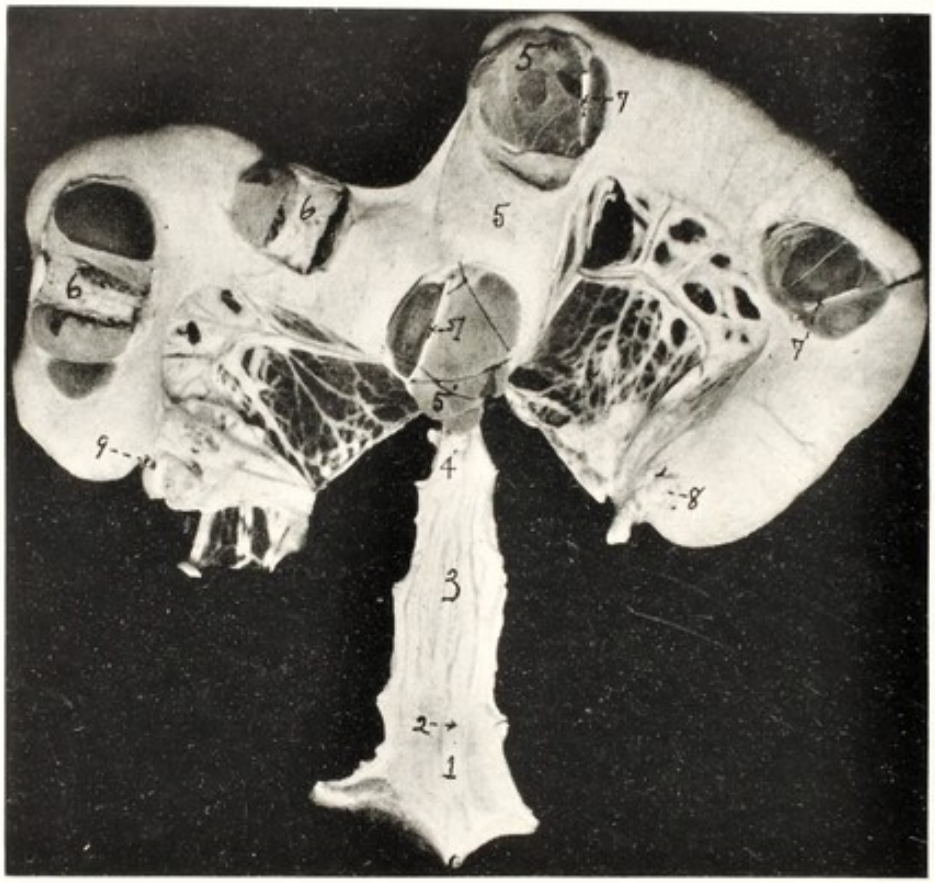


FIG. 17. Pregnant Canine Uterus Viewed Dorsally.

1, Vulva; 2, urinary meatus; 3, vagina; 4, cervix; 5, 5′, 5″, first fetal sac of right uterus completely filling the so-called "uterine body" and projecting at 5′ into the cervix and thus possesses right-of-way at the commencement of labor; 6, 6, the placental bands of the two fetuses in left uterus. The sac of the proximal fetus abuts against the sac of 5 at 7; 7, 7, 7, points of contact between fetal sacs—strips of white paper have been inserted between the abutting sacs in order to mark the division; 8, the right ovary hidden in its ventricle; 9, the left ovary exposed by drawing back the ventricle.

unipara, deficient ovulation apparently occurs in multipara. This has not been well studied. Solitary ovulation cannot be securely diagnosed upon the number of fetuses found in the uterus, because an indefinite number of unfertilizable or of non-viable ova may disappear unseen. The most important evidence of solitary ovulation in multipara is found in the occurrence of bicornual pregnancy in the bitch, calling for cesarean section for delivery. Cuille[37] has reported two cases in which he performed cesarean section. Colleagues en-

gaged in small animal practice advise me that such bicornual pregnancies are usual in cases of a solitary fetus. Apparently it occurs mostly in breeds of low reproductive power, in which the size of the litter is small, and solitary ovulation may be suspected as the foundation.

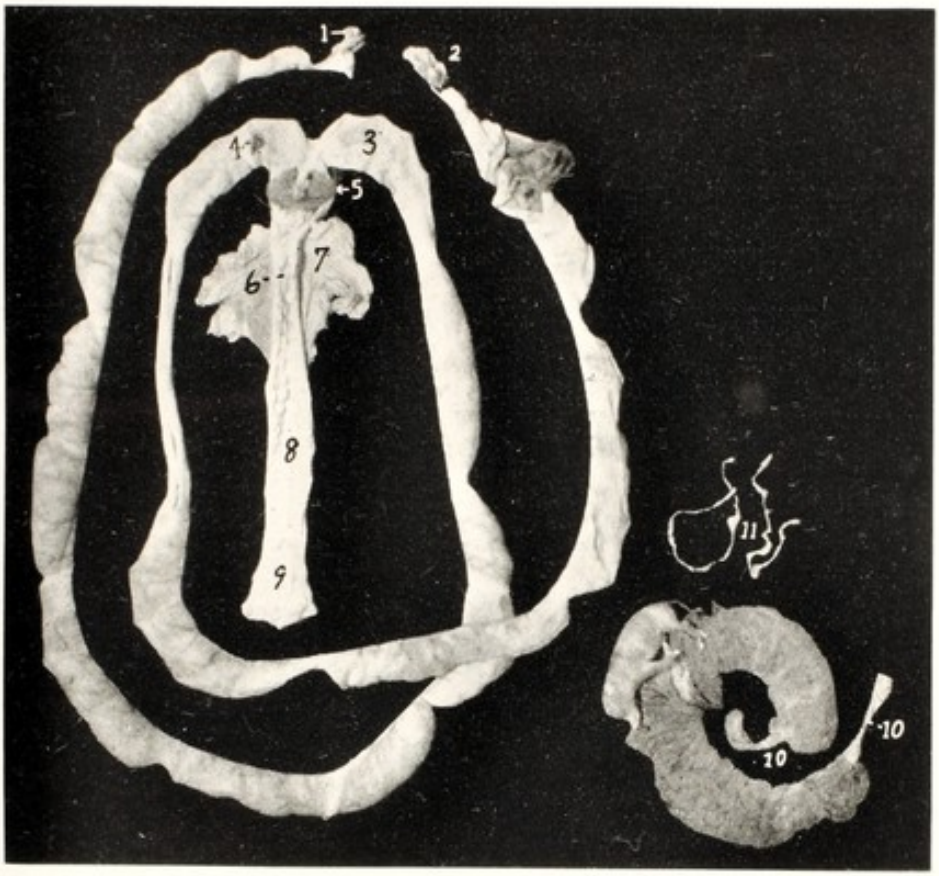


FIG. 18. Gravid Swine Uterus containing 13 fetuses about 2.5 cm long, or about ¼ term. The cornua, with a combined length of twelve feet, when held by the mesometrium, 7, are thrown into convolutions like those of the small intestines. In order to expose the length of the cornua, the broad ligament, 7, has been cut away from the cornua, and the latter arranged in coils about the birth canal (cervix, vagina and vulva).

1, Left ovary with eleven corpora lutea; 2, right ovary with two corpora lutea; 3, right cornua with eight fetuses; 4, the left cornu with five fetuses; 5, the sac of the proximal fetus of the left horn projecting across the median line into the base of the right cornu, assuring it the right-of-way at the onset of labor; 6, cervix; 7, broad ligament; 8, vagina; 9, vulva; 10, 10, necrotic tips of fetal sac near term; 11, embryonic cadavers from same uterus as 10.

The Muellerian ducts of swine, Fig. 18, fuse for but a brief distance cranially to the cervix and the two cornua diverge abruptly as independent organs. The endometrium is functional throughout the uterine cornua in swine and carnivora. The placenta of swine is diffuse and adeciduate; that of carnivora deciduate and zonular.

The fertilized ova of swine habitually become located at about

equidistant points in that cornu corresponding to the ovary from which they originated. Pathologically the two ovaries may ovulate unequally, in which case, as has been well shown by Corner,[33] an effort is commonly made to balance the load by internal migration. Many fertilized swine ova die. According to limited observations, it appears that the ova migrate very early and that there is present within the ovum itself an intrinsic power of migration. In one sow there were two corpora lutea in the right ovary and four normal fetuses in its cornu; there were six corpora lutea in the left ovary, and two dead and two live fetuses in its cornu. The numbers of ova in the two cornua had been balanced, but the two non-viable ova had remained on the side of origin, while two viable ova had migrated to the opposite horn. One of the normal and the two dead fetuses from the left uterus are shown in an insert in Fig. 18. Another sow uterus with fetuses varying between 15 and 20 cm. had ten corpora lutea in the right and one in the left ovary. The left cornu had six fetuses and the right two. Three ova were missing. Lying beyond both the cranial and caudal ends of the fetus located at the ovarian pole of the right cornu, were masses of necrotic debris, apparently representing the missing ova. These observations are too limited, but permit the inference that the migration occurs early and that non-viable ova do not migrate as freely as those which are healthy.

It appears to be the general opinion that the fertilized ovum is passive and that its movements prior to the completion of birth are wholly dictated by the oviduct and uterus. On the contrary, the above stated view would attribute to the ovum highly significant intrinsic powers which would fundamentally affect its destiny: it would share with the oviduct the responsiblity for the safe passage of the ovum to the uterus; would ascribe to the ovum, in coordination with the uterus, the determination of the physiological location of the embryo during gestation; and at the close of pregnancy the fetus would cooperate with the uterus, in the control of the presentation and position. Such view does not relegate fetal dystocia to mere chance, nor definitely to uterine inefficiency, but regards the fertilized ovum as a new individual, clothed from the beginning with important intrinsic responsibilities and powers. If the ovum is pathological, its destiny is clouded.

It has been previously noted that tubal pregnancy is not rare in woman. As a rule the tubal embryo is teratological, the monstrosity being apparently attributed to its pathological location. It largely follows a history of gonorrheal salpingitis. The salpingitis affords an

explanation for the lodgment of the fertilized ovum in the tube. It does not, however, exclude the possibility that the gonorrhea had affected the function of the ovary and caused the release of a pathological ovum. Such disease of the ovum might favor its lodgment in the tube and its teratological development. At least in domestic animals the conclusion seems wholly justified that the pathology of the ovum plays a highly important role in the welfare of the intra-uterine young, and, if it survive to be born, influences the health of the post-natal animal throughout its life.

The fertilized ova of unipara physiologically become located near the base of the cornu on the side from which the ovum emanates. The equine blastoderm is at first spheroidal. It elongates as gestation advances and the chorion, and later the fetus, protrudes into the uterine body and advances to the internal os uteri. That portion of the allantochorion without fetal parts occupies the non-gravid cornu.

Owing to the cruciform uterus, Fig. 13, the allantochorion, with the advancing end of the contained fetus, may pathologically pass directly across into the base of the opposite horn, instead of toward the cervix, to constitute bicornual pregnancy first definitely described by me[227] in 1889. When parturition sets in, the abnormality commonly provides a true transverse presentation, a phenomenon not otherwise possible in the tubular uterus, and causes one of the most serious types of dystocia known in equine obstetrics. Hypothetically the fetus may migrate so far into the nongravid horn that the end remaining in the normally gravid cornu may slip out and become directed toward the cervix within the uterine body, thus leading to a reversal of the presentation.

The didelphic uterus of ruminants, the two organs firmly bound together and lying parallel in immediate contact, offers poor opportunity for the migration of ovum or embryo, although the possibility of such migration cannot be authentically denied. Sometimes the corpus luteum in the cow is located in the ovary on the opposite side to the cornu in which the fetus lies. This does not prove migration. The cow, especially the genitally unsound dairy cow, sometimes comes in estrum during pregnancy owing to atrophy of the corpus luteum of pregnancy, causing the ripening of an ovisac in the opposite ovary. So I have definitely recognized by rectal palpation, in a cow in heat, pregnancy in one horn and a ripe follicle in the opposite ovary. Such bicornual pregnancy as mentioned above, in the mare, is not frequent in ruminants. I have not observed a case. Krill[110] reports two well defined cases in the cow. The cephalic end

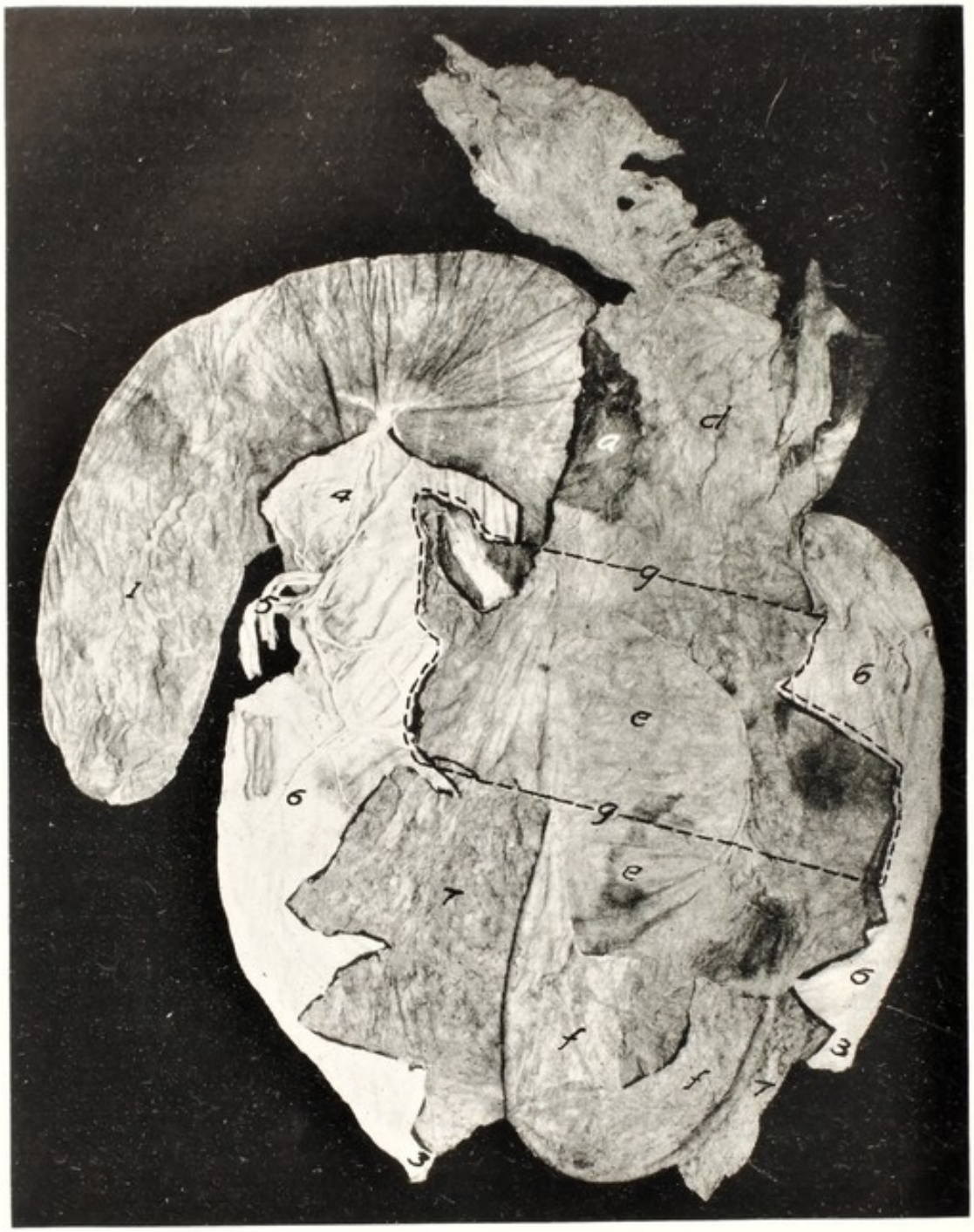


FIG. 19. The Placentation of Equine Twins.

The membranes are those from the twins shown in Fig. 184. For the normal allantochorion compare Fig. 161.

1, the cornual allantois of the larger twin, the outer, or placental side showing; 3, 3, the parturient rent through which the larger fetus was expelled—the distal end of the sac of the lesser fetus, *f*, *f*, is not ruptured. Instead the lesser fetus dropped out through the rent at *a*, and remained in the uterus until manually removed; 4, the base of the invaginated sac of the cornu of the larger fetus incised to expose the amniotic side; 5, the umbilical cord of the larger fetus; 6, 6, 6, the amnotic side of the body chorion of larger fetus; 7, 7, placental side of that portion of the sac of the larger fetus pushed before the sac of the lesser fetus, *e*, *e*, *f*, *f*, from the position of *d*, to the vicinity of the cervix; *e*, *e*, the amniotic side of the lesser sac; *f*, *f*, the placental side. *Thus from d, to the rounded end beyond f, f, the placental sides of the two sacs, 7, 7, and f, f, are devoid of placental function, the two sacs being in contact with each other instead of with the endometrium. The larger, invaginated fetus had access to the endometrium of one cornu and of the body, while the lesser fetus had access to the endometrium of one cornu only. There was feeble adhesion (inflammatory?) in the area surrounded by the dotted lines, g, g.*

of the fetus lay in one horn, the caudal in the other. The presentation was U-shaped, with the mid-dorsal area of the fetus presenting opposite the os uteri internum. The fetus must be sharply bent, as may be well understood by studying Fig. 187. Krill performed embryotomy in one of his cases, and the cow survived: the other cow was beyond help and was destroyed. Recently Professor Fincher attended a case which he was able to deliver by pushing the entire fetal body into one horn and converting it into a longitudinal presentation.

The fetal sac of ruminants, elongating early and very rapidly, promptly occupies both uterine cornua from one ovarian pole to the other. Hence in uniparous ruminants and in solipeds the fetus promptly establishes contact with the entire endometrium. In multipara the placental membranes of each embryo are physiologically restricted to a well defined zone, in which the occupant is entitled to the full use of nutritive supplies. When these laws of placentation are violated through abnormal ovulation or otherwise, important results may follow.

Twin or Multiple Ovulation

EQUINE TWINS

Twin ovulation is the most infrequent and most uniformly disastrous in the mare of any mammal known. One ovum usually emanates from each ovary and becomes located near the base of the corresponding cornu. The two allantochorions pass out into the so-called uterine body, and their rounded, blunt ends inevitably collide. It seems that one of the chorions usually precedes the other into the uterine cavity. Unlike the twin ruminant chorions, those of the mare do not generally fuse. Instead, as shown by Errington and Williams in 1933,[51] one of the sacs invaginates into the other, as indicated in Fig. 19. The invaginated sac has placental contact with the endometrium of its cornu and with the body, while the invaginating sac lies within the former and has placental relations with the endometrium of the one cornu only. Through the kindness of Professor Fincher I have recently been able to study the afterbirths of a second set of equine twins, illustrated in Fig. 19*a*. The mare was of low fertility and required numerous services before pregnancy was recognized. She foaled unseen at 342 days and when discovered, both foals (colts) were alive. The large foal was unable to stand and soon died. The afterbirth of the dwarf foal was removed manually. The figure with its legend serves to make clear the relations existing between the two allantochorions. The naked portion of the invagi-



FIG. 19*a*. Allantochorions from full term, living equine twins.

A. The invaginating allantochorion of the viable dwarf; *B*, the invaginated allantochorion of the non-viable twin: 1, the placental area of the dwarf; 2, the cornual placental area of the larger fetus; 3, the body placental area of the larger fetus; 4, a portion of the non-placental (invaginating) area of the chorion of the dwarf; 5, the border line between the placental and non-placental areas, indicating the extent to which it had invaginated *B*; 6, the everted non-placental portion of the chorion which had been invaginated into the body chorion, 3; 7, the parturient rent; 8, the navel of larger fetus, having the normal two arteries and two veins, and slightly twisted to the right (clockwise); 9, the navel of the dwarf, with *one artery and one vein*.

nated sac, 6, has been everted. Prior to birth it had been invaginated into the chorion of the uterine body, 3, and there had rested within it the cervical pole of the dwarf chorion, part of which is shown at 4. Unfortunately the latter was mutilated and portions are missing. It originally equalled the size of the invaginated sac, 6, and completely filled it. The borderline between the placental, and non-placental allantochorion of the dwarf is indicated by the dotted line, 5.

Measurements of the placental surfaces of the collapsed membranes yielded 1674 square inches for the invaginated allantochorion, and 576 square inches for the dwarf, or a ratio of 3:1. This conflict in placentation inevitably leads to the dwarfing of the invaginating fetus, the most spectacular dwarfism observed in domestic animals. The abnormal placentation apparently plays the leading part in the enormous intra-uterine and early postnatal mortality of equine twins—the highest twin mortality known—and seriously imperils the reproductive and physical life of the mare. I have personally known of but one pair of viable soliped twins, mules of opposite sexes. Apparently they had made a workable adjustment in their placentation. I have known five pairs of twins in which one has been born viable, in each case the worthless dwarf.

Effective chorionic fusion of equine twins possibly occurs, but is extremely rare. Keller[104] records equine twins at the seventh month, in which he detected limited vascular anastomosis, but the anastomosis was evidently very defective; the weight of one of the twins was only fifty-five per cent that of the other.

Richter[167] reports that in the Graditz stud, out of a total of fifty-nine twin pregnancies, fifty-one terminated in abortion of non-viable foals, seven gave one viable foal each, and in one case both twins lived. These were typical for all the extensive data of Richter. I made observations[253] in a small stud of thoroughbred mares, which for some years had been largely mated with stallions once famous for the racing ability of their get and had later become infamous for their infertility and infectiousness. After handling for genital infections, they were mated with highly fertile young stallions, and in the first breeding season, among twenty-six mares which conceived, there were six twin pregnancies. Eleven of the fetuses were aborted or born non-viable, and one worthless dwarf survived. All six dams had been sterile prior to twinning and most of them were infertile afterwards. In a second series of observations[262] I have reported the occurrence of five twin pregnancies in a small stud, with one viable dwarf foal. Dr. R. N. G.

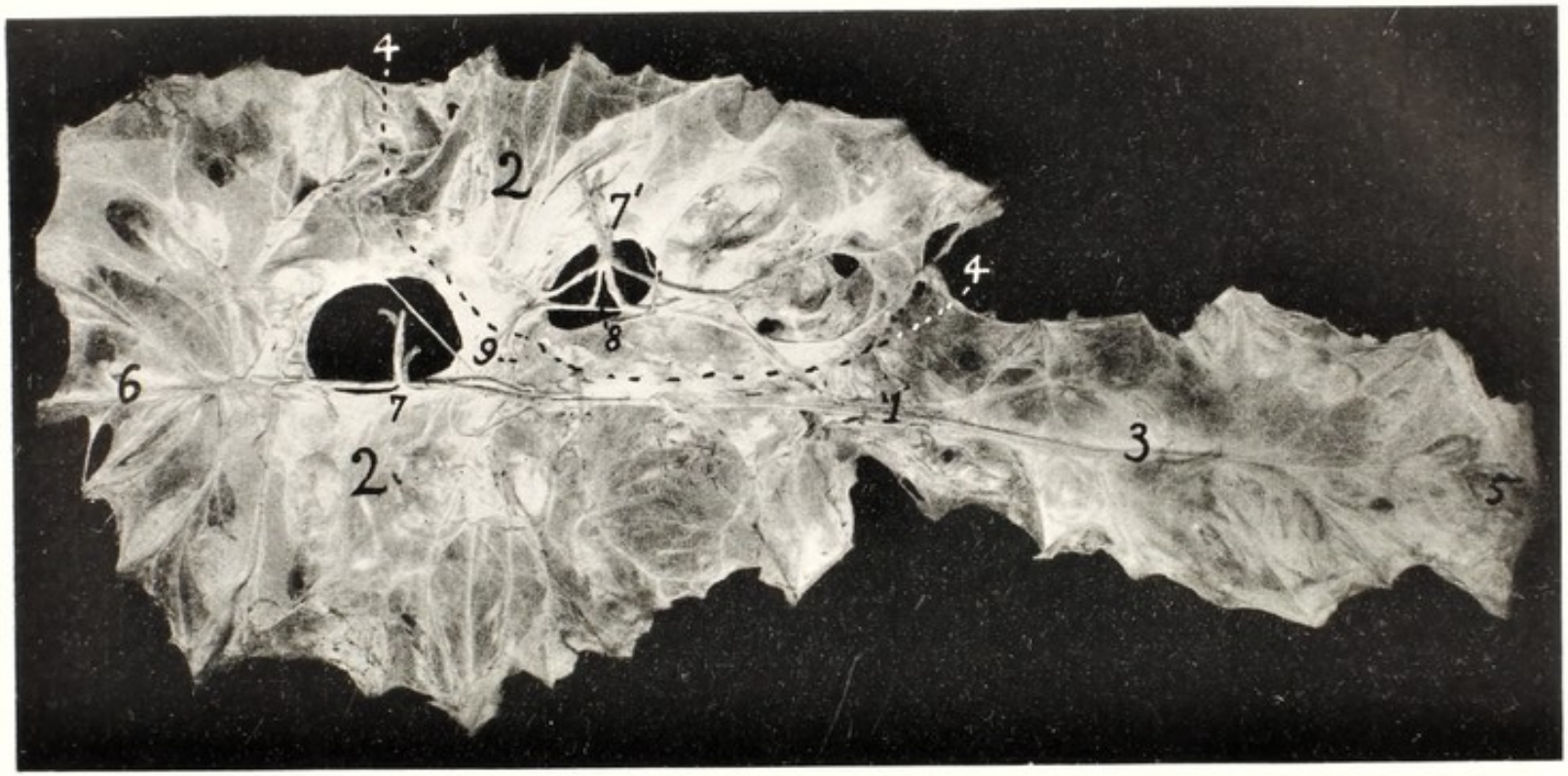


FIG. 20. Fused Chorions of Unicornual Bovine Twins.

1, Cervix; 2, gravid chorion; 3, non-gravid chorion; 4, 4, boundary between two amnions; 5, ovarian pole of non-gravid cornu; 6, ovarian pole of gravid cornu; 7, 7', the two navels; 8, normal inter-arterial anastomosis; 9, inter-chorionic (interfetal) arterial anastomosis. *(Cornell Veterinarian)*

Darby reports that a stallion, which I had condemned as dangerous because a large ratio of his spermatozoa had bent tails, served in 1940 six mares, with an average of five services per mare. All became pregnant. There were three twin pregnancies, all of which were aborted. Two mares carried their foals over 12 months; one was expelled dead, the other was non-viable. There was one viable foal from six pregnancies. Two of the mares were again bred to the sexually diseased stallion, resulting in two twin pregnancies, with one viable dwarf foal. It is not presumed that the stallion could directly cause twin pregnancy or twin ovulation. It rather appears that having repeatedly served the mares, they were pregnant several times each without the pregnancy having become recognizable. Owing to the morphologically defective spermatozoa the fertilized ova were weak and perished early; the decomposing embryos caused metritis or other important injury, disturbing ovulation and causing two or more ova to be simultaneously discharged, which were later fertilized.

The foregoing data are representative. Although exceptions occur in which twinning appears to be a mark of exalted breeding power, and viable twins prove valuable animals, as an overwhelming rule equine twin pregnancies follow genital disease; they almost always result in abortion or non-viable foals; the few viable foals are usually dwarfs; the mortality of mares is high; and most of the survivors are permanently infertile.

BOVINE TWINS

It has already been noted that the ruminant uterus is essentially didelphic with a single cervix. This fits it in a degree for twinning. The chorions of the ruminant twins or higher multiples normally fuse and the walls of the abutting ends disappear, thus constituting a continuous allantoic sac from the ovarian pole of one cornu to that of the other, whether the contained young be one, two or more. The twins are usually bicornual, but may be unicornual. I have reported two cases of unicornual bovine twins [256, 260] in which the amnions, as well as the allantochorions, were fused, as shown in Fig. 20. The twins were binovular, two corpora lutea having been present in one ovary. The fusion of bovine fetal sacs is complete, inclusive of a communal vascular supply. The proximal umbilical artery of the one twin is continuous with that of the other. When triplets are present, both umbilical arteries of the median fetus pass into the two proximal arteries of the mates on either side. The communal allantoic cavity equalizes the tension of the allantoic fluid throughout and the communal vascular supply insures uniform nutrition to all fetuses. Ap-

parently owing to this fact, bovine twins and multiples are far more uniform in size and vigor than are twins in other species of domestic animals or the litter-mates of multipara.

The fusion between twin and multiple sacs of the goat and sheep embryos is less complete. Normally no anastomosis of the vascular supply is macroscopically present, and the individual chorions have well defined boundaries indicated by an opaque band encircling the tube. The fusion is complete, however, for all other tissues, and as in the cow there is a continuous allantoic sac from one ovarian pole to the other, each fetus normally having its distinct amnion. Logically, ovine and caprine twins are somewhat less constant in size than bovines. When triplets or higher multiples occur in the goat or



FIG. 21. Fused Chorions of Ovine Triplets.

1, Chorion containing a 5.8 pound lamb; 2, chorion of 3.3 pound non-viable lamb; 3, chorion of 7.0 pound lamb; 4, 4, fusion lines between chorions without visible vascular anastomosis. *(Cornell Veterinarian)*

sheep, serious conflict arises regarding the nutritive supply and, as in equine twins, those deprived of adequate placental contact may become seriously or fatally dwarfed, as indicated in the triplet ovine chorion shown in Fig. 21: the non-viable lamb in chorion 2 weighed 3.3 pounds and the vigorous lamb of chorion 3 weighed 7 pounds.

When the fetal sacs of bovine twins have completely fused and one fetus dies, so far as known, the death of the other promptly follows. When the two sacs are distinct, in either ruminant or mare, one fetus may die and undergo papyraceous mummification and the other continue to live, or in the cow with viable independent twins they may be born at widely separated intervals. Independent twins are usually bicornual, but may be unicornual.

Twin gestation is better borne by the cow than by the mare, but is far from being desirable. I have elsewhere shown[253] that in the

cow, as in the mare, twin conception is most common in those females having a history of prior genital disease. Clapp[31] has vividly shown that twin ovulation and conception is highly probable following convalescence from nymphomania, if the cow be bred within thirty-five days after the last recognition of the cysts. Clapp observed eleven of these, of which twin or triplet gestation followed in six, while of thirteen held out of breeding for more than thirty-five days only three experienced twin conception. In nymphomania in the cow, multiple cystic follicles are regularly present, and during convalescence the ripening of two or more follicles at a given period persists in lieu of the multiple cysts.

The heifers in Table I were born during a virulent outbreak of puerperal sepsis in the Cornell University dairy herd described by Wing.[271]

The members of the group were the progeny of diseased cows and were themselves in bad health as calves. Early in their first pregnancies they were given abortion bacterins (killed *Bacillus abortus*) then popular as a preventive of abortion. The table shows disastrously low reproduction with a twin ratio of 6:100. None of the twinning cows produced viable young during the preceding year. Only one of the three produced young later, one lone bull after a delay of two years. I have recorded[257] a group of seventeen Holstein-Friesian heifers drawn from a highly fertile herd which averaged, at the date of publication, 8.4 viable calves per cow, with a twin ratio of 0.72:100. Thirty-eight other heifers from eight herds of ordinary fertility, bred to the same bulls and constantly associated with the seventeen of high fertility, produced an average of 5.8 viable calves, with a twin ratio of 3.90:100.

In seeming contrast Wing[271] records sixteen twin pregnancies in the Cornell University Holstein-Friesian herd (Table I, including all breeds in the dairy, need not be confused with Wing's bulletin), showing sixteen (50%) of the twins aborted or non-viable. Among the viable twins were three pairs of heifers and one fertile heifer twin to a bull. The group of seven averaged seven calves, far above the average for the herd. Twin heifers occasionally breed well. But of the thirty-two twin fetuses only seven can be traced as fertile females. After having twinned, twelve of the sixteen twinning cows produced no more viable young; the other four yielded a total of six calves, or an average for the sixteen twinning cows of 0.375 calves per cow after having twinned.

Lubbehusen, Fitch and Boyd[125] vaccinated twenty-seven non-pregnant females with living cultures of *Bacillus abortus*, and

TABLE I

Breeding Record of 18 Heifers Bred to Calve First in 1912.

A—Abortion. B—Bull. H—Heifer. S—Sterile. X—Premature Birth.

No.	BORN	1912	1913	1914	1915	1916	DISPOSITION
1	12- 6-09						Destroyed 1912 at term, fetal emphysema, maternal sepsis.
2	10- 6-09	A					Died of metritis.
3	2-24-11	A					Discarded account sterility.
4	10-30-10	A					Discarded account sterility.
5	9- 6-10	A	A				Discarded account sterility.
6	9-29-10	BX	S	BH	S	B	Discarded account sterility.
7	11- 6-10	A	BB				Died of metritis.
8	4-26-11	H					Discarded account sterility.
9	7-20-10	B					Retained afterbirth, sterile, discarded.
10	1- 1-11	B	B				Gangrene of udder, discarded.
11	2- 5-11	A	B	B	B		Discarded, inefficient.
12	9-22-10	A	H	B			Died, indigestion.
13	10- 5-10	H	B	H	B		Discarded.
14	10-20-10	H	S	B	B	B	
15	10-18-10	B	H*	S	BB		*Died.
16	10- 5-10	A	B	B	B	H	
17	10- 5-10	A	H	B			
18	12- 9-10	A	H	H*	H	B	*Died.

Original females remaining in herd in 1916	5=31.2%
Viable heifer calves produced in 5 years	10=0.6 per cow
Viable bull calves produced	25
Ratio viable bull to viable heifer calves	250:100
Total pregnancies	51
Pregnancies terminating in abortion or non-viable young	12=23.5%
Twin gestations	3=6:100

studied them through fifty-one pregnancies, with three twin conceptions, or a ratio of 5.88:100. All three "calved normally," two of them retaining the afterbirth and the third dying during parturition. The two survivors were discarded as sterile. Among thirty-three controls, with eighty pregnancies, there were no twins. The vaccinates with a twin ratio of 5.88 had averaged 1.9 calves; the controls with

a twin ratio of 0 averaged 2.4 calves. Any critical analysis of bovine twinning, when ample material is used, shows beyond all question that twin gestation is disastrous. The abortion rate is many times that of single pregnancy; the mortality among twinning cows is very high; the sterility following twinning is appalling; many of the viable twins are inferior in size and fertility; and a great many are asexual monsters—free-martins—which will be considered presently. Nevertheless, when a given cow expels an abnormal number of young, be they alive or dead, the occurrence of the phenomenon attains the headlines in the press. Thus Fleming[62] cites McGillivray, who reported a cow which bore twenty-five "calves" in seven years. The highest number at one birth was six, all dead. It is not recorded that any one of the twenty-five calves was of any value, but the interesting adventure is suggested of establishing a twin-bearing breed of cattle, without having presented any evidence of the value of such biparious cow constructed from a uniparous animal.

Although some breeds of sheep appear to have a marked trend toward biparity, Thomas[194] submits data showing that in his observations the mortality of young for singles was 0, for twins 9 per cent and for triplets 25 per cent. Twins are well borne. In triplets two are necessarily located in one horn and serious conflict arises over placental area available for each. This, added to the increased genital load, multiplies the mortality as the numbers advance. Extensive data in sheep and goats are wanting, but such limited observations as have been available seem to indicate that, especially in triplets and higher multiples, the ewe follows the rule of mares and cows: the multiple conception is preceded by a period of genital disease. In ordinary flock breeding, according to my very limited observation, triplets occur in higher ratio toward the close of the lambing season, suggesting that the triplet-bearing ewe had required repeated copulations at several heat periods for conception. Triplet ewes appear to be largely sterile.

Medical authorities view the twin problem from a somewhat different angle, emphasizing chiefly the high incidence of abortion, premature birth, non-viable young and the mortality of the mothers. They fail to dwell upon the occurrence of preceding sterility or the fertility of the woman following twinning.

Uniovular or identical twins occupy a prominent place in the discussion of human twins, occurring in 15-25 per cent of all twin gestations, and are said to be accompanied by higher mortality of both mother and young than occurs in binovular twins. Uniovular twins are not authentically known to occur in domestic mammals.

Medical writers generally hold that when definite fusion of the placentae and chorions occurs, the twins are uniovular. The general belief is that the newly fertilized ovum undergoes complete fission of its cells, while yet few in number, not later than the morula stage. This hypothesis is vigorously attacked by Prof. Alfred Greil of Innsbruck,[73] who submits important evidence that the alleged fission of the morula or earlier embryo does not occur. Instead he believes that the uniovular twins have their origin in the pathological development of two germinal areas upon the blastoderm or the primitive vitelline sac. He denies that uniovular twins are uniformly identical and claims that there may be an indescribable number of variations, dependent upon the vascular supply and other factors, which may lead to inclusion or parasitic monsters, mongolian idiocy and other discordant forms without end. Identical twins are due to the fortuitous circumstance that the two germinative areas have an equal number of cells, have access to equal nutritive supply and keep pace in their growth.

The hypothesis that a common placenta with vascular anastomosis is proof of identical or uniovular twins is not credible in domestic mammals. In the cow, twin or multiple ova regularly have a common chorion, due to early fusion, and a very conspicuous communal vascular supply is formed. The same chorionic fusion occurs in the sheep and goat, less the vascular fusion. In the sow I have observed complete fusion, including the vascular supply, of the allantochorions of six swine ova, with corpora lutea indicative of the independent origin of all six.

The familiar occurrence of identical twins in man, and the absence of conclusive evidence that they are developed in domestic animals, seems highly confusing if the current hypothesis is accepted that they are due to fission of the cell-mass of the fertilized ovum at a period not later than the morula stage. The acceptance of the views of Greil may throw light upon this apparent discordance between twinning in man and in domestic animals. Under the current hypothesis of fission of the ovum not later than the morula stage, it would logically be expected that uniovular twins would be at least as frequent in domestic animals as in man.

Medical authorities commonly regard acardiac and amorphous monsters as unequal fission of a fertilized ovum. If the hypothesis of Greil be accepted, and uniovular twins be regarded as a pathological deviation in which two, instead of one, germinal areas develop upon a single blastodermic vesicle, the phenomenon is brought into

essential harmony with observations in domestic animals, in which the monsters unquestionably arise from separate ova but must establish vascular anastomosis (placental union) with viable ova. Fincher and Williams[57] record substantial evidence that the two fertilized ova emanate one from each ovary and are located one in each cornu. So it follows that acardiac and amorphous monsters are common in woman, where uniovular twins are not rare, with a common placenta; and in the cow where binovular twins, usually with one ovum from each ovary, immediately fuse and establish a common allantochorion and placenta.

Medical writers do, however, recognize the important pathological significance of twinning. De Lee[40] says, "Placing this subject in the pathology of pregnancy might elicit comment, but the succeeding pages, showing the frequency of maternal disease and of fetal death, will prove that in the human female, having more than one offspring at a time is distinctly abnormal." Medical writers fail to indicate that prior to twin conception of primipara subnormal sexual or general health was recognizable; that in pluripara twin pregnancy has frequently been preceded by abortion, retained placenta or endometritis; or that the twin conception has occurred unduly early after a prior birth. There is general agreement that there is increased frequency and virulence of the diseases of pregnancy and birth—toxaemia, eclampsia, puerperal sepsis, post-partum hemorrhage—and that premature labor occurs in more than seventy-five per cent of cases. J. Whitridge Williams[226] emphasizes the higher incidence of amniotic dropsy in twin gestation, and it seems to be the general consensus of opinion that the hydramnios is most prevalent when the twins are uniovular or identical. Monstrosities are common.

The ratio of intra-uterine death of the young seems to be very much higher in twins than in singles. Apparently there is a contrast in the outlook for the lives of new-born human twins, and for those of the mare and cow. De Lee considers the prognosis for the lives of new-born twins as bad, although he believes that Ahlfeld has placed the mortality figure too high when he estimates it at seventy-five per cent during the first year of childhood. Adding the abortions to the high mortality during the first year, the death rate of human twins becomes appalling, and with this high mortality the inference is unavoidable that among those which survive for more than a year after birth there is an undue ratio of weaklings.

The abortion rate of twin pregnancy in the mare is so nearly total that little opportunity is given for the study of those born.

It is known that a large ratio of those which are alive when expelled die immediately, leaving but very few viable foals, many of which are dwarfs and dismissed as not worthy of study.

I would estimate that fifty per cent of bovine twins are aborted. Among those expelled alive there are numerous immediate deaths, and during early calfhood the twin mortality is very high. Retained placenta is extremely common at the termination of twin pregnancy. When birth is associated with retained placenta, the calves largely die young, whether twins or singles. It does not seem, however, that so many as seventy-five per cent of twin calves die during the first year, as Ahlfeld estimates for human twins.

The primary cause of the high mortality of new-born human twins is not definitely stated. Obstetricians are generally agreed that twin conception is abnormal, from which it might be inferred that the basic cause is within the sex cells themselves. Later, however, there is apparently a tendency to attribute the mortality of the young to the prevailing premature birth of twins. The two causes are difficult of accurate measurement. Numerous premature births occur in which the child and its placenta are regarded as physiological but the infant too immature to live. Such premature births are essentially unknown in domestic animals. An abundance of non-viable young are born, either prematurely, at term, or after prolonged gestation, and die immediately. Their death is not, at least usually, dependent upon the duration of gestation; on the contrary the duration of gestation and date of birth are dictated by the intra-uterine disease. The mortality of new-born bovine twins is inverse to the duration of gestation. Many of them are born with profuse diarrhea, the amniotic cavity being filled with thin meconium and the calf thickly smeared with it, as is frequently observed in abortion. The other non-viable young generally show definite and serious disease of the placenta. The diseased fetus is expelled in behalf of the life of the mother.

The apparent contrast in the mortality of new-born human and bovine twins may be partly due to the marked differences in the prenatal development in the two species. The clinically healthy calf, born at twenty, or slightly more days below the average, is on its feet in about thirty minutes, and when forty-eight hours old can outrun a ten-year-old child. Unless the dairyman is on his guard, the heifer calf may become pregnant at six months, before the infant can creep.

The conflict between twins does not end with intra-uterine location and placentation. In all species of uniparous mammals there is

frequent dispute of right-of-way in the birth canal, which leads to twin dystocia, because parts of two fetuses enter the canal simultaneously. This is virtually unknown in multiparous mammals, since there are effective safeguards which insure that but one fetus shall enter the birth channel at a time. This is well illustrated in Figs. 17, 18.

In all unipara the fetal membranes of the single fetus completely fill the utero-cornual cavity and the contained young has command of the total energies of the entire placental organ; in multipara each ovum has its established placental zone. After birth, when the mammae replace the placenta in the nutrition of the young, the new-born of the uniparous mare or ewe takes milk from both glands, and the calf sucks all four teats alike; each multiparous young pre-empts one mammary gland, to which it ordinarily has undisputed right during its nursing life. Conflict in multipara only arises when more young are born than the female has mammae. When twins are born, conflict arises, and the stronger one chooses the teat it shall use. When triplets are born to the ewe or goat, the weakest one is automatically without milk.

ASEXUAL (FREE-MARTIN), ACARDIAC AND AMORPHOUS TWINS OR MULTIPLES

The name *free-martin* is of unknown origin and indefinite meaning. Probably it is most frequently defined as *an imperfect heifer born twin to a bull*. Others regard as a free-martin any calf born twin to a bull and having its urino-genital opening sub-anal, although it may be a perfect heifer of normal fertility. Imperfect bulls are born twins to bulls and imperfect heifers twins to fertile heifers.

This monster is by far most frequent in cattle. It has been authentically described in the sheep by Roberts and Greenwood[172] and in the goat by Keller.[105] It is probably not rare in the sheep and goat, in which infertility of a supposed female arouses scant attention. It occurs in swine.

Two hypotheses have been advanced regarding the etiology of these asexual, or imperfectly sexual monsters. At almost the same period, Keller and Tandler[103] and Lillie[121] arrived at the conclusion that the genesis of the free-martin is the passage of testicular hormones from a male to a female twin, owing to vascular fusion between their chorionic sacs. The hypothesis is in accordance with the tradition, which has not yet been established as a fact, that the free-martin is primarily a heifer. It is assumed that testicular hormones, not proven to exist at the period under discussion, pass from the



FIG. 22. Double Monster, Showing Almost Identical Color markings on the Median, and on the Distal Sides of the Divided Portions.

male twin to the ovaries of the female, arrest their development, and cause changes in the glands simulating in some cases the histology of the testes. This hypothesis, advanced in 1916-1917, has been generally accepted.

In 1934, I[256] expressed the belief, based upon clinical observations, that the free-martin has its genesis in a defective ovum, without the required anlagen for the evolution of a physiological genital system. It is commonly held that a fertilized ovum, without heart or general organization, does not undergo fetal development except it be in vascular communication with a normal, or near-normal, ovum. It is equally true, clinically, that a completely asexual embryo of any species of domestic mammal does not survive except it have vascular connection with a normal twin. Single fetuses of both sexes are born with very defective reproductive organs, which may bar reproduction, but they must have gonads with definite resemblance to ovaries or testicles.

According to this latter hypothesis, acardiac, amorphous and asexual monsters should be most frequent in those species in which vascular anastomosis between the intra-uterine young is most frequent and complete. In harmony with this presumption, this series of monsters is by far most common in cattle, where twin conception is not rare, and in which vascular anastomosis occurs generally in twins and multiples. Some believe that acardiac monsters originate by unequal fission of a single ovum. This view is not supported by clinical observations upon domestic mammals. Uniovular twins are not definitely known to occur. It needs be admitted that, had the alleged fission in Fig. 22 been completed, identical twins would have resulted. The acardiac and amorphous monsters are not in immediate contact with the normal twin, but the former have definite vascular cords (an afferent artery and efferent vein) which communicate with the allantoic vessels of the host at some distance from the umbilical cord. In a case cited by Fincher and Williams[57] Conklin diagnosed bicornual twin pregnancy. A fertile heifer was born from one horn and an amorphous monster, weighing nine pounds, from the other. Its vascular cord, about one foot long, anastomosed with the allantoic vessels of the normal twin at a point about two feet from the navel cord of the latter. Each ovary had a corpus luteum. Clearly the monster was due to a separate ovum, not a fission. As in ordinary twins, so in acardiac or amorphous monsters, there is commonly a history of precedent genital disorder in the cow.

The hypothesis that the free-martin is a female which has been unsexed by the invasion of testicular hormones, through vascular

anastomosis with a male twin or litter mate, is highly confusing, in many respects, to the clinician. Hughes[95] describes asexual swine fetuses in which vascular anastomosis with male litter mates was evident. In one case, however, the male had died at 12 cm. and the female had reached 15 cm. Her genital system was approximately normal. Hughes concluded that the female genitalia had recovered after the death of the male. Since the growth of the swine fetus, with a gestation period of about 115 days, is very rapid after the fetus

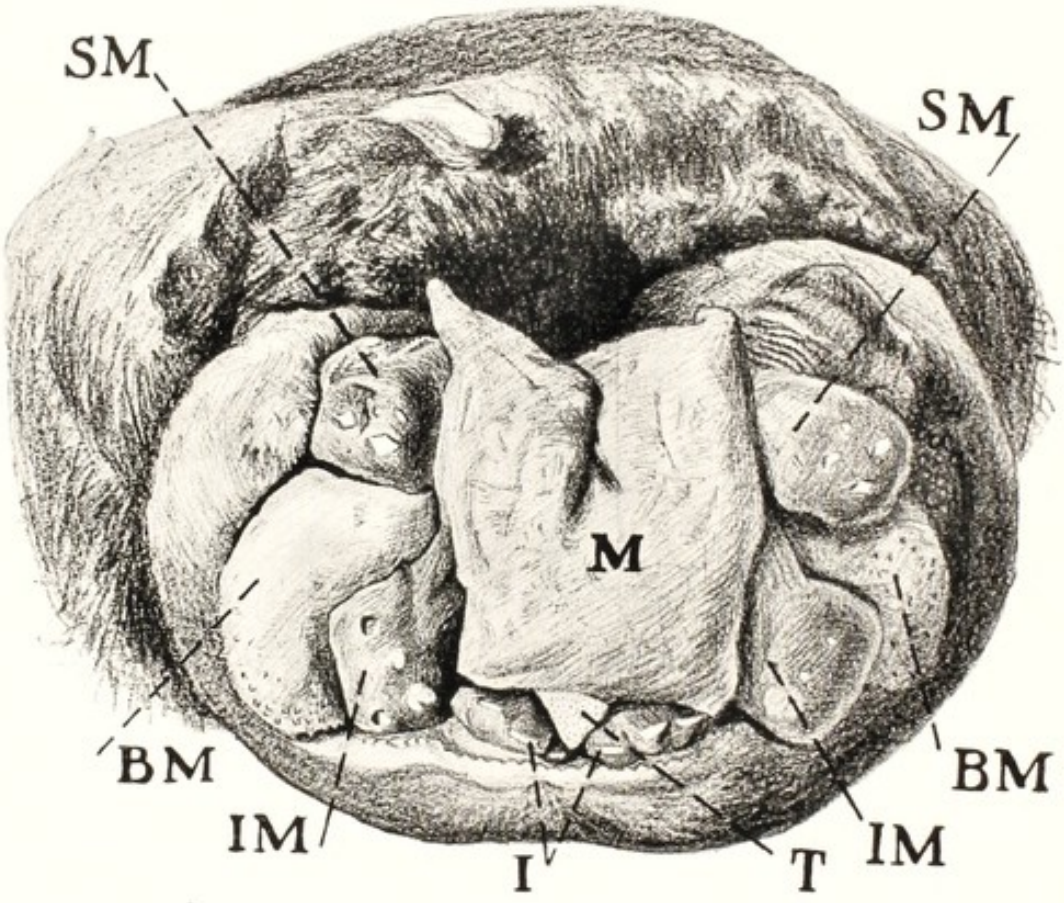


FIG. 23. *Amorphus globosus*. Bovine.

M, Muzzle; *SM*, superior maxilla showing white denticles; *IM*, inferior maxilla with denticles; *BM*, buccal mucosa with papillae; *T*, tongue; *I*, incisors.

has reached 12 cm. in length, the conclusion of Hughes that the female genital organs, repressed by testicular hormones, could recover while the fetus grew 3 cm. appears extremely doubtful. Fincher and Williams[57] have recently shown, beyond dispute, that vascular anastomosis between swine litter mates does not always, if ever, unsex the females involved. The uterus of a sow contained eight fetuses varying in length from 15 to 20 cm. At the ovarian pole of the right horn there was a healthy female fetus in an independent chorion, and a healthy male fetus at the ovarian pole of the left

cornu, with independent sac. The other six fetuses, four males and two females, occupied a common chorion, with a common vascular supply. The vessels were large and readily followed by the naked eye from end to end of the common chorion, about 3 m. long. The two females were perfect. Their genital organs were quite as well developed as those of the independent female. When four males, in a common chorion with two females, fail to interfere visibly with the genital development of the latter, through the agency of

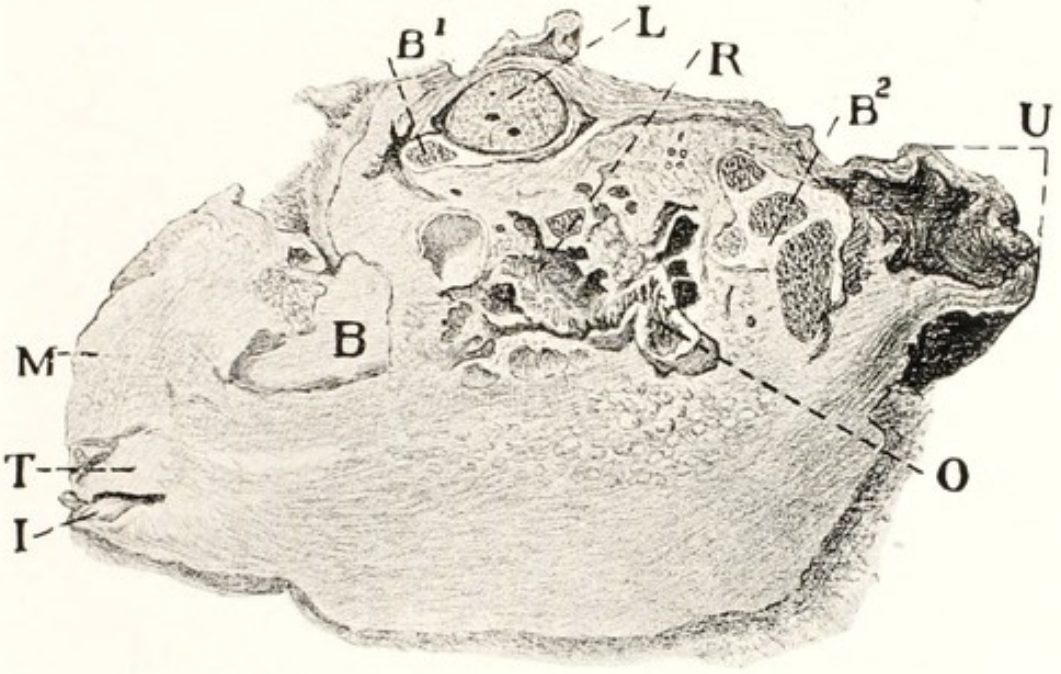


FIG. 24. Sagittal Section of Fig. 23.

I, Incisors; *T*, tongue; *M*, muzzle; *B*, skull bone?; *B'*, *Sternum?*; *B"*, unidentifiable bone; *L*, lung; *R*, rumen; *O*, omasum; *U*, "navel."

testicular hormones, the validity of the Lillie hypothesis is seriously challenged.

According to the hypothesis I have offered, amorphous, acardiac and asexual monsters constitute a chain of phenomena, which pass, by imperceptible gradations, one into another; they have definite scientific interest and are of distinct economic importance. The most primitive type of the group, *Amorphous globosus* (Gurlt), consists of a mass of fibrous, or other primitive tissue, enveloped in a thick, hairy skin. Spheroidal in shape, it may be but two or three inches in diameter. It has a vascular cord, consisting of an afferent artery and an efferent vein. No evidences are present that a neural groove has existed nor that the germinal area of the blastoderm has folded ventralwards, in a movement to form a gut. No amnion has

been recorded. In the absence of a hind gut, no allantois appears possible. There is observed only the ball of hairy skin, covering tissues which do not form organs.

A more advanced type is shown in Figs. 23-24. In this monster, there has apparently been a defective neural groove, without a visible cerebro-spinal nervous system. There has been an imperfect ventral

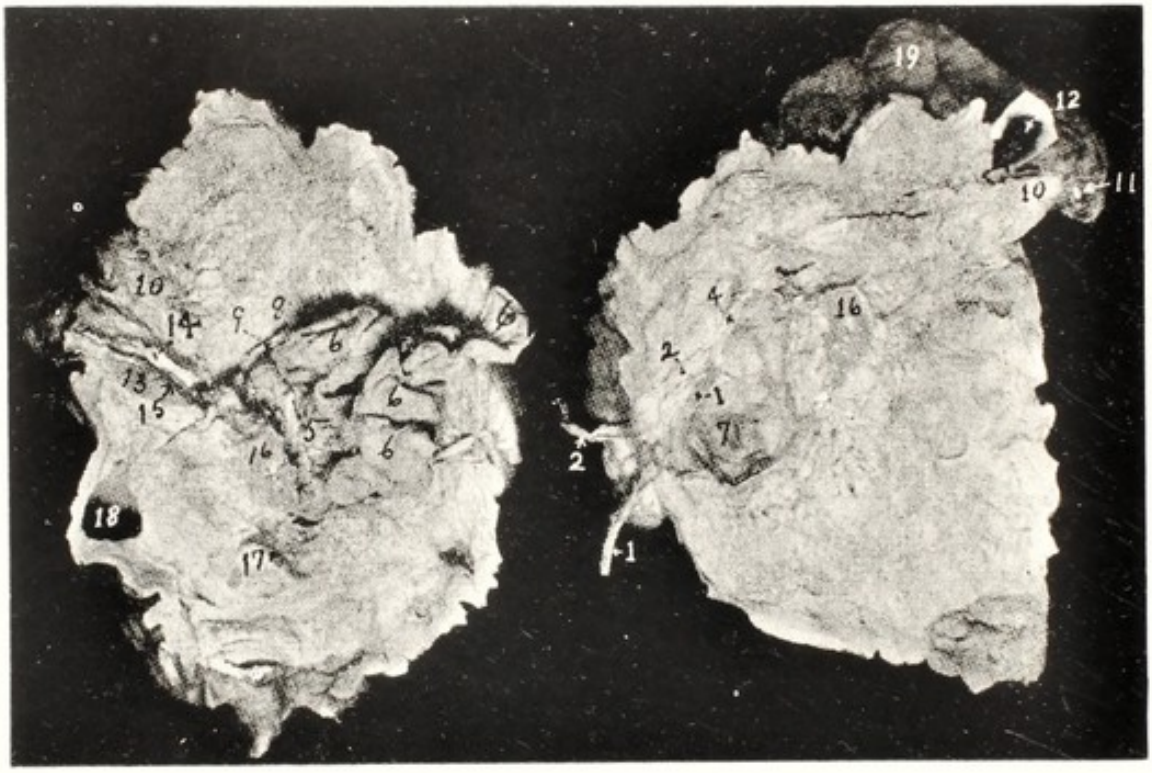


FIG. 25. Bovine Acardiac Monster, Twin to a Viable Bull.

The host was born at 295 days: the acardiacus, with a diameter of 43 x 30 cm. caused dystocia and was removed manually.

1, 1, the afferent artery; 2, 2, the efferent vein; 4, the vein and artery in a common cord—at, 1, and, 2, the vessels have been separated by dissection; 5, mesentery of small intestine with an intricate plexus of mesenteric arteries; 6, 6, 6, small intestine; 7, empty peritoneal cavity, the gut, 6, being attached to the opposite half; 8, vacant pleural cavity; 9, diaphragm; 10, 10, oral cavity; 11, incisor; 12, ear; 13, cranial vault; 14, cranial base; 15, brain and spinal cord. The anterior neuropore remains open, as is not rare, especially in the *Schistocephalus bifidus* bovine monster; 16, the spinal column transversely divided; 17, unidentified osseous mass; 18, cyst; 19, projecting mass of tissue covered by skin.

folding of the germinative area of the blastoderm, with the definite formation of the stomachs, but not of the hind gut. There may have been an amnion, but in the absence of the hind gut, and hence of the allantoic stalk, there could be no allantois. The stomatodeal pit is comparatively gigantic, and shows clearly the teeth, the characteristic buccal mucosa, the tongue, etc. No pharynx is detectable, and hence

no heart expected. There are no visible signs of the Wolffian system, or of its derivatives, and no endocrine glands.

Further advancement in organization is shown in Fig. 25. In this case there is a definite, though imperfect, cerebrospinal nervous system, and the blastoderm has undergone well defined ventral folding, to constitute a gut, enclosed within a definite peritoneal cavity. Only the mid gut is present, with a definite mesentery and a liberal vascular supply. Pharynx, heart, aorta and allantois are wanting. No Wolffian or endocrine system is present. The absence of a reproductive system can not be attributed to the invasion of the monster by testicular hormones from the viable male twin; the arrest occurred prior to the formation of the Wolffian bodies and endocrine glands.

As the teratological approaches the physiological, the pharynx and heart appear. The heart may remain in the pharyngeal or cervical zone. The Wolffian system appears, from which develop later the urinary and genital systems, both of which are often simultaneously teratological, as indicated in Fig. 7. In this monster, with the gastrointestinal system protruding through the navel, but one kidney formed, with a single median ureter, and a one-horned uterus. From the primitive amorphous monster to the physiological fetus, every imaginable defect occurs in both twins and singles. The otherwise physiological, but sexually neuter free-martin, like the acardiac monster, must have vascular communication with a viable twin in order to attain fetal development.

Instead of having a normal heart, like that of the free-martin, the amorphous monster is without intrinsic circulatory system. The afferent artery and efferent vein are possibly the derivatives of the vitelline vessels. The circulation of the monster is reversed, and its blood supply is from the used blood of the host embryo on its way to the placenta. The monster is accordingly dwarfed, owing to its defective vascular supply.

The reproductive, arising secondarily from the Wolffian system, is the last to be formed in the embryo. While all other systems have been so completely developed during fetal life that they may take up their ultimate function prior to or immediately after birth, the reproductive organs, not at all essential to the life of the individual, are functionless.

Two forces conspire to cause a high ratio of asexual young in cattle: (1) dairy cattle suffer especially from genital diseases which disturb ovarian function, and frequently cause the simultaneous discharge of two or more ova, some of which are immature or

teratological, and (2) the allantochorions of bovine twins or multiples regularly fuse, including ample vascular anastomosis, enabling amorphous, acardiac and asexual monsters to survive as parasites upon normal, or near-normal twins.

Histologists fail to discover in typical free-martins either spermatozoa or ova in the primitive gonads. Thus in the gonads of the neuters of triplets and quintuplets which I have described,[260] the histological study showed that no differentiated sexual tissues were present in the gonads, and that, as has usually been found, the structure of the tissues is most suggestive of the male. The general tradition that the typical free-martin is a female which has become unsexed by hormones from a male host, inferentially denies that an immature or asexual ovum may escape from a pathological ovary and become fertilized. It is undeniably known that immature spermatozoa are discharged by the male and that certain sires with defective spermatozoa beget defective young. It seems inevitable that the same principles should aply to the reproductive cells of both sexes.

The advocates of the hormone hypothesis contend that the asexual individual has the general appearance of a female. This belief is perhaps largely due to the fact that in most cases the urino-genital outlet is sub-anal in the normal vulvar zone. But Fincher and Williams[57] show authentically that the urino-genital exit may be located anywhere between the anus and umbilicus, and its form and structure may be such as to call for vivid imagination to permit the observer to designate it as vulva or by other term used in normal anatomy. Although some contend that the adult asexual looks like a cow, and others that it resembles a bull, or a castrate, any experienced clinician can generally, if not always, identify at a glance the typical adult free-martin, without observing the urino-genital outlet; it looks like a free-martin and not like any other known animal.

A second argument advanced is that, unless the free-martin is classed as a female, the expected sex ratio is upset and there is a gross excess of males. It has been shown, however, by McPhee, Russell and Zeller[132] that when swine are inbred, in addition to a decline of the average number of pigs in a litter, and decrease in average birth weight, there is a distinct advance in the ratio of males. In all animals the aborts and non-viable young are predominantly male. In cattle aborts I have recorded[257] a ratio of 270 males: 100 females in primipara, and 252:100 in pluripara. In Table I, it is shown that in a group of genitally unsound cows the ratio of live male to female calves was 250:100. The rule, at least for dairy cattle and for swine, is that when the reproductive health of a

large group of breeding animals becomes distinctly subnormal, the ratio of male young born becomes excessive. Wells[223] reports upon two cows which offer a remarkable contrast in the sex ratio. One bore eighteen single calves and at twenty-three years dropped twins. Of the eighteen singles, 14 were heifers. The second cow produced twins in each of her three first pregnancies and triplets in her fourth. All nine were bulls. She next dropped one single, a heifer. Admittedly they are extreme cases.

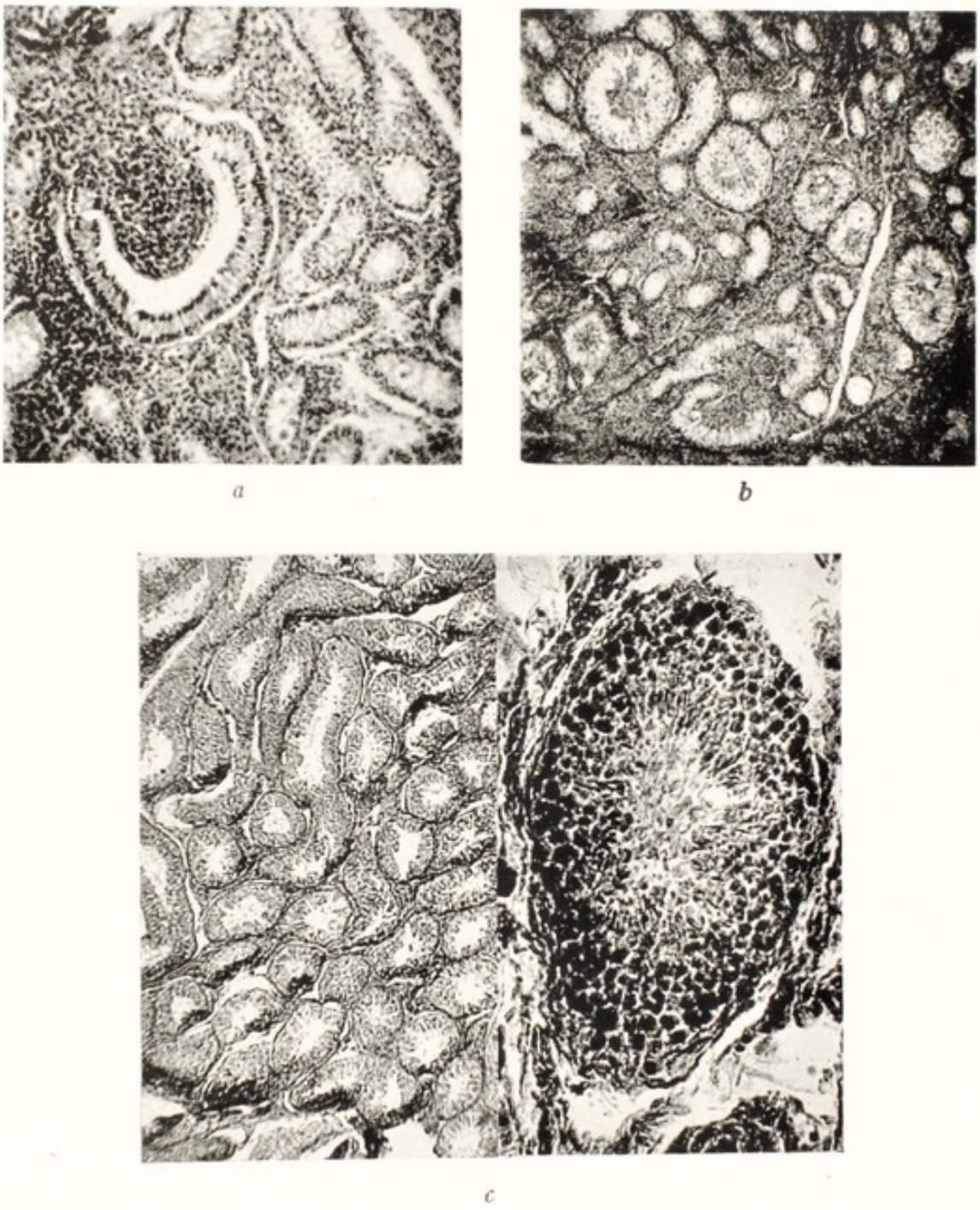
Aborted human ova are predominantly male, and the earlier the abortion occurs the higher the male ratio. Taussig[193] cites Mall as having found an excess of males in early abortions. Among the abortions occurring during the first month of gestation, 80 per cent were teratological.

Early abortion is rarely observed in either the mare or the cow. Since they are indeciduate, the abortion is not accompanied by notable hemorrhage or other clinical phenomena; the ovum is expelled unseen and the event is unavoidably designated sterility.

Many early swine embryos die but are not expelled if their litter mates live. Some of them are resorbed; other older ones undergo papyraceous mummification. The latter have not been studied.

Teratological deviations of the reproductive organs of dairy cattle abound in singles, twins and higher multiples. They are of every possible gradation, and the degree of arrest in singles and multiples overlaps. In general it may be stated that the higher the multiple the greater the incidence of monstrosities and the higher the combined ratio of males and sexually defective or asexuals to fertile females. Thus I have related[260] one instance of quintuplets consisting of four neuters and one male, and of triplets with two neuters and one male. In the quadruplets of Hutt[96] there were no fertile females, and among eight recorded progeny the dam produced no fertile female.

While it is not known that such marked neuters as are frequently observed in bovine twins and multiples occur in singles the aberrations in the reproductive systems of the latter may be very extensive. Fincher and Williams[57] describe a Brown Swiss single monster of fashionable pedigree, sold as a bull when a young calf. It failed to develop the appearances of sexual desire of either sex, was unthrifty and hypognathic. It had intraperitoneal, spheroidal gonads, 5 x 4.75 cm., a large uterus with a grossly excessive number of caruncles, and a normal pelvic urethra and penis. The gonads were not definitely differentiated; some groups of cells, as shown in Fig. 26, resembled seminiferous tubules without spermatozoa or



a *b*

c

FIG. 26. Sections of Gonads from Single, Mixed-sexed Bovine Monster, with Section of Normal Bovine Testicle for Comparison.

a, Section of arrested gonad suggesting ovisacs; *b*, section of same gland resembling tubuli seminiferi; *c*, sections from normal bovine testicle, shown under high power at the left, and low magnification at the right. *(Cornell Veterinarian)*

spermatids, while other groups were more suggestive of Graffian follicles without ova. The dam of the monster, so far as her history was available, never had normal heat periods, suffered from nymphomania, was repeatedly handled because of sterility, had aborted, and at the termination of each gestation lasting to term had dystocia and

retained placenta. A monster, therefore, was simply in harmony with a universally abnormal breeding history. In contrast, Roberts and Greenwood[172] studied a twin to a normal bull. The urino-genital exit, located as shown in Fig. 27, somewhat ventral to the normal vulvar zone, consisted of irregular folds of the skin and subcutaneous tissues, so shaped that Roberts and Greenwood designated it as a *vulva* and the erectile organ within it as a *penis*. A definite pelvic urethra was present, surrounded by the prostate gland, the *penis* protruded slightly from the urinary exit, curving upward, the urethra opened upon the dorsal surface and the urine was projected "straight up into the air," a quite common phenomenon in free-martins. The cause of the upward direction of the stream when some free-martins urinate has not been studied. It is probably due in some cases to the failure of the urino-genital canal to form a urethra, remaining open as a groove along the dorsal side of the phallus. I saw casually, without available history or opportunity for autopsy, an adult bovine monster, which behaved asexually. There was a deep urino-genital groove, lined with mucosa, extending downward from the anus for over one foot. Roberts and Greenwood designate a small pocket ventral to the end of the phallus as a *vulva*, despite the presence of a definite pelvic urethra with a normal prostate—structures which occupy the normal vulval zone. They concluded that there had been a *rotation* which had placed the *penis* dorsal instead of ventral to the vulva. Another plausible interpretation would



FIG. 27. The Urino-Genital Exit of a Bovine Monster, Twin to a Bull and Designated "Free-Martin."

It will be observed that the urinary outlet is abnormally distant from the anus, the clitoris-penis projects upward and the tuft of hairs growing from the ventral border of the opening is monstrously long for either preputial or vulvar tuft. (Roberts & Greenwood)

be to say that the phallus had beneath it a small sac analogous to the prepuce of the clitoris, and that no vulva was present.

In conformation the animal resembled a bull and bellowed like a bull. The grossly abnormal urino-genital exit, Fig. 27, had a tuft of hairs nine inches long—a length of hairs not observed with a normal vulva, and not occurring with a normal prepuce. The single described by Fincher and Williams[57] was definitely more arrested in genital development than was the free-martin of Roberts and Greenwood. It is contended, under the prevailing hypothesis of free-martinism, that the latter monster was primarily a heifer and had been converted into an almost complete bull by the testicular hormones from its host. If that be admitted, the further conclusion becomes inevitable that in bovine twins of opposite sexes, the testicular hormones may transform the female ovum into a complete male and lead to the birth of twin bulls, one of which was originally a heifer.

Single and twin monsters of distinctly female type also overlap in the intensity of aberration. The incestuously bred singles of Fincher and Williams,[53] as indicated in Fig. 6, were quite as greatly arrested in development, in so far as may be learned, as were the infertile quadruplets of Hutt[96] and Curson.[38] The host in bovine asexuals need not be a male. Wagner[211] observed a typical free-martin twinned with a fertile female. Not infrequently the defective twin to a male has functioning ovaries without the presence of a vagina. She may come in estrum, and rupture of the vulva may occur from the impact of the penis of the bull in attempted coitus. I have known of one such case. Galli[67] reports a series of defectives, born co-twin to males, which came in heat, and were peremptorily slaughtered owing to peritonitis caused by rupture of the aberrant vulvo-vaginal canal, following coitus.

Scant study has been given to asexuals in multipara. Schäper[177] observed in a herd of purebred swine, in which inbreeding was practiced, a peculiar type of offspring designated *pummels* or roly-poly pigs. They appeared normal at birth but early became excessively fat and, upon attaining a weight of eighty to one hundred and sixty pounds, would unexpectedly be found dead. None of them bred, or was known by Schäper to have been in heat. In a personal communication, Schäper relates that no study had been made of the genital organs.

According to my belief, the hosts of most free-martins are males, not because the asexuality is due to the action of testicular hormones, but owing to the clinical fact that when a female is suffering severely

from genital disease or sexual overloading, fertile females are few among her progeny and the ratio of males is excessive. Reference has already been made to the quadruplets described by Hutt. It is clear that the cow had suffered from genital disease. Her breeding history indicates that at twelve years of age she had produced four bulls, no fertile heifers, one typical asexual, one non-viable twin classed as a heifer. Two of the quadruplets came in estrum but proved infertile. Inevitably no free-martin could emerge otherwise than as co-twin to a bull, because at each parturition she gave birth to a bull.

Further evidence that free-martinism is not the destruction of female genitalia by the action of male hormones is provided by monsters of mixed sex. It is not very rare to find in swine a testicle upon one side and an ovary upon the other, with near-normal derivatives of the Muellerian ducts (oviducts, uterus, cervix and vagina) succeeded externally by either a vulva or a penis. Corner[34] reports a case in which there was an abdominal testicle on one side and a normal ovary upon the opposite side, with 4 recent corpora lutea. One ovum was identified in the oviduct. The testicle, as is normal for cryptorchid gonads, was infertile. If one of the gonads had inhibited the development of the other, by its hormones, the result was the reverse of the Lillie hypothesis, and the female had inhibited the male organ. But abdominal testicles are regularly infertile and the action of ovarian hormones is not necessary.

Equine monsters are not very rare in which two cryptorchid testicles are present, with normal oviducts, uterus and vagina, succeeded by a vulva of normal size, and a compromise clitoro-penis. The testicles do not inhibit the development of the derivatives of the Muellerian ducts and of a vulva. The individual may display bisexual behavior of an abnormal type.

If male hormones unsex a female twin, because of anastomotic circulation between the two, it would appear even more certain that in single monsters, with one testicle and one ovary, the male hormones would inhibit the development of the ovary, the two having a common circulation. On the contrary the female gonad takes precedence and sometimes functions while the male gonad is not known to have functioned in any case. This reminds one of the effects of hybridization between *Equus caballus* and *Equus asinus*, and between *Bison Americanus* and *Bos domesticus*. The male hybrid is infertile in each case; the female mule rarely breeds, while the female American buffalo-domestic cow hybrid is regularly fertile.

In themselves the hypotheses concerning free-martins and acardiacs, like many hypotheses, are of little practical concern. There is another side to the question. Under the prevailing hypothesis, free-martins and acardiacs arise as the whims of chance, and there is nothing to be done but to accept the loss. Neither prophylaxis nor therapy is available. If, however, the alternate hypothesis be accepted, the incidence of the phenomena may be effectively controlled. The release of the twin ova and their fertilization may be prevented and the consequent monsters avoided. The outstanding cause of twin or multiple ovulation is an unstabilized state of ovarian function, usually manifesting itself in definite clinical signs. Several factors leading to twin ovulation stand out clearly:

(1) Heifers which have been unhealthy as calves, like those in Table I, ovulate erratically, and when they produce two ova simultaneously, one is probably immature or otherwise defective. If fertilized, it will probably become a free-martin, acardiac or other type of monster. The remedy lies in the application of proper health measures during early post-natal life.

(2) Ovulation in dairy cows is unstable during the first sixty days or more after the termination of a pregnancy, especially when associated with abortion, abbreviated gestation, retained afterbirth or other important pathological phenomena, as indicated in Table XI. The palpation of the ovaries of such cows per rectum reveals that for a considerable period of time following the termination of pregnancy, twin or multiple ovisacs or corpora lutea are frequent. Consequently early conception favors twinning. The danger is avoidable by withholding the animal from breeding for 80 to 100 days or more until stable ovulation is signified by the advent of heat periods at normal intervals and the normal behavior of the cow during heat. Ignoring these precautions invites twinning and the accompanying disasters.

(3) The ratio of twin pregnancy in the mare is far higher in those which are bred and conceive at the first (9th day) estrum postpartum than in those in which breeding is postponed until the thirtieth day or later after having foaled.

(4) The most spectacular antecedent of twin gestation, with the production of free-martins, acardiac and other monsters, is nymphomania. Numerous cases recover, but, as pointed out by Clapp,[31] if rebred early, twinning becomes extremely frequent. During nymphomania there are usually present two or more cystic ovisacs. During convalescence the tendency for several ovisacs to ripen simultaneously is well marked. The danger is avoided by patiently awaiting

the recurrence of estrum at regular intervals. Additional security is readily available by means of rectal palpation of the ovaries while the cow is in heat, and withholding the animal from breeding if two or more ovisacs have ripened.

(5) After mares have been repeatedly mated with famous stallions which are infamously diseased, some fertilizations occur, with death and disappearance of the ovum, leaving the reproductive system in a state of chaos, and physiological ovulation interrupted or destroyed. When such mares are tottering toward recovery, twin ovulation and, if bred, twin pregnancy are probable. The remedy is obvious.

Among valuable dairy cattle it is sometimes desirable to determine if a given calf, born co-twin with a bull, is a heifer or a neuter. Observations lead to the inference that in such mixed twins the ratio of heifers to neuters is about 1:7 or 8. With such a ratio it is imprudent, economically, to retain the group of 7 or 8 until of breeding age, pending an authentic diagnosis.

The phallus of the free-martin is commonly a compromise between a clitoris and a penis; it is too large for a clitoris and too small for a penis. The organ often curves upward toward the tail, the urethra is not surrounded by the phallus and the urinary stream strikes against the concave anterior surface of the phallus and is deflected upwards; when the calf urinates the jet arches upward and backward, wholly unlike either heifer or bull. If a large sound is used which can not readily pass into the urinary bladder, if the animal is a heifer, the sound passes into the vagina to the cervix; if a neuter, the sound lodges against the anterior end of the pelvic urethra where the hymen is located in the female.

Some practitioners state that reliable diagnosis may be made by rectal palpation of the new-born calf, especially within the first few days. The index finger is passed into the rectum and the tip bent down toward the bladder. If the calf is a heifer, the cervical cone is readily palpable, and just beyond, the uterine cornua may be identified. If the calf is an asexual, the finger-tip contacts the urinary bladder, covered by a very thin membrane. Instead of the uterus one may feel the vestiges of the Muellerian ducts as parallel fibrous cords. Other clinicians report difficulty in thus reaching a reliable diagnosis.

A visual diagnosis may be made with the aid of a suitable speculum or an ordinary test tube. The latter may be passed into the vulva and, if desirable, the area illuminated by means of a flashlight. The meatus urinarius then becomes visible at the anteroventral end of the vulva. If the animal be a free-martin, no vagina

is present. Very rarely, an otherwise normal heifer may have a persistent hymen, which may mislead the examiner. That is not important, because the female should be discarded as a breeder, although the persistent hymen may be overcome by surgical operation.

Usually a finger may be passed into the genito-urinary opening. If the individual is a heifer, the finger passes into the vagina and contacts the cervix; if a neuter, the finger is halted just above the meatus urinarius. If the examiner is not experienced, a normal heifer calf should be used for comparison. When the calf has become sufficiently developed to permit the introduction of the hand into the rectum, identification is readily made. Sometimes the twin to a bull is an imperfect female, with functioning ovaries. Galli,[67] as stated above, has observed coital rupture of the urino-genital canal. This suggests that, before breeding such animals, rectal and vaginal examination be made and the safety of the coitus be determined.

Generally speaking, bovine twins are undesirable as breeding animals. They are usually born after abbreviated gestation, which importantly lowers their breeding strength. A more serious obstacle to their breeding value is that they are largely teratological and are frequent bearers of structural defects, especially of the reproductive system. Some heifers twin to a bull breed well, but quite frequently their reproductive behavior is bad. Thus an extreme example is recorded by Andres,[5] in which a cow born twin to a bull produced five bull calves, two free-martins and ended her career, after a gestation of 369 days, with a male fetal cadaver weighing 217.8 pounds, removed postmortem because of irremedial dystocia. The fetus had suffered from profuse diarrhea and the amniotic cavity contained numerous hairballs. Unless there exist special reasons for the retention of twins as breeding stock, they should be discarded.

The importance of twin conception does not end with the twinning alone, but the incidence of twin pregnancy affords a valuable meter for the reproductive health of the herd, flock or stud. When adequate measures are applied for the control of twin pregnancy, free-martins and acardiacs, the same measures operate favorably upon the incidence of sterility, abortion and other interferences with reproduction.

Pathological Spermatogenesis

The sire constitutes one-half the breeding unit, which may vary from ten to one hundred females, or in the current boom in artificial

insemination in dairy cattle, the females assigned to a sire may be several hundred. Authetic data are not available for accurate measurement, but the ratio of infertile and subnormally fertile males closely approaches that of females. The low fertility of uniparous males does not stand out clinically so clearly as that of females. When physiologically but one ovum is discharged during a given estrual period, its failure of fertilization or viability stands out prominently. In contrast, Schmaltz,[178] citing von Bernhardt, places the number of spermatozoa at a single ejaculation by a healthy stallion at a minimum of ten billion. Since but one is essential to fertilization, the ratio of physiological spermatozoa in a given ejaculation of semen may be very low and fertilization remain possible, as is well shown by the stallion illustrated in Figs. 28, 121, and 131. The presence of vast numbers of motile spermatozoa has in the past given a false feeling of security in the fertility of a male despite the presence in many cases of a high ratio of defective cells. The belief has apparently prevailed that all motile spermatozoa are physiological.

Early writers upon sterility of male domestic animals concerned themselves chiefly with those in which spermatozoa were not produced or were not ejaculated at coitus, or, if ejaculated, were not motile. Such cases are not at all rare and the remedy is obvious. A far more serious economic problem arises when a sire visibly fertilizes a healthy female only after repeated copulations, or, when used upon a group of healthy females, causes recognizable pregnancy in only a small ratio of animals. It is even more disastrous when a normal ratio of females bred to a given sire become evidently pregnant and the young die during intra-uterine life, or are born nonviable, or non-fertile.

There is the further serious peril that, though a male is able to fertilize the ovum, because the spermatozoa are pathological the fertilized ovum perishes early without the pregnancy having become clinically recognizable. In such cases the ovum may undergo decomposition and resorption with damage to the placental tissues. It is not essential in such case that the male shall transmit infection to the female: when the ovum dies, its presence as a dead body invites infection from any and every available source and the dead ovum may thus be said to cause infection.

There is an ancient tradition that interference with the production of ideal young in quantity and quality is referable to the female. Out of that tradition, apparently, arises the word *gynecology* (the diseases of woman). This suggests pathological forces which affect only

the female sex, and in the causation of which the male can have no part. The tradition is not yet dead, nor are there important symptoms that it may perish in the near future. In veterinary medicine *contagious abortion* is yet chiefly a *female complaint* highly infectious from one female to another, but not readily transmitted from one sex to the other. So, with sterility, during recent decades much has been said of the causes which act upon the female and very little concerning those which affect the male. Spectacular propaganda are launched of panaceas for sterility which are to be given to the female—vitamins, minerals, hormones—which are not essential for the male in order that he may produce healthy spermatozoa. As yet we are largely in the dark in many highly important respects, but recent clinical studies have served to throw some important light upon the problems involved.

In 1922, W. W. Williams[265] showed that in a large dairy herd the six leading sires varied widely in their reproductive efficiency. The best sire required an average of 1.58 services per observed conception; the poorest, 3.85 copulations. The recorded abortion rate in the recognized pregnancies of the former was 25.8 per cent; in the latter 66.7 per cent.

In 1923 I[240] reviewed the work of W. W. Williams, adding further data, which appeared to show conclusively that a genitally unsound male may readily transmit to a healthy female with which he copulates disease which may prevent recognizable pregnancy, or recognizable pregnancy may be induced to be followed by abortion or the birth of non-viable young. The metritis associated with abortion may prove mortal, or, after apparent recovery, may serve as a permanent barrier to conception when mated with a healthy male. Table II, taken from the cited contribution, reveals some interesting clinical facts in the breeding history of a definitely diseased bull compared with healthy sires in a herd where all females occupied common paddocks and were in constant and intimate contact day and night. The diseased bull was purchased from a highly fashionable Holstein-Friesian herd, as a calf, and had not been used previously. The table speaks for itself. Upon physical examination I could discover no genital abnormalities among the unfertile females. The seminal vesicles of bull 2 had undergone abscessation and, as is the rule, the abscess had ruptured into the rectum as shown in Fig. 138. The clinical facts are related to show that a bull calf, not previously used, went into a healthy herd, caused sterility, abortion and fatal metritis, and virtually ruined the females which were bred to him. It is immaterial, from the clinical standpoint, whether the

TABLE II

Breeding Records of a Diseased, Compared with Healthy Bull, in a Purebred Herd.

Year	Bull	Number of cows	Numbers of services	Sterile		Aborted		Viable Calves		Fatal Metritis		Non-fatal Metritis		Average Duration of Gestation in Days of Viable Calves
				Number	Per Cent	Number	Per Cent	Number	Per Cent	Number	Per Cent	Number	Per Cent	
1918	1	27	40	1	3.7	0	0	26	96.3	0	0	0	0	285.96
1919 and 1920	1	35	42	4	11.4	1	2.9	30	85.7	0	0	0	0	282.79
	2	34	49	5	14.7	8	23.5	21	61.8	3	8.8	6	17.6	277.00
1921	1*a*	3	3	0	0	0	0	3	100					
	1*b*	15	22	5	33.3	1	6.67	9	60					
	x*b*	9	10	8	88.9	0	0	1	11.1					

1*a* signifies cows bred to Bull 1 which had not been bred previously to Bull 2; 1*b*, cows bred to Bull 1 which had been bred to Bull 2 in 1919 and 1920; x*b*, cows bred to Bull 2 in 1919 and 1920 and bred to two other bulls in 1921.

bull was or was not infected with *Bacillus abortus*. If it were that organism, the losses which followed his use were not due to its invasion by way of the mouth, as is generally claimed. It spread only by sexual contact. Otherwise the animals not bred to him would have suffered equally with those which were. The disaster might have been avoided by a competent examination of the diseased sire prior to purchase.

A second example presented in the same contribution was that of five sires used in a stud during 1920-1921. The mares were said to have been immunized against contagious abortion by the use of bacterins. No record was available showing that any other abortifacient organism was present in the stud. As indicated in Table III, there were few recognized pregnancies to protect against equine infectious abortion. Stallion 1 in the table is interesting. He had been an illustrious sire in the famed Blue Grass region of Kentucky but, having become a sexual wreck, he was presented to the stud with a fanfare of trumpets. His semen contained few spermatozoa, with subnormal motility. Three microscopic fields were used in order to assemble the four defective spermatozoa shown in Fig. 28. No two are alike and no one of the four is physiological. Fig. 29, showing healthy spermotozoa, is inserted for comparison. The stallion was

TABLE III

Breeding Records in a Stud During 1920–1921.

Stallion	Total Mares Served	Total Services	Number Conceptions	Services per Viable Foal	Number Viable Foals	Per Cent Viable Foals	Number Abortions	Per Cent Abortions	Per Cent Barren
1	19	27	3	13.5	2	10.5	1	5.3	84.2
2	19	23	12	1.92	12	63.1	0	0	36.9
3	17	23	7	3.23	6	35.3	1	5.9	58.8
4	12	13	4	3.25	4	33.3	0	0	66.7
5	12	12	2	6.0	2	16.7	0	0	83.3

technically fertile: he produced 3 recognized conceptions, one of which ended in abortion, and a second illustrated the opposite ex-



FIG. 28. Spermatozoa from Stallion 1 in Table III.

A sire of extremely low fertility, whose badly diseased genital system is illustrated in Figs. 121 and 131. Three fields were used in order to assemble four spermatozoa. No two of the cells are alike and no one morphologically normal.

TABLE IV

Breeding Records of Three Hereford Bulls During Ten Years.

	1911		1912		1913		1914		1915		1916	
Bull	Services	Per Cent Calves	Services	Per Cent Calves	Services	Per Cent Calves	Services	Per Cent Calves	Services	Per Cent Calves	Services	Per Cent Calves
1	0	0	4	50	21	48	48	40	77	34	22	16
2	8	100	39	33	82	34	117	32	73	27	106	39
3	23	83	70	54	61	33	37	41	35	40	6	0

	1917		1918		1919		1920		Totals			
Bull	Services	Per Cent Calves	Services	Per Cent Calves	Services	Per Cent Calves	Services	Per Cent Calves	Services	Per Cent Calves	Heifer Calves	Progeny from Heifer Calves
1	16	19	2	0	0	0	0	0	213	32	32	41
2	106	27	86	26	64	28	10	90	691	32	85	163
3	0	0	3	33	2	0	1	0	213	50	44	162

treme by being carried far beyond normal term, and born a worthless darf. Seventeen mares bred to stallion 1 were later bred to presumably fertile sires, which made an average of 13.26 services each for the four viable foals produced.

In 1923 I[240] observed that certain sires, like No. 3 in Table IV, produced a greater number of daughters of high breeding power. At that period such a phenomenon was passed over as a mildly interesting vagary of chance. Further and more extensive studies show that the data are clinically significant, that reproductive power is transmitted by parent to offspring, and that the higher the breeding efficiency the more closely the ratio of females equals that of the males. Since the sire is half the breeding unit, his reproductive health and vigor constitute a highly essential element in pedigree.

In 1925 Williams and Savage[266] submitted the results of the micropathological study of the spermatozoa from 237 bulls, most of which had been used as sires in large purebred herds. Authentic data were

obtained regarding the fertility in 198 cases, of which 34.4 per cent were definitely low in reproductive power. Many pathological changes in the morphology of the sperm cells were described and illustrated, and it was clearly shown that in most instances the fertility of a bull could be authentically determined by a microscopic study of the fixed, stained spermatozoa, even though it was impossible by ordinary clinical examination to detect the presence of disease of the genital organs. The erroneousness of the previously accepted belief



FIG. 29. Healthy Equine Spermatozoa. *(Cornell Veterinarian)*

that the presence of numerous motile spermatozoa in semen indicated satisfactory fertility became equally evident. It remained evident, of course, that freshly ejaculated semen without motile spermatozoa was non-fertile, but the far more important truth became obvious that semen might have many motile spermatozoa and the male not only be of low fertility, but, far worse, be highly dangerous as a transmitter of genital disease, causing abortion, retained afterbirth and other phenomena ending in sterility.

It was further shown by Williams and Savage that, by the more technical microprojection method of accurate measurements of spermatozoa, the fertility of bulls could be authentically determined in

most cases by the variation in the head lengths. This was not put forth as a better method so much as a verification of the simpler one of estimating the variations in head measurements of spermatozoa directly under the microscope.

Numerous investigators had previously reported the existence of dimorphism in spermatozoa, and had reached the conclusion that one size was male and the other female, and that, by separating the two sexes and resorting to artificial insemination, sex could be controlled. Williams and Savage and others have failed to recognize the dimorphism of bovine spermatozoa.

In ideally healthy sires the spermatozoa are strikingly uniform in morphology. Bulls of fairly satisfactory fertility may show 160 to 170 abnormal sperms per 1000, but beyond that ratio the evidence indicates low fertility with high intra-uterine disease. The ratio of abnormal spermatozoa in the presence of fair fertility seems to be somewhat higher for the stallion, and may sometimes reach as high an incidence as 200 per 1000, possibly owing to the vastly greater number of cells in a single ejaculation.

In 1930, W. L. Williams and Savage[250] found that the measurements of the head lengths of equine spermatozoa by microprojection were reliable indices of the fertility of the stallions and that the findings were in harmony with the usual microscopical study of the stained spermatozoa and with the breeding records of the sires.

In 1934 Lagerlöf[114] published the most exhaustive researches in this field which have yet appeared, along with a very extensive bibliography and many excellent illustrations. The studies of Savage, W. W. Williams and myself were fully supported in all essentials and the investigations pushed further to include the histology of the testicles and epididymes. Lagerlöf was able to show that, when the spermatozoa are morphologically abnormal, the epithelium of the tubuli seminiferi shows an endless variety of pathological changes corresponding in degree with the ratio and extent of morphological abnormalities in the sperm cells. The morphological abnormalities are not, therefore, at least in most cases, due to changes occurring after the spermatozoa have left the tubuli seminiferi and entered the epididymis or the vas deferens.

Accordingly, females mated with sires having an excessive ratio of morphologically defective spermatozoa, conceive with difficulty; the resulting pregnancies are largely interrupted by abortion; the duration of gestation is erratic; the new-born are frail and the survivors of sub-normal value; and the reproductive and physical life of the female is placed in peril.

Sometimes the study of the stained spermatozoa has failed to reveal the cause of a high incidence of sterility and abortion when it is otherwise shown that the sire is at fault. A conspicuous failure to trace the prevalence of abortion and sterility to morphological abnormalities of the spermatozoa was reported by Lothe in 1929.[124] His observations were made in a Bang-negative herd in which abortion, sterility and pyometra flourished. Lothe stated that in the case of a certain bull "a considerable number of cows bred to him failed to show estrum following service and were found to be carrying no calves, but instead their uteri contained from one to several quarts of white fluid." Apparently material had been supplied to competent bacteriologists for technical examination. *Bacillus abortus* could not be incriminated. Semen was forwarded to a recognized authority but no abnormalities of the spermatozoa were found which might explain the clinical phenomena.

Not long afterward Abelein[1] submitted, in 1932, convincing evidence that a trichomonad was the cause of much abortion and pyometra in herds where *Bacillus abortus* could not be incriminated. Later many reports appeared announcing the presence of this destructive parasite in numerous locations in the United States. The trichomonad was known to exist in Europe as early as 1900, and was believed by a few to be pathogenic, but the *Bacillus abortus* so monopolized the attention of veterinarians and breeders that other pathogenic forces interfering with reproduction were virtually ignored. The overwhelming evidence submitted by Abelein compelled attention to the trichomonad disease. Looking backward to the contribution by Lothe in 1929 it becomes reasonably clear that trichomonad infection caused the confusion.

It is important that the fact be recognized that serious genital infection may exist and cause great losses without having caused recognizable injuries to, or abnormalities of the spermatozoa at the moment of ejaculation. So far as at present known, with the exception of the trichomonad disease, an intelligent study of the stained spermatozoa definitely establishes the fitness or unfitness of the sire.

Most American states require that purebred cattle must pass a satisfactory test for contagious abortion before they may be sold for breeding purposes in another state, or shall be shown at fairs. This test furnishes no assurance of fertility or freedom from important genital disease. The prudent buyer of valuable horses or cattle for breeding purposes should have a dependable examination made of the animal, which in the male should include an examination of his spermatozoa.

Such examination is to the mutual interest of seller and buyer. With a colleague, I examined the semen from a fashionably bred bull under negotiation for sale into a foreign country. We unhesitatingly pronounced the animal infertile. Despite this opinion the breeder sold the animal, shipping him about 5000 miles, The purchase price was necessarily refunded and the breeder lost prestige.

One of the trying problems in veterinary ethics arises in connection with the public services of famous race horses. Such stallions command service fees as high as $1,000 to $2,500 for the season without assurance of a foal or even pregnancy. The stallion owner sometimes dictates that ere a mare be served she shall pass a satisfactory bacteriological examination by a person of his selection. While this may afford some protection for the stallion, it provides no safety for the mare. In order to make a showing of consistency, the stallion owner may also have the same party examine the semen of his stallion, so that he may assure the owner of the mare of the sexual health of the sire. A client had booked mares to a famous sire at $2,000 each. In behalf of my client I procured semen from the stallion and supplied samples to two highly competent colleagues. Each of the three of us working independently found 600 to 700 defective spermatozoa per 1000 and unhesitatingly advised my client against breeding to the stallion. Meanwhile another veterinarian of high repute in the field of equine genital diseases had examined the semen of the stallion and had submitted a report that the motility of the spermatozoa was "approximately 80 per cent" and that the "stained smears of semen showed considerable numbers of abnormal cells." My report to my client created a furore and the stallion owner forwarded a copy of the report of his veterinarian to show that I was in error. As a matter of fact his veterinarian had not stated that the stallion was either fertile or free from transmissible infection. The veterinarian was under important obligations not to condemn the stallion, and hence submitted a diplomatic report which was noncommittal. Not long afterward the sterility and infectiousness of the stallion became so obvious to all that he was retired from the stud.

No effective method for the control of these perils has yet been devised. Perhaps a satisfactory plan could be adopted by breeders' associations, by which a sire offered for public service might be examined by an expert not dependent upon the owner of a diseased stallion for his living. Somewhat similar, but less urgent conditions apply to bulls belonging to bull associations.

The technic for the examination of semen will be described in conjunction with the discussion of the diseases of the testicles.

Pathological Duration of Gestation

Much has been thought, and far more written, concerning the great peril of abortion. Almost nothing has been said regarding the ultimate effect upon mother or young of those abnormally short, or abnormally long gestations which result in the birth of living young. While discussing twin gestation, it was observed that in a vast majority of cases, abbreviated gestation ensued, followed by an appalling death rate among the young and the parturient females. It inevitably follows that in such a group of subnormal vigor, the surviving members have a low average resistance and are wanting in fertility. Abnormalities in the duration of pregnancy are inseparably linked with abortion, retained afterbirth, dystocia and other interferences with reproduction. The intelligent animal husbandman selects as breeding stock those animals which appear to have been well nourished throughout their lives, but he habitually regards *birth* as the beginning of life. He fails to fully realize that the life of the individual begins with the union of the ovum and spermatozoon and that proper nutrition is just as essential during intra-uterine, as during extra-uterine life.

The physiological duration of gestation in domestic animals has not been directly studied. It has been largely assumed, though neither proven nor alleged, that a gestation period has been normal if the young or mother survives. That is not the end. Prominent writers sometimes say that a cow "calved normally" when the calf was dead and the cow died. The most important variations in the duration of gestation occur in unipara, since in multipara the presence of a healthy fetus tends to prevent the early expulsion of others present which may be diseased or dead; the maturity of a healthy fetus dictates its birth and with it the expulsion of diseased or dead fetuses.

Schmaltz[178] places the typical duration of gestation for the mare at from 310 to 360 days, or a variation of 50 days. He admits, however, that wider variations occur, and cites Tessier as placing the extremes at 287 to 419 days. Most writers are silent regarding the vigor, fertility and longevity of the young born at various durations of pregnancy.

Premature birth, if it is meant thereby that a foal was born in an otherwise healthy state, though immature in development, is virtually unknown clinically. A foal may be born, after an abbreviated gestation, so badly diseased that it may not live, but that is pathological. Upon the other hand, premature birth is common in cattle, and as in the mare is associated intimately with genital disease. Prolonged

gestation, less common in the cow that in the mare, apparently has the same significance. There is no known duration of gestation in the mare or cow, at which living offspring may be born, that the young may not be weak and prove short-lived or infertile. Fortunately some facts are now available which, intelligently applied by the breeder, may aid him in determining the predestined value of a young animal at the time of its birth.

ABBREVIATED GESTATION

Abbreviated gestation is highly prevalent in cattle, especially among the dairy breeds where genital diseases are rampant. In Table II it is clearly shown that the average duration of pregnancy was 5.79 days shorter in those cows bred to Bull 2 than among those of

TABLE V

Breeding Behavior of Two Jersey Bulls in a Herd.

No. Bull	Number of Live Calves Produced	Average Duration of Pregnancy	Number of Services per Calf	Per Cent Abortions
7	30	276	4.1	34.8
3	19	284.5	2.9	13.6

Bull 1. Abortion and metritis were rampant among the cows of Bull 2, and many of the cows which survived were sterile.

Many of the short durations of pregnancy were naturally in heifers, the diseased bull having been purchased chiefly to serve the daughters of Bull 1. First pregnancies in dairy heifers are usually said to be shorter than those of pluripara, but in this case the difference is quite beyond the usual estimate. Moreover, as will be shown later, abbreviated gestation does not occur in heifers which were healthy when born, are maintained in good health during calfhood and are permitted to attain ample maturity prior to breeding. So far as could be determined, the females bred to Bull 2 were healthy until bred. The seminal vesicles of Bull 2, as shown in Fig. 138, were virulently diseased, and so far as yet learned, such disease is uniformly associated with serious genital disease in the females served. A second illustration is submitted in Table V, showing the breeding behavior of two Jersey bulls in an agricultural college herd. The original herd sire, No. 7, was doing badly, while the new sire, No. 3, introduced to take his place, although used upon cows which had previously aborted to No. 7, shows a definitely more physio-

logical duration of gestation and a far lower incidence of abortion. Such examples abound without number.

Individual exceptions occur in which calves, born at 270 days or less, grow well and are highly fertile and efficient. When large numbers are carefully studied, it is found that heifers born at 279-288 are eventually the best breeders and that when gestation falls below 270 days the breeder is on dangerous ground if he retains such an animal for breeding purposes. The fertility and reproductive strength of a herd of cows may be authentically measured by the ratio of individuals carried in utero between 279 and 288 days, to those falling outside this optimum.

Recorded data seem to teach that the duration of pregnancy is greater for the male than for the female young. That can only be alleged for unipara, since twins and multipara are born simultaneously regardless of sex. According to the data I have had opportunity to study, the variation in the duration of pregnancy of the two sexes is largely, if not generally pathological. More males than females are aborted. If these were included in the calculations, the duration of gestation would be longer for females. In the mare prolonged gestation is frequent and the foals carried grossly over time, according to my observations, are males and severely diseased. It is these diseased male foals which largely serve to lead to the belief that gestation is longer for males than for females.

A careful study of twin gestation in the cow tends to throw some light upon the problem of the variations in the duration of pregnancy. Twenty-four twin pregnancies in a large Holstein-Friesian herd have been arranged in Table VI. There were 7 pairs of bulls, BB, 9 mixed pairs of one bull and one heifer or neuter, BH/N, and 8 pairs of heifers, HH. Among the mixed twins was one heifer known to have been fertile. Accordingly there were 23 bulls, 17 heifers and 8 neuters or unrecognized heifers. The viability of heifer twins was 87.5 per cent compared with 56.2 for the bulls and mixed pairs. While these data are too limited to provide a basis for definite conclusion, other clinical observations support the inference that the apparently greater duration of pregnancy for single males, and the higher incidence of abortion and non-viables for them is pathological, not physiological.

PROLONGED GESTATION

Prolonged gestation is most common in the mare, but by no means rare in the cow. The most spectacular prolongations of gestation commonly observed are those cases in the mare in which pregnancy

TABLE VI

The Duration of Gestation and the Mortality of Bovine Twins According to Sex.

		NUMBER	AVERAGE DURATION OF PREGNANCY	EXTREMES OF DURATION	VARIANT IN DAYS
Viable	BB	4	273.75	268–277	9
Non-Viable		3	272.67	252–301	49
Total		7	273.29	252–301	49
Viable	BH/N	5	280.20	275–286	11
Non-Viable		4	262.00	225–288	63
Total		9	272.11	225–288	63
Viable	HH	7	276.71	268–283	15
Non-Viable		1	277.00	277	0
Total		8	276.75	268–283	15

closely approaches, or exceeds 365 days and the foal is frequently born mortally ill from sepsis—the so-called "sleepers."

Von Oettingen[153] in his admirable study observed that "Many foals born in the Trakehnen stud after a period of gestation of but 315 to 320 days have developed into excellent brood mares and sires. On the contrary, according to observations in Trakehnen, the foals with gestation periods of more than 11 months seldom, those of more than 345 days almost never attain distinction in the stud as adults." In 1901 Sohnle[183] made the highly significant statement, "Mothers of very weak foals quite largely go over their time." He thus clearly indicates that, according to his observations, prolonged gestation in the mare was inseparably linked with utero-fetal pathology. This view is in accord with all available records.

In a small stud with unusually accurate breeding records I have made studies[262] upon the fertility of the mares according to the duration of time they were carried in utero. Table VII from these studies shows, as graphically as is possible with so few mares, the vital influence of the duration of pregnancy upon the future fertility and breeding value of the fetus. It shows that the state of nutrition of the fetus within the uterus is of fundamental importance in determining the value of the individual in its post-natal life.

When carefully studied, the few available facts regarding prolonged gestation in cattle indicate that, whenever the female fetus is carried beyond 288 days, there is an ever-increasing peril that, if she survives, she will prove a poor breeder. Wagner[211] supplies a highly illustrative example from the Braunvieh herd of the Hohen-

TABLE VII

The Breeding Efficiency of Mares According to the Duration of Time They Were Carried in Utero as Fetuses.

Duration of Gestation of Mares	Number of Mares	Number of Services	Number of Pregnancies	Number of Viable Foals	Per Cent Viable Foals	Services per Viable Foal	Average Viable Foals Per Mare	Aborts and Non-Viable Foals	Per Cent Aborts and Non-Viable Foals
Mares born at not over 320 days	20	159	58	48	82.8	3.3	3.3	10	17.2
Mares born at 321–339 days	25	152	71	57	80.3	2.7	2.3	14	19.7
Mares carried in utero over 339 days	7	69	21	14	66.7	4.9	2.0	7	33.3

heim agricultural school. Wagner observed that the 13 daughters, 9 granddaughters and 2 great-granddaughters of Bull X were extremely poor breeders, while the associated females of the herd, including the dams of the 13 daughters, were highly fertile. The average duration of gestation for the 27 pregnancies of Bull X was 295 days. The average durations for 8 other bulls in the herd were 285; 289; 289; 289.5; 289.5; 290; 291 and 291.5 respectively. Wagner advises me personally that 9 of the 15 heifer calves by Bull X were carried in utero 292 to 320 days. The duration of gestation is apparently high in Braunvieh (Brown Swiss). Wagner[212] finds that in the Hohenheim herd, among 342 single, and 11 twin births which he classed as of normal duration, the general average was 291 days or 4 days less than the average pregnancies established by Bull X.

The daughters of Bull X have been arranged in Table VIII, with the 9 granddaughters, and 2 great-granddaughters—a total of 24 females, with 69 pregnancies. Of these, 44 are recorded as physiological calvings (C). There were 8 abortions, 2 premature births and 3 dead at birth, a total of 18.8 per cent. Retained afterbirth followed 11.6 per cent of the pregnancies and nymphomania occurred in 8.7 per cent. The family promptly became extinct. Wagner concludes that the sire carried hereditary defects in his reproductive system which rendered his progeny of low fertility.

None of the 24 female descendents of Bull X died from genital disease; sterility is the only cause recorded for discarding them. With

TABLE VIII

Breeding Record of Female Progeny of Bull X (Wagner).

Cow number	Result first breeding period	Non-pregnant interval	Result second breeding period	Non-pregnant interval	Result third breeding period	Non-pregnant interval	Result fourth breeding period	Non-pregnant interval	Result fifth breeding period	Non-pregnant interval	Result sixth breeding period
1	C	118	C	158	C	51	CC	216	S.K		
2	C	321	C	93	C	109	C.R	N.K			
2′	C	162	X	182	S.K						
3	C	286	C	118	C	142	C	105	C	229	S.K
3′	A	275	C	91	C	100	C				
3″	S.K										
4	C	158	C.M	462	C.R.N	112	C	175	C	Sd	
4′	C	120	C	110	C	116	A	Sd			
5	C.M	K									
6	C	187	C	121	C.R	198	D.C	170	C	730	K
7	X	912	S.K								
8	C	?	A.N	K							
9	C	185	D.C	N.K							
10	C	187	C	252	C	87	C	82	C	?	A.K
10′	C.R	172	C.R	K							
10″	C.R	K									
10*	C	?	S.K								
10**	A	?	D.C	K							
11	CC.M	K									
12	C	274	C	220	C.R	124	C.N	326	C.N	K	
12′	A.N	139	C	68	C	S.K					
13	A	229	C	207	C	173	C	103	CC	272	C.R.S
13′	A	362	A	S.K							
13″	C	244	S.K.								

C = Calf; CC = Twins; A = Abortion; X = Premature birth; R = Retained afterbirth; M = Metritis; N = Nymphomania; D = Dead at birth; S = Sterile; K = Killed; ′ or ″ = Daughter; * or ** = granddaughters.

an average duration of gestation in the entire herd of 291 days, it would be necessary for a cow to become pregnant at 74 days after parturition in order to calve again within one year. Table VIII shows that but two pregnancies came within this limit, Nos. 1 and 12. The former bore twins and was then sterile; the latter was sterile after the one early conception. There were six conceptions at less than 100 days post-partum, and of these three became pregnant later once each. The interval between parturition and the next conception is given for 41 animals, with an average of 174 days. The period during which sterile females were retained and efforts made to breed them is recorded in only 6 cases, with an average of 407 days. There were 3 twin pregnancies: 2 cows failed to again become pregnant; the third became pregnant once after twinning, had retained afterbirth and was thereafter sterile.

Professor Udall personally relates that in a large, highly fertile Guernsey herd, under his professional supervision, the pregnancies by a supposedly valuable bull were grossly prolonged. When his heifers came into breeding, their milk yield was subnormal and all were discarded. Fertility, including a normal duration of pregnancy, is fundamentally necessary to satisfactory dairying efficiency.

Prolonged gestation in the mare, so far as known, is commonly associated with subnormal development and weight of fetus, apparently because of interferences with nutrition. In contrast, the bovine fetus grows continuously during prolonged pregnancy and frequently, if not generally, results in fetal giantism with dystocia. This is well illustrated by Andres[5] who records a cow slaughtered at the 369th day of gestation because of irremediable dystocia owing to fetal giantism. The fetus weighed 217.8 pounds, its hair was superabundant and very long, dentition was far advanced beyond the normal of a new-born calf and some of its organs were gigantic for a fetus. Andres cites incomplete reports upon 53 cases of prolonged gestation. All the fetuses were gigantic; several of them had suffered from fetal diarrhea, accompanied by hairballs in the amniotic liquid. Apparently the hairballs were meconial masses consisting chiefly of the superabundant hairs. The sex is mentioned of but few, with a marked predominance of males. The Andres cow had given birth to five bulls and two neuters (free-martins) and the giant fetal cadaver he describes was a male.

DEFECTIVE NUTRITION OF THE FETUS

After a healthy ovum has been fertilized by a sound spermatozoon, it immediately becomes important that the new individual shall have

at its command an ample quantity and quality of nutriment. It has been already pointed out that when the uterus is diseased neither the quantity nor quality of nutrient material can be supplied; it has been equally shown that in cases of twin or multiple pregnancy, one or more young may be deprived of adequate food supply because it is denied sufficient placental area.

I have presented evidence[257] aiming to show that in a large herd of Holstein-Friesian cattle in the tropics, out of doors day and night, and dependent wholly upon grazing throughout the year, the incidence of conception and the vigor of the resulting calves rose and fell in regular cadence with the rainfall, shown in Chart I, which, by controlling the quantity and quality of forage, fixed the state of the nutrition of animals. When drought came and the grass was scant and dry, conception decreased and the resultant calves were of subnormal vigor; when rains were generous and grass abundant, fertility rose and the calves born were of greater value. When food falls below physiological requirements in quantity or quality, whether in intra- or extra-uterine life, disease follows. By disinfection, isolation or immunization, an individual may be protected in a measure against this or that bacillus or virus, but disease in some form still persists. Depriving the fetus of physiological nutrition can not be segregated from the supplying of the fetus with toxic elements. If the maternal uterus is diseased, or if during a prior pregnancy important areas of the placenta have been destroyed or seriously damaged, then inevitably the health of the fertilized ovum within it is in peril. Thus Sohnle[183] cites the breeding histories of 3 mares which evidently had chronic uterine disease:

1. Born 1874; aborted 1878 and 1879; live foal with arthritis 1880, 1881, 1882; aborted 1883; foal with arthritis 1884; aborted 1885. Discarded, no healthy foal.

2. Born 1875; foal with arthritis 1879; barren 1880; healthy foal 1881; healthy foal 1882; foal with arthritis 1883; aborted 1884, 1885. Discarded.

3. Born 1875; aborted 1880, 1881; foal with arthritis 1882; barren 1883; barren 1884; foal with arthritis 1885. Discarded, no healthy foal.

An imported thoroughbred mare under my immediate care foaled in 1925 at 329 days and retained her afterbirth 1.5 hours. Bred 8 days post-partum, failed; bred 22 days post-coitum, failed; bred 25 days post-coitum, conceived. Foaled at 380 days, dystocia. Foal, Fig. 190, small and extremely emaciated. The two extensor pedes muscles of the anterior limbs had parted from their tendons above

the carpus and the free ends of the tendons dropped down in their sheaths as in Fig. 189. The foal could not stand for several days. The flexors pedes of the posterior feet pulled away from their attachments to the pedal bones and the foal stood upon the distal ends of the metatarsals. It lived, but was worthless.



FIG. 30. Pathological Equine Placenta, Following 387 Days Pregnancy.

The placental surface is thickly covered over with dirty-gray necrotic masses, one of which at, A, is more than one inch across. (Cornell Veterinarian)

A thoroughbred mare born in 1911 was barren in 1915; non-viable filly (duration of gestation unknown) 1916; barren 1917; viable filly at 384 days 1918; non-viable filly at 387 days 1919; barren 1920, 1921, 1922; non-viable colt at 387 days 1923; barren 1924; critically diseased colt at 387 days 1925. I personally observed the colt. He was unable to get up for several days and could not stand when helped up. He was semi-comatose and extremely emaciated.

The fetal membranes (Fig. 30) were grossly overweight and the placental surface thickly covered with necrotic masses as great as 5 cm. in diameter. The membranes are more minutely described elsewhere.[245] Despite the atrocious record, the mare and her then apparently vigorous colt sold in a dispersal sale at $5,200, inferentially upon the belief that they were of value for breeding or racing. Viable young born under such environment can not be vigorous and fertile. As indicated also in preceding discussions, diseases of the reproductive system constantly tend toward the production of monsters, as is well illustrated by the observations of Fincher and Williams.[57] in which a thoroughly diseased cow, with a history of repeated sterility, dystocia, abortion, retained placenta and nymphomania, produced in the normal course of pathological events a monster with large gonads having imperfect histological characters of seminiferous tubules and ovisacs, a large uterus with gross multiplication of caruncles and a comparatively normal pelvic urethra and penis.

Wing[271] records that in 1910-1911 there occurred in the Cornell University dairy herd a severe outbreak of metritis. I was the veterinary attendant during this period and realized but partly that the infection was essentially one of saturation, involving most, if not all animals, without causing marked clinical disease in the majority. The bulletin of Wing does not include all breeds, but only Holstein-Friesians. There were born in the herd, chiefly during the prevalence of the metritis, a group of 18 heifers comprising Holstein-Friesians, Jerseys, Guernseys and other dairy breeds. When the heifers had arrived at breeding age they were subjected to an experiment with abortion bacterins, as a test of the alleged power of the bacterins to prevent abortion and allied ills. The behavior of the heifers after the administration of the bacterins is illustrated in Table I. It is impossible to separate accurately the relative effects of the ante- and post-natal factors which operated. My daily contact with the herd before, during and after the puerperal sepsis disaster, which will be more fully discussed under *metritis*, compels me to conclude that intra-uterine disease constituted the primary factor in causing their extremely low fertility. The heifers of the herd had been breeding poorly for several years, but no preceding or succeeding crop of heifers bred so badly.

Chapter IV

THE INFLUENCE OF THE HEALTH OF THE NEW-BORN UPON ITS FERTILITY AS AN ADULT

LITTLE study has yet been given to the problem of the influence of the health of the young animal upon its ultimate reproductive power. Little direct evidence can be drawn from human medicine, to which the veterinary profession so largely turns for fundamental principles. The duration of time between birth and reproduction is so incomparably greater in man than in animals that the correlation is less readily studied. Dairy heifers are commonly bred at 15 months, and dairy bulls are placed in heavy breeding service at a much earlier age. The milk-feeding period for dairy calves usually covers six months, during which time they are notoriously unhealthy. Then follows an interval of not more than nine months, for the recovery of vigor, before the heifer is bred.

In 1916 I submitted data[236] from a dairy herd of 500 cows. The establishment had been having serious and increasing losses from calf mortality. Of the surviving heifers an appalling ratio aborted their first pregnancies, following which many had to be discarded because of sterility. Changes were made in the feeding of the calves, primarily for the purpose of lowering the calf mortality. This having succeeded, it was found that, when the calves fed under the revised plan arrived at breeding age, their fertility was excellent, and the abortion rate in primipara dropped amazingly. Some of the facts are presented in Table IX.

As is inevitable in all herds, other forces were at work to modify the ratio of reproduction. So far as could be determined by clinical observation the betterment was clearly due to the changes in the feeding of the calves. Naturally, also, in harmony with the marked rise of reproductive health, the milking efficiency of first-calf heifers advanced greatly.

I have recorded[257] more extensive data regarding the influence of the health of young calves upon their vigor and fertility as adults in the large Holstein-Friesian herd shown in Table X. All cattle in the herd, except young calves which were being fed milk, were dependent upon grazing. The quantity and quality of forage were controlled by the rainfall. The precipitation, which was extremely variable, was accurately recorded month by month. The foundation

animals consisted of 55 females and 5 males bred in the northern United States, in 8 large herds from Massachusetts to North Dakota. One of the males had been used sparingly, almost if not wholly upon a group of 17 heifers included in the purchases. None of the females had calved; 31 were pregnant and 24 non-pregnant when purchased. Since the assembling of the herd in 1920-1921 no fertile female has been sold or discarded and no females have been added. In 1932 two bulls were added.

The Holstein-Friesian herd is completely encircled by a highly fertile herd of 25,000 Herefords, the two breeds separated only by a

TABLE IX

Changes in the Fertility of Heifers Following Improvements in the Feeding as Young Calves.

Plan of Calf Feeding	Duration of Period in Years	Numbers of Heifers Born	Died from Calf Diseases		Died or Discarded for Other Causes		Conceived	Calved		Aborted		Remaining Pregnant	Under Breeding Age
			No.	%	No.	%	No.	No.	%	No.	%		
Original Plan	3.3	593	184	31.0	239	40.3	170	95	56	75	44	—	—
Revised Plan	4.2	907	203	22.4	77	8.5	382	203	90	22	10	157	245

wire fence, and approximately 5000 Hereford calves are born and raised annually in the paddocks immediately surrounding the dairy herd. Both breeds are kept on ground having essentially the same altitude, have the same forage, rainfall, trade wind, and drinking water. The soil consists of disintegrated lava from the same volcano. It would be difficult, if not impossible, to imagine a more completely identical environment. The outstanding difference in the handling of the two herds is the inherent contrast between the dairying and beef-producing industries. The dairy calves are removed from their dams when about one day old, fed milk manually and bred at 15 months; the Hereford calves take naturally the little milk their dams have to offer and are first bred when two years old. The dairy cow is expected to produce an economically profitable amount of milk through twice as long a period as the nursing time of the Hereford, and during the greater portion of this time to nourish a rapidly-growing fetus. Each of the two herds serves as a control upon the other. The vacillations

in the rainfall from 1920 to 1933 are graphically shown in Chart I. The rainfall for 1934, 1935, and 1936, not included in the chart, has been 14.10, 15.95, and 33.17 inches respectively. In December 1936 the precipitation was 11.37, compared with 11.41 inches for the entire year of 1932, emphasizing the gross inequality and consequent immense variation in the quantity and quality of forage.

In Table X the vacillations in reproduction are shown. A careful study of the chart and table indicates that the conceptions of the

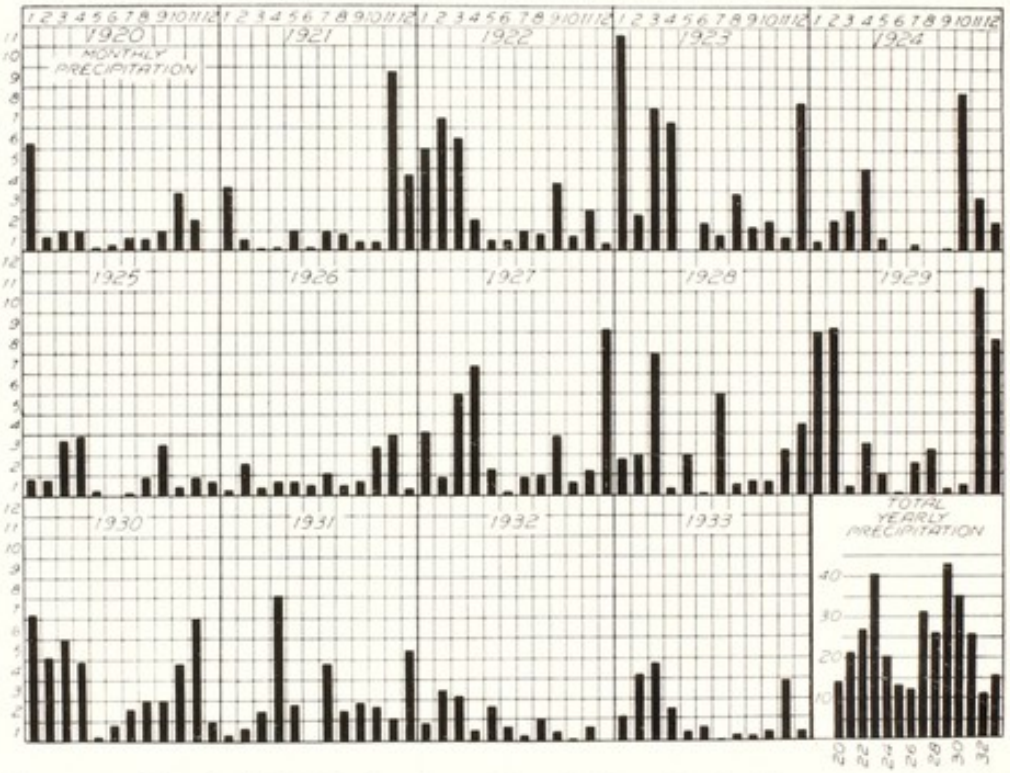


CHART 1. The Rainfall in the Area Occupied by Herd Shown in Table X.

cows, involving presumably the fertility of bulls and cows alike, rose and fell in cadence with the precipitation; within about 30 days subsequent to copious rains, when the grass was lush and the animals had advanced in condition, the rate of conceptions rose. Of even greater importance, it appears that calves carried in utero during good grazing, when mature, were better breeders.

(a) Calves born prior to October 25, 1929, were the product of the plan of breeding in force since the establishment of the herd; (b) those born after October 25 were grown under revised plan; (c) when the calves handled under the revised plan had reached breeding age, the heifers were mated with bulls grown under the same management, and in 1932 two new bulls were introduced for outcrossing. The revised plan for feeding calves will be considered when dealing with the diseases of the new-born.

Apparently it is believed that, if a young animal is born normally and survives, its reproductive system will develop physiologically, regardless of defective health from birth to the commencement of sexual function. Those heifers produced within the herd depicted in Table X, and born from 1920 to 1929 inclusive, showed general genital hypoplasia. The degree of hypoplasia varied. A large ratio of heifers 2.5 to 3.0 years old, and weighing 1000 pounds or over, suffered from severe dystocia. The vulva was so small that the application of traction for the removal of the relatively small calf, in normal presentation and position, occasionally resulted in the com-

TABLE X

Breeding Records of a Holstein-Friesian Herd from 1920 to 1933 Inclusive, Showing with Chart I the Influence of Rainfall upon Fertility.

Group	Year of purchase Groups I and II Year of birth Group III	Number in group	Number in which first breeding period has terminated	Results in first breeding period						Per cent of increase of females during first breeding period	Total completed pregnancies to January 1, 1934						
				Viable bull calves	Viable heifer calves	Non-viable young	Sterile	Died or discarded without viable young	Per cent			Viable bull calves	Viable heifer calves	Non-viable young	Average viable calves per cow	Original cows died or discarded before January 1, 1934	Cows remaining in herd January 1, 1934
I	1920	17	17	7	10	0	0	0	0	58.8	151	79	63	9	8.4	10	7
II	1920–21	38	38	17	10	10	1	3	7.9	15.8	263	109	112	42	5.8	25	13
III	1920	5	5	1	1	3	0	0	0	20.0	25	11	6	8	3.4	5	0
	1921	9	9	3	5	1	0	0	0	55.5	73	34	29	10	7	2	7
	1922	14	14	6	6	1	1	1	7.1	28.6	64	21	32	11	3.8	8	6
	1923	21	21	6	10	4	1	0	0	42.9	80	26	40	14	3.1	11	10
	1924	28	28	5	13	10	0	2	7.1	39.3	99	34	43	22	2.75	11	17
	1925	30	30	6	10	12	2	8	26.7	0	77	25	30	22	1.8	13	17
	1926	41	41	10	10	21	0	11	26.8	−2.4	96	27	33	36	1.5	14	27
	1927	37	37	4	8	20	5	4	10.8	−2.7	67	18	23	26	1.1	11	26
	1928	42	42	12	8	17	5	2	4.8	2.4	56	18	19	19	0.9	7	35
	1929(a)	26	24	4	8	12	?	2	8.3	25.0	31	9	9	13	0.69	2	24
	1929(b)	14	11	4	4	3	?	2	14.3	14.3	12	4	5	3	0.64	2	12
	1930(b)	8	8	3	1	4	0	0	0	12.5	10	3	3	4	0.75	0	8
	1930(c)	19	7	5	2	0	0	0	0	10.5	7	5	2	0	0.37	0	19
	1931	35	6	0	6	0	0	0	0	17.1	6	0	6	0	0.17	0	35

plete rupture of the perineum, followed in some cases by fatal hemorrhage. I personally attended a number of these heifers, and although proceeding with the utmost caution, could not avoid important abrasions and contusions. One heifer of about 1000 pounds was in abortion-dystocia at about the fourth month. I worked for almost an hour to pass my hand into the vagina. I then passed Albrechtsen's cervical forceps alongside my arm and grasped the head of the fetus, after which I found it both difficult and painful to withdraw my hand.

The hypoplasia was not limited to the vulva, but involved vagina, uterus and ovaries. I failed to recognize, by clinical examination

corresponding genital hypoplasia in the bulls, but later studies of their fertility revealed substantial evidence of low reproductive power.

At the outset of this treatise, the position was taken that reproduction is a luxus function, and that whenever the nutritive supply or the energy falls below physiological demands, the function of reproduction suffers, and the demands of physical life take precedence. Since the young animal is born with undeveloped, non-functional sexual organs, before it may breed physiologically it is essential that the development of these shall have become completed. The advent of puberty has been enormously advancd in the chief dairy breeds, so that a heifer, instead of first coming in estrum at two or three years, commences to ovulate at six months or earlier. The herd shown in Table X is not unique in the arrest of genital development. Miller and Graves[139] found that a large ratio of heifers in first pregnancy produced "dead calves" and that "The increase in the percentage of calves born dead can be partly explained by the fact that a number of heifers in the abortion-negative group had genital tracts which were too small to deliver live calves." In other words, the calves of the herd had been so managed that their entire available vigor was demanded for the maintenance of their physical lives, and the due evolution of their reproductive organs was held in abeyance until finally the hour struck and the reproductive system remained permanently hypoplastic.

Veterinary obstetrists describe a type of dystocia in which the fetus is "relatively too large" but fail to define the cause. Physiologically there is harmony between the size of the fetus and the capacity of the female birth canal. Among dairy cattle, however, when a female is badly nourished during her early calfhood, there occurs arrest in the growth of the reproductive system. In such cases, according to my experience, the fetus is actually subnormal in size. Dystocia occurs because the genital passage of the dam is grossly hypoplastic, and the reproductive system has less expulsive power.

Pursuing the problem further, it is perfectly well known to dairymen and clinicians that dairy heifers, in general, conceive tardily, and carry their gestations badly. In the most markedly sterile of these, if a critical examination is made, it will be realized that the arrest in sexual development is not restricted to the vulva, but involves as well the uterus and ovaries. The hypoplasia is most recognizable, next to the vulva, perhaps, in the ovaries, where it will

be found that little ovarian tissue is present and the volume of the organ is dependent to an abnormal degree upon the presence of a corpus luteum or one or more large follicles. Such ovaries can not be logically expected to develop perfect ova. Not infrequently in their uncertain development they discharge simultaneously two fertilizable ova, so that the lower the fertility of the group of heifers the higher the ratio of twins, of twin abortions and of mortality of the twinning heifers. So, in the 18 heifers in Table I, the twin ratio was 6:100. Only one of the three heifers calved again and the calf was a bull. Or, turning to the contribution of Lubbehusen, Fitch and Boyd,[125] it is recorded that 2 primiparae among 27 vaccinated against contagious abortion conceived twins after a total of 11 services, or 5.5 each, while the average for the other 25 first pregnancies was 2.12 services. One of the heifers died in parturition; the other was discarded sterile.

In those dairy herds where the calves are badly handled from birth to the advent of ovulation and spermatogenesis, corresponding roughly to the milk-feeding period, an excessive number of services is usually required for the first recognizable pregnancy, and an excessive ratio of the heifers are absolutely sterile. As a rule, the more frequently a heifer is served by a fertile bull before she recognizably conceives, the more probable it is that she will abort, or suffer from dystocia, retained afterbirth or other type of disease. Doubtless some fertilized ova are too frail to undergo independent development, and perish, unless, as in twin gestation, the imperfect embryo attains vascular communication with a potent host. In a group of heifers of low fertility, some conceive at the first service, not always because they are more fertile than their associates, but more probably owing to the fortuitous discharge of a comparatively normal ovum at the time of service. The condition resembles somewhat the status of a cow which has lost the uterine caruncles from one cornu following retained afterbirth: if bred when ovulating from the ovary on the side where the caruncles have been destroyed, she almost certainly fails; if bred when ovulating upon that side where the caruncles are more or less healthy, she may conceive.

The reproductive life of the heifer having genital hypoplasia is erratic and brief. In a large group, such as illustrated in Table X, some individuals are endowed with sufficient energy, in part inherited, to meet the strain and do well, but in the long run the herd in which calves are indifferently or badly reared must eventually provide for its continuity through the introduction of vigorous replacements.

The dairyman may employ eminent sterility experts, illustrious geneticists, noted food chemists; he may quarantine; he may disinfect his cow externally and internally; he may feed vitamins, hormones and essential minerals to, or beyond the limits of his purse; he may use abortion vaccines, bacterins and nostrums without number: but the heifer which has suffered important arrests in the development of her genital organs during the first year of her life can not become a successful breeder.

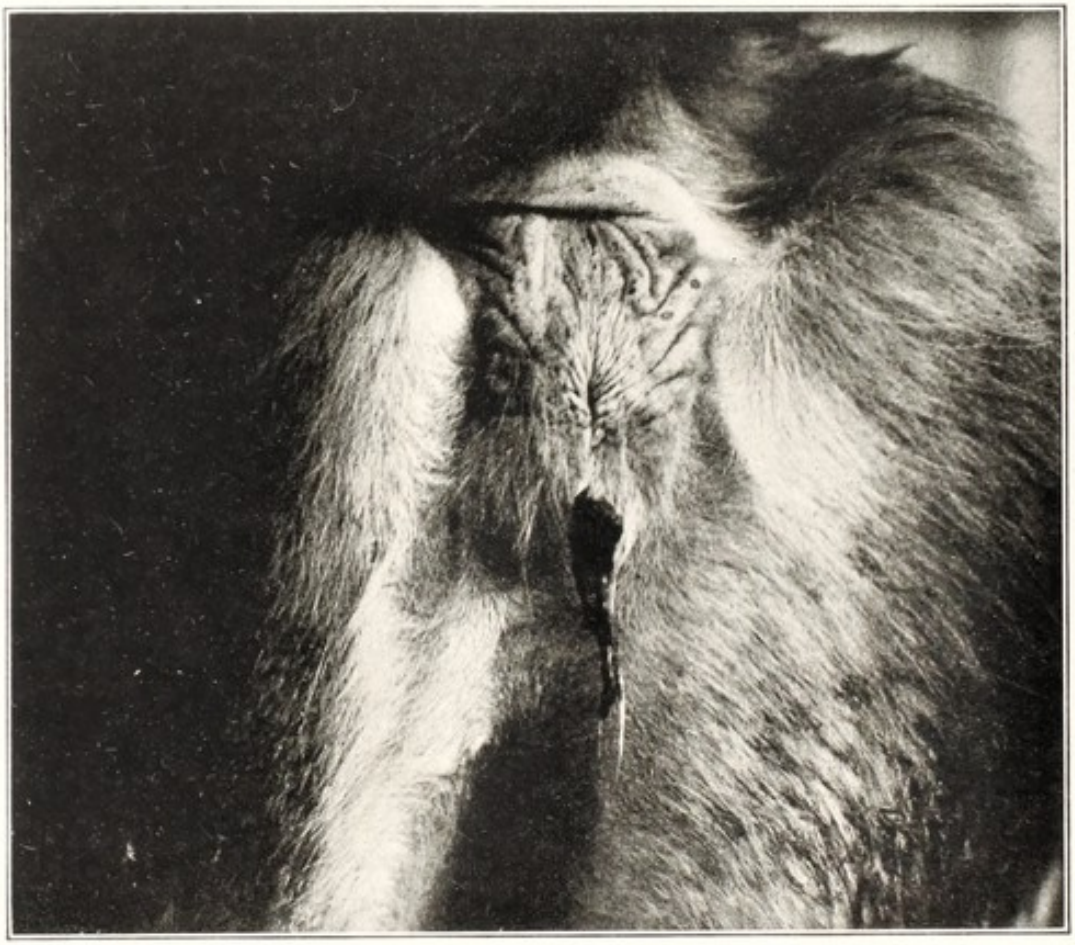


FIG. 31. Soiled Vulvar Tuft of Heifer Calf Imperfectly Fed.
The hairs of the vulvar tuft are firmly matted together in a black mass of pus.

While dairy heifers offer the most vivid illustrations of the principles involved, the rule, so far as known, is universally applicable. When making important changes in the methods of handling the large Holstein-Friesian herd shown in Table X, it appeared prudent to continue temporarily the use of bulls grown with the heifers which suffered from genital hypoplasia. Ultimately it was found highly advantageous to discard the badly grown males and rely upon bulls grown under the revised plan, supplemented by importations. Lagerlöf[114] in his distinguished and extensive researches found

numerous young bulls with distinct testicular hypoplasia, recognizable clearly both by the study of the stained spermatozoa and by histologic examination of the testes themselves. Lagerlöf attributes the hypoplasia to heredity. The line of demarcation between heredity and environment is difficult to locate accurately. The hypoplasia of the genitalia in heifers undeniably falls within each of the two categories. The arrests of the Muellerian ducts in the group of heifers described by Fincher and Williams[53] were certainly hereditary, and



FIG. 32. Heifer with Clean Vulvar Tuft, having been so fed that no appreciable Amount of Muco-pus was discharged from the Vagina.

the hypoplasia in the large herd described by me[257] was apparently due to environment. There is no known reason why both factors may not act upon the same individual.

Health and disease glide so imperceptibly one into the other that no universally acceptable definition is possible. My meaning is perhaps best expressed by the photographs shown in Figs. 31-35. Ordinarily the dairy heifer calf, when a few weeks old, has a black, dried mass of muco-pus upon the tuft of hairs at the inferior commissure of the vulva, as shown in Fig. 31, unless the dairyman,

especially the breeder of pedigreed stock for sale, maintains appearances by washing; a calf in the state of health indicated in Fig. 32, although not washed or otherwise artificially cleansed, does not have such accumulation of dried pus. So, in the male calf, a very ordinary type is shown in Fig. 33 in which the hairs about the prepuce are



FIG. 33. Soiled Preputial Tuft of Bull Calf.

It will be noted that the long tuft of hairs surrounding the preputial opening are firmly matted together and stained a deep black. Under the ordinary method of feeding dairy calves such staining and matting is evident at ten to twenty days after birth. The general coat is notably rough and shaggy. The matting and staining constitute a valuable index of the health of the calf.

matted together as in the heifer and have a black, dirty appearance regardless of the basic color of the hair; if originally white it would be just as black as in the photograph. In sharp contrast, the preputial tuft of hairs shown in Fig. 34 is clean and free from stain. Note that in Fig. 33 the coat is rough and dull and appears dirty. If the rump were shown it would almost certainly reveal smears of feces. In an unfortunate majority of dairies the calves are so fed that feces adhere to the tail and buttocks; in the typically healthy calf



FIG. 34. A Bull Calf so fed that the Preputial Tuft of Hairs remains Clean and Free from Stain.

this does not occur. The adherence of feces to the tail of the calf is proof of digestive and nutritive derangement of importance. Most



FIG. 35. A Group of Healthy Calves in the Herd Illustrated in Table X and fed as related in Chapter XXXIX.

dairy calves are definitely pot-bellied, but when properly fed their bodies are cylindrical, like that shown in Fig. 35.

Chapter V

THE BREEDING SEASON

THE statement has already been made that reproduction is a luxus function occurring only when the parent animals possess reserves of energy beyond the needs of the individual for the maintenance of growth and physical life. In harmony with this principle, domestic herbivora naturally produce young at that season of the year when food is most plentiful. Accordingly, when domestic herbivora are maintained under pastoral conditions, in the temperate zone, reproduction occurs chiefly during spring and early summer, when grass is most abundant and lush. Ordinary cattle of the common type, having abundant food during spring and early summer, but with very limited forage of poor quality during autumn and winter, do not tend to mate during the season of want, but await the nutritive reserves of good pasturage, and the cows eventually calve in spring. This is doubly advantageous: the cow has abundant reserves for the burden of parturition and for the secretion of milk for the new-born; and the calf, when a few weeks old, may gradually accustom itself to feeding upon tender, readily digestible grass.

Domestication has changed this. With artificial foods and housing the more highly bred animals have largely lost their seasonal limitations and may breed throughout the year. The fundamental nature of the animal has not been completely changed, and fertility remains higher during the season of best pasturage. Various objectives frequently lead animal husbandmen to attempt to displace these basic principles and to cause the young animals to be born at other seasons of the year. This is most notably true of the dairyman engaged in the production of fresh milk for table use. Generally, under natural conditions, milk production is highest during the season of richest pasturage, the market becomes oversupplied and the price falls. When winter comes, the milk supply drops and the price advances. Hence for seemingly economic reasons, the dairyman producing fresh milk often prefers to have his cows calve in autumn and thus assure him of a heavy yield of milk during winter. For this transgression of a fundamental law of reproduction the dairyman must pay an appropriate penalty, the precise extent of which has not been authentically determined. In the United States also the agricultural colleges

desire the heaviest flow of milk during the fall and winter because it is then most desirable for purposes of teaching.

It is commonly admitted by all concerned that the fall and winter calving has disadvantages which detract somewhat from the desirability of the plan. No extended study of all the facts concerned is found recorded. It is admitted that abortion, retained afterbirth, metritis, calf scours, calf pneumonia, etc., are most common and costly during winter and early spring. How nearly these disadvantages offset the merits of the plan is not clearly known.

One disadvantage has not been sufficiently studied to acquire definite recognition. A dairy calf born, let us say, on October 1 is six months old on April 1. At that age the heifer is in heat and ovulates. She has been allowed to remain with her dam for perhaps twenty-four hours, and permitted to take an abnormal quantity of milk in a somewhat normal fashion. She is then fed milk in an artificial manner and volume for an uncertain period. Later she is allowed hay, grain or other processed or preserved food. Succulent grass, which naturally constitutes the first herbaceous food of new-born herbivora, is not available. It is during this period that the genital hypoplasia, as mentioned in the preceding chapter, becomes established, and the animal may be irreparably of low fertility, or infertile. Fortunately this peril may be largely overcome by thoughtful care of the calf.

The dairyman engaged in the sale of fresh milk to an established clientele whose needs are alike throughout the year, naturally desires a uniform production. This may be economically attained by selecting cows for winter calving whose progeny are not desired as breeding stock. If the dairyman considers it necessary to have cows whose progeny it is desired to retain as breeders, freshen in winter, the peril may be largely ameliorated by extra post-partum rest. This assures a more favorable duration of pregnancy, greater security against abortion, dystocia, retained afterbirth, etc., with a stronger, more vigorous calf, which by careful handling may grow into a satisfactory breeder. One of the great dangers is that the dairyman will go to the other extreme and in order to have a cow freshen in autumn or winter, breed her after too brief a rest from her prior pregnancy, with such results as those shown in Tables XI and XII. Accordingly, so far as available data lead, it is highly desirable that cows whose progeny it is wished to retain as basic animals in the herd, should preferably be bred to calve at the natural season of the year. This is equally important, so far as the literature indicates, for other species of herbivora and for swine.

For economic reasons some sheep breeders desire early lambs for the market. This out-of-season breeding of ewes in the temperate zone, associated with imprudent feeding and close confinement in late pregnancy during winter months, seems clearly responsible for serious mortality of both ewes and lambs from *pregnancy disease* and *abortion* as recorded by Roderick and Harshfield,[173] Bruce[20] and other writers. The reproductive efficiency of sheep which have been born out of season is not found recorded. It is fairly safe to conclude that their reproductive strength falls below that of their progenitors.

Chapter VI

THE AGE AT WHICH YOUNG ANIMALS SHOULD FIRST BE BRED

MOST countries which enjoy advanced civilization, have statutory laws forbidding marriage until the contracting parties shall have attained approximate physical maturity. This state of development is ordinarily not reached until some four or five years subsequent to the appearance of puberty with technical possibility of parenthood. When such statutory laws are broken, and a girl becomes pregnant, there is great danger of abortion and other disasters.

The age at which an animal may become capable of physiological reproduction varies according to species and is modified by domestication. The reproductive systems of the wild ancestors of domestic herbivora have been so evolved, in harmony with environment, that the available nutritive supply is wholly devoted to the growth of the animal and its physical security until it has essentially reached its adult size and power of endurance. The nutritive energy formerly required for growth may then be diverted, without serious detriment, to reproduction. When domestic herbivora are permitted to run wild they promptly resume their primitive characteristics. The modifications are most obvious in cattle. Heifers of common stock, running free upon the open range, do not ordinarily ovulate as yearlings and may safely be permitted to run with the herd without material danger of conception. As a rule they will first be in heat and accept the bull at about two years of age and first calve at three. Thus du Toit and Bisschop[46] relate that Africander cattle upon the usual South African veld commonly first calve at three years, while upon a poorer veld in Nigeria the native cattle do not ordinarily calve until about six years old, after which age they are fairly fertile. The scarcity of forage merely delays the growth and the advent of reproductive power.

The advent of ovulation and spermatogenesis in horses and cattle has been profoundly modified by domestication, through selection, feeding and housing. Instead of ovulation beginning at 24 to 27 months, as it does in wild, or semi-wild heifers, in highly bred dairy heifers, estrum appears at 6 to 8 months, when they may not yet have attained one-third their expected size. Hence the dairy heifer,

if opportunity be offered, conceives long before she is able physiologically to produce young. Among improved breeds of horses, most fillies are in estrum and readily conceive as yearlings, but those I have observed have quite uniformly aborted. Dairy heifers conceive readily, according to my observation, when six to eight months old, but they largely abort. Those which do not abort almost always calve pathologically after an abbreviated gestation, the heifer is permanently injured or ruined, and the calf is of inferior size and value.

It needs be noted here that the duration of pregnancy is importantly modified by the vigor of either or both parents, in which age plays an important role; and that the reproductive value of an individual is definitely influenced by the duration of its intra-uterine life. The more the problem is studied, the clearer it becomes that there is for each species an optimum duration of gestation, and that any departure from this favorable period lowers the reproductive power according to the degree of variation from the physiological length of intra-uterine life. The law appears to be that the reproductive and general vigor of the individual is profoundly affected throughout its post-natal life by its health and strength during its life within the uterus of its mother. However much later environment may affect its post-natal life, its intra-uterine health constitutes its primary foundation for a lifetime of strength or weakness.

So far as available data indicate, the dangers are essentially alike in their ultimate effect, whether the gestation is abbreviated or prolonged. There is extraordinary divergence in the relative frequency of prolonged and abbreviated pregnancies in mares and cows: in the former, prolonged gestation is prevalent and abbreviated pregnancy is rare; in the latter, abbreviated pregnancy is very common, especially in dairy cows, while prolonged gestation is rare. The reason for this peculiarity is unknown; it possibly is somewhat dependent upon the plan of placentation. In harmony with these specific tendencies it is found that primiparous mares usually carry their young overtime, while heifers constantly tend to abbreviated gestation, especially in dairy heifers, which are commonly bred about one year earlier than are those of the beef breeds. If, however, the minimum breeding age of dairy heifers be advanced from 15 to 24 months, page 114, the duration of gestation may advance an average of 7 days and be essentially the same as for mature, healthy cows.

Hence firstlings are commonly of comparatively low fertility, but when the first breeding of the primipara is properly coordinated with her age, development and vigor, her duration of pregnancy

and the reproductive quality of her offspring shift from pathological to physiological.

In harmony with the above the average duration of pregnancy for the mare, as recorded by most writers, is advanced beyond the optimum duration by the inclusion of foals born alive after prolonged gestation, upon the inference that because they were expelled alive their duration of pregnancy had been *normal*, and therefore inferentially *physiological*. Such inference is open to serious criticism. Thus for example, while Fleming[62] gives the average gestation period for colts as 336.5 days, von Oettingen[153a] states that in the famous Trakehnen stud there were born between 1896 and 1904 16 colts which proved to be outstanding sires, and included 4 firstlings with an average gestation period of 333 days. Hence while colts are said to generally have longer gestation periods than fillies, these valuable stallions showed a duration of pregnancy significantly below the average. Likewise a heifer, born of healthy, vigorous parents, prudently handled, held out of breeding until well matured and mated with a bull of similar quality, will have a physiological duration of pregnancy and bear prolific progeny of high quality.

It is of great importance to the animal husbandman, especially to the dairyman, that his animals should begin reproduction at the earliest date consistent with the maximum health, longevity and efficiency. The efficiency of dairy cows is variously computed. Breeders' associations quite generally compute efficiency upon the amount of milk and butter-fat produced by an individual in a week, month or year, and, exceptionally, upon the lifetime production of unusual individuals. The most valuable standard for computation would appear to be the lifetime production of young, measured by both number and quality of calves and the amount of milk and butter-fat per unit of time and cost of sustenance. For example, if we turn to the excellent bulletin by Professor Wing,[271] Glista Ernestine lived 16 years and 28 days. She bore 13 calves, 12 of which grew to adult life. She thus showed one pregnancy for each 451 days of life. She produced 202,006 pounds of milk, or 34.42 pounds for each day of her life, from birth to death. She produced 7342 pounds of butter-fat, or 1.25 pounds per day for her entire life.

Her 7 heifers, all high producers according to the common method of computation, lived a total, in so far as may be learned from the bulletin, of 43 years, with one cow living at the date of publication. During this 43 years there were 29 pregnancies, or one pregnancy for each 541 days, a decline of 20 per cent from that of Glista Ernestine. The average daily milk production for life was approxi-

mately 24 pounds, a drop of almost 33 per cent below the record of the dam. The annual milk yield for two of the cows was greater than that of Glista Ernestine herself. Since they lived for shorter periods, the daily average for life drops far below that of the dam.

Dairymen largely breed their heifers at about 15 months of age so that they may be in milk at two years. There is ample room for conflict of opinion regarding the best age for the commencement of breeding dairy heifers. The subject has not been fully studied. It is well known that abortion in dairy cattle is by far most common and destructive in primipara. In data I have elsewhere recorded[232] it is shown that, in a large herd, during a series of years the ratio of abortion in heifers was 50 per cent, and that the heifer abortions accounted for 46.5 per cent of the total. Other forces besides the early breeding of heifers were operating, and the precise value of each can not be authentically determined. The health of the heifer as a young calf doubtless played an important part. The evidence seems convincing, however, that early breeding inflicts a dangerous burden upon the heifer and is directly responsible for many pathological terminations of pregnancy, comprising abortion, still birth, retained afterbirth, etc. Few authentic data are available which follow the animals under study through a sufficiently long period to become actually informative. Most dairy cows are bred young, and a vast majority of them die or are discarded young from genital disease. A few possess unusual strength, live to old age and produce many calves. Glista Ernestine, described by Wing,[271] was a great cow. She calved first at less than two years, but there is no definite proof that she was great because of early breeding.

The 17 cows constituting Group I in Table X produced 145 viable calves, counting one pair of twins as a single. This implies 145 full lactation periods. The cows lived a total of 230 years, which signifies one full lactation period for each 1.58 years. Fifteen of the group calved before reaching three years of age and averaged 8.06 viable calves each. They supplied all abortions and non-viable calves (9) and the one pair of twins (bulls) for the entire group. Two were bred to calve first after they had reached three years. They have produced 24 viable calves, and no aborts, non-viables or twins. They accordingly averaged 50 per cent more viable calves, and hence 50 per cent more physiological lactation periods. Admittedly these data are entirely too limited to be acceptable as a foundation for definite conclusion. They are in accord with the biologic law that mammalian reproduction is a luxus function which is best performed by mature animals having the maximum of vigor.

It was not blind chance that caused the two most mature and vigorous of the 17 heifers to produce one-half more viable calves per capita than their companions: the phenomenon is in harmony with the laws of animal life.

This law regarding the age at which heifers should begin breeding is frequently seen from a distorted angle. In Table I, the oldest heifer among the 18 died without calving, or even aborting. I remember her vividly. The unvarying practice of the department was to breed to calve first at two years or less. She had evidently been bred unsuccessfully her first year. If she conceived at fifteen months of age or soon thereafter, the embryo or embryos had perished unseen. The pregnancy recorded in Table I quite certainly followed repeated services to a genitally diseased heifer. She was a quite large animal and appeared rugged. Apparently well one evening, when I saw her the next morning the fetus was enormously emphysematous, the uterus gangrenous and the animal moribund from acute puerperal sepsis. She died, not because first bred at too late a date in life, but because her reproductive system had been wrecked long before, almost certainly during her milk-feeding period as a young calf. Heifers may conceive at any age after six months. It is not wholly a question of conception. A more important question is the ability of the heifer to successfully carry the load placed upon her. Thus in Table XI, seven of the twenty-four heifers were bred prior to two years of age, of which 57.1 per cent failed to conceive at the first service; of the other seventeen, not bred until two years old or over, 17.7 per cent failed at the first service. Then follows the highly significant fact, that those heifers which conceived at first service produced on the average 65 per cent more viable calves than did those which failed in the first breeding. It is well known that dairy heifers, bred to calve first at two years, in addition to a high rate of abortion, retained afterbirth and other pathological phenomena, have a short duration of pregnancy, and the calves are subnormal in size and vigor. Some observant dairymen, while adhering to the custom of breeding at fifteen months, to calve at two years, regularly discard these small, inferior firstlings because, in their experience, these poorly developed calves fail later as breeders. In the large Holstein-Friesian herd shown in Table X, the plan for the first ten years was to breed heifers to calve first at about two years. The calving age was then changed to a minimum of 33 months. The average duration of gestation increased seven days under the latter plan. Other factors came into play, but the evidence seems clear that the greater size and maturity of the heifers played

an important part. My personal observations and all available data seem to indicate that a dairy cow lives longer, produces more and better calves, and therefore yields more lactation periods per unit of time, if bred to calve first at about three years. In the Wing[271] herd, for example, with 3.35 pregnancy terminations per cow, 30 per cent of the reproductive life of each cow was exhausted for each lactation period. Too many of the heifers, like several of those in Table I, were maintained for two or more years only to die without having lactated.

Few data are available regarding the effect of breeding beef heifers to calve early. McCampbell[127] included 80 Hereford heifers in four lots of 20 each. Two lots were bred to calve first at 2 years; the others at 3 years. In one of the lots bred to calve at 2 years, and in one bred to calve first at 3 years, grain was fed in winter, in order to simulate the feeding of dairy heifers. The growth of the heifers bred to calve at 2 years was permanently arrested. Their calves, including three annual crops, were definitely smaller than the young of those bred to calve first at 3 years. Later the superintendent of the station reported personally that it had been found necessary to discard all the heifers bred to calve at 2 years, because of their want of vigor, the prevalence of abortion and other breeding troubles, and the inferior size and quality of the calves. Not only were the original experiment heifers discarded as valueless, but their heifer calves proved distinctly inferior and were also sold.

In contrast, a breeder of high-class Herefords for sale as breeding animals experiences no material losses from abortion or allied disasters in his primipara bred to calve first at three years, but suffers his greatest losses in the second breeding year. That is, the three-year-old heifers have the requisite vigor to carry their first pregnancy safely, but it exhausts their reserves and renders them incapable of immediately resuming the burden of gestation. The same phenomenon is sometimes observed in dairy herds.

Pregnancy is notably unsafe in those heifers which, having reached the desired age for breeding, be it 15 or 27 months, fail to conceive until after repeated matings with a fertile sire. Observation further shows emphatically that the maximum reproductive efficiency of a heifer is definitely fixed by the outcome of her first gestation. So in Table X it is clearly shown that the lifetime breeding efficiency of each group of heifers is authentically forecast by the behavior of the group in the first breeding year. In the halcyon days when "contagious abortion" could be eliminated at will by immunization, its propagandists averred that "it is a well known fact that after a

heifer has aborted she is therefore permanently immune." It has required several decades of clinical observation to make it clear that, once a heifer or cow aborts, she is never again as good as she appeared before and that in a large proportion of cases the reproductive life of the animal is ruined.

Adequate data are not available concerning the age at which a young male may safely be used for breeding. He may be used for an occasional service at an earlier period than that at which the female should be permitted to assume the load of pregnancy. It is not prudent, however, to use him heavily. A large ratio of bulls are ruined while young, especially when used in *pasture breeding*, and their fertility is often destroyed in one or two breeding seasons. The loss of the fertility of the individual male is not so important as the defective vigor and efficiency of his get. A very limited number of services with fertile females may serve as a test of fertility and provide semen for a study of his stained spermatozoa. It should upon no account be forgotten that the best sire is the mature male whose vigor and fertility have been effectively conserved in his earlier years.

Some eminent dairy husbandmen have adocated the heavy use of the young dairy bull in the belief that by so doing he may early be classed as a *proven sire*, although the proving ordeal may ruin the animal. The available data indicate that the heavy breeding of young males is not usually conducive to advancement in the quality of the progeny. Some outstanding individuals may carry the load successfully, but the rule is otherwise. A study of Table IV reveals interesting facts in this connection.

Chapter VII

THE BREEDING OF OLD ANIMALS

IT IS impossible satisfactorily to define old age in its relation to reproductive power in female domestic animals. The menopause of woman, a cessation of ovulation at a fairly uniform age, after which she may enjoy a healthy, vigorous life for 30, 40 or more years, has no counterpart in domestic animals. The breeding life of the latter may cease: (1) owing to physical decrepitude at any age, whether due to the invasion of disease or to defective nutrition; (2) when the reproductive organs become worn out or destroyed by disease. One of two paired genital organs, such as the testicles and ovaries, may be destroyed and reproduction remain possible. As a rule however, when one gonad or other paired genital organ becomes diseased, the other participates.

Reproductive exhaustion in domestic animals is most abundant and economically important in dairy cattle. The Dominion of Canada and the United States of America, with their freedom from the major bovine plagues except tuberculosis, now well on the road to elimination, provide an unsurpassed opportunity for the study of the ravages of genital diseases undisturbed by the prevalence of epizootics which markedly repress reproduction.

The mammary glands may become damaged or destroyed without barring conception and calving, but generally the mammae and placentae are intimately correlated: the placentae supply nutriment to the intra-uterine young; the mammae to the extra-uterine. Logically the diseases of the ovaries and uterus and those of the mammary glands should run somewhat parallel. *Bacillus abortus* is virtually as common in the mammary gland as in the placenta. To what extent other infections occur in both organs is unknown. It is believed that *Bacillus abortus* migrates from the uterus to the mammae. I have observed that, in post-partum uterine hemorrhage in the cow without traumatism, the mammary gland constitutes the chief avenue of exit for the coloring matter of the blood. It appears possible that various infections within the uterus may find their way to the udder. Aside from such possibility, serious disease of the uterus or ovaries interferes with lactation, not alone because the utero-ovarian disease interferes with conception, but also because the milk yield is lowered whenever the gestation is pathological in duration and termination. So it occurs that probably 95 per cent of



FIG. 36. Healthy Allantochorion of Cow.
1, The chorionic navel; 2, the cervix; 3, the non-gravid cornu.

dairy cows die or are discarded because of diseases of the reproduction system before they reach 12 years of age, although a thoroughly healthy cow continues to breed regularly until 20 to 25 years of age. There is accordingly no recognizable age of senility or cessation of reproduction, but breeding ceases because of systemic disease or poverty or of diseases of the reproductive system.

In 1924, my colleagues and I[243] contributed the results of extensive researches, principally concentrated upon a detailed study of fetal membranes from cows, the breeding histories of which were generally available. It was authentically shown that the placental surface of the afterbirth constitutes a replica of the caruncles and endometrium. Essentially no afterbirths were obtained in which

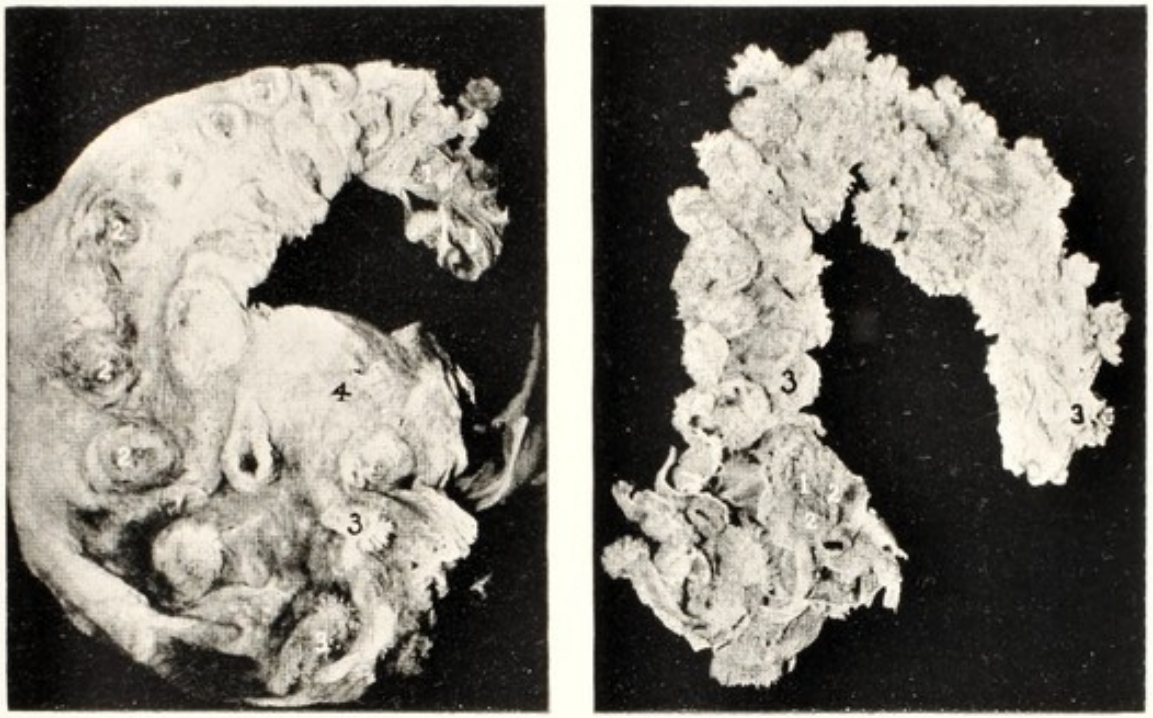


FIG. 37, left. The Diseased Allantochorion from the Gravid Cornu of a Cow aborting at 234 Days and the Afterbirth manually removed after 24 Hours. 1, 1, 1, Apical cotyledons in advanced necrosis; 2, 2, 2, cotyledons showing peripheral necrosis with the central tufts matted with blood coagula; 3, 3, cotyledons near the base of the cornu with some near-normal chorionic tufts; 4, transparent, punctiform areas in placenta; right. The Non-gravid Cornu of Allantochorion, with Some Portions of the Base of the Gravid Horn. 1, Primary cotyledon from the base of the gravid horn, matted with old blood coagula; 2, 2, secondary or adventitious placental growths matted with blood coagula. *From 3, near the base of the cornu to the apex, the cotyledons are almost normal. The focus of infection had been at the apex of the gravid horn, 1, 1, and had travelled toward its base, causing abortion when it had neared the cervix. The chorion of the gravid cornu is enormously thickened. It could supply almost no food to the fetus, which forced it to rely upon the non-gravid cornu for sustenance. Bacillus abortus obtained from fetus. (Cornell Veterinarian)*

careful, detailed study failed to reveal necrotic, or other pathological changes. Minor changes exerted no visible influence upon the fertility of the cow, but as the volume and intensity of the changes increased, definite interferences with reproduction appeared.

Fig. 36 shows the placental side of the afterbirth of a young cow of excellent breeding history. The placenta is not ideal. There are present in the non-gravid cornu, at 5, some adventitious placental



FIG. 38. Diseased Allantochorion of Cow in which the Endometrium had been wholly destroyed in One Horn during a Preceding Pregnancy, and most of the Uterine Caruncles of the Remaining Horn had disappeared.

1, Ovarian pole of the gravid cornu; 2, cervical pole of the pregnant cornu, where the uterine caruncles and endometrium had been previously destroyed, the chorion being thin and transparent; 3, the non-gravid uterine cornu, which had lost its endometrium completely and into which no fetal membranes protruded. *(Cornell Veterinarian)*

growths, testifying that definite injury had occurred at some prior date, not sufficient to interfere visibly with reproduction.

Fig. 37 shows the afterbirth of a cow born in 1918, calved in 1920, 1921 and 1922. Sterile in 1923, she was repeatedly bred, and aborted at 237 days in 1924. The gravid cornu of the chorion was grossly thickened, and its cotyledons destroyed. The fetus had obtained its insufficient nutriment from the non-gravid horn.

Fig. 38 shows the membranes from an abattoir cow estimated at

150 to 180 days pregnant. In a prior gestation the non-gravid cornu had lost its entire endometrium and the horn pregnant at time of slaughter had lost most of its caruncles. Conception was virtually excluded if bred when ovulating upon the side of the naked, non-gravid cornu.

Fig. 39 illustrates 3 cotyledons from an afterbirth. The placental tufts are normal in 1; the uterine caruncle of 2 had lost almost all of

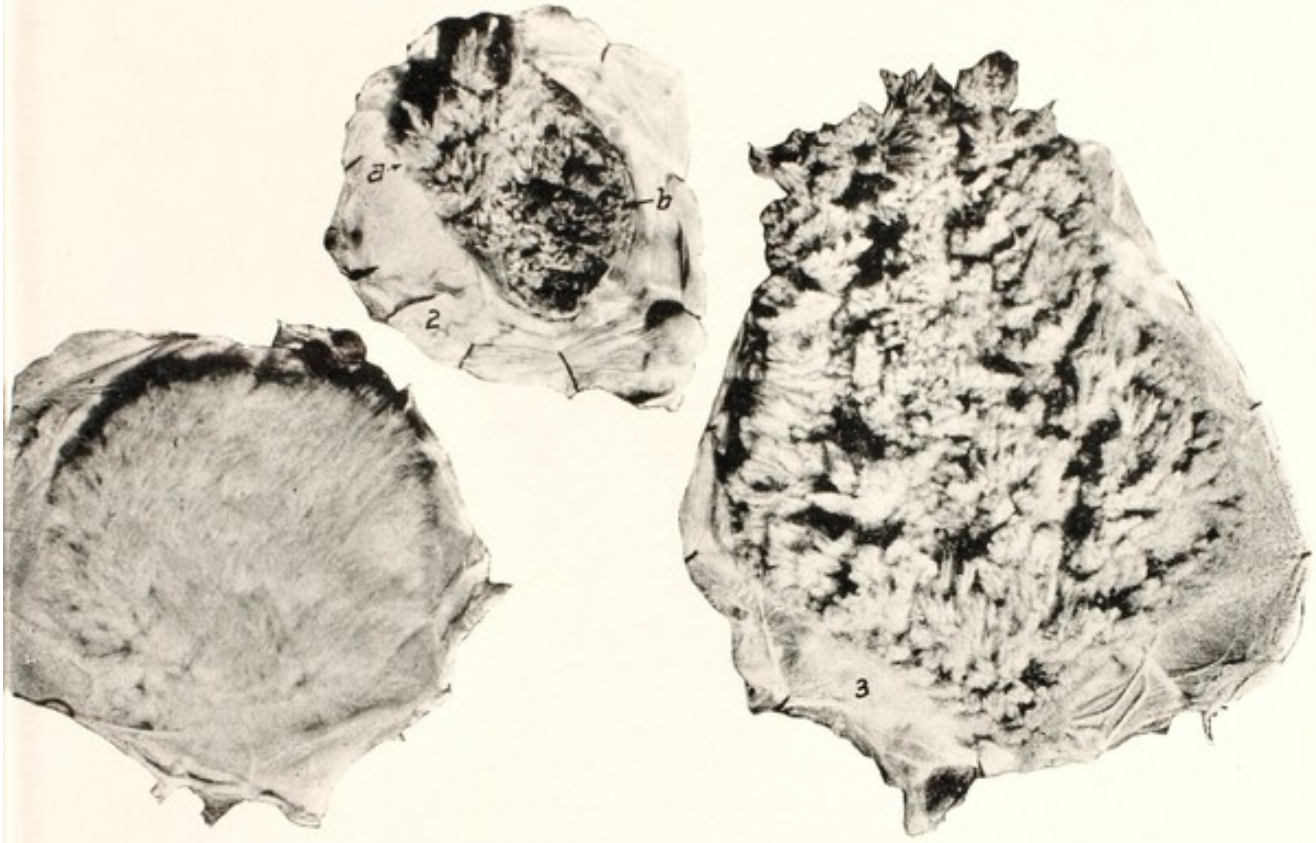


FIG. 39. Healthy and Diseased Bovine Cotyledons.

1, A typically physiological cotyledon from a healthy cow. The chorionic villi are very fine and closely packed; 2, a cotyledon from which all placental villi have been destroyed by necrosis, *a*, a few, coarse, imperfect tufts, *b*, an extensive area of complete necrosis; 3, a gigantic cotyledon with extremely clumpy tufts with large naked areas between the clumps. *(Cornell Veterinarian)*

its endometrium and could supply little nutriment; and the tufts in 3 had come from a caruncle badly damaged in a prior pregnancy, causing the chorionic tufts to be coarse and in clumps.

Fig. 40 is the afterbirth from an aborted fetus. The conception had followed repeated services. The pregnant horn, 1, had lost all its caruncles during a prior gestation, and their places had been taken by frail adventitious placental growths incapable of sustaining fetal life. The endometrium of the non-pregnant horn had been so completely annihilated that no placental structures, either maternal or fetal, could be formed, and the chorion of that cornu consisted of a thin, transparent membrane.

The five preceding figures are submitted in an effort to illustrate the destruction, or wearing out of the placental tissues of the uterus. The study of the fetal membranes, when available, affords the most valuable index yet discovered for determining the past history, the present genital health, and the reproductive future of the puerperal animal. The breeding female is as old as her placenta. If numerous caruncles are missing from the uterus of a cow, she is proportionately senile: a uterine caruncle once destroyed can never be restored, and each gestation adds to the wear. Great cows, like Glista Ernestine,



FIG. 40. Fetal Membranes from a Wrecked Bovine Uterus.

All uterine caruncles had been destroyed following a prior pregnancy. Conception occurred after many breedings. The embryo could command only very scant nutrition through adventitious placental growths (placenta previa of woman?).

1, Dome-like, inconspicuous non-pregnant cornu; 2, the amnion at the middle of the pregnant horn; 3, naked thin prolongation at ovarian pole of pregnant horn. *(Cornell Veterinarian)*

described by Wing[271] retain the integrity of their placentae; otherwise they could not be great. I studied the fetal membranes of Glista Ernestine's twelfth calf and found them more nearly perfect than those from most dairy cows at second calving. Glista Ernestine did not die from genital disease and was not discarded because of infertility. The *age* of a breeding female can not be authentically computed so well by the recorded date of her birth, or by her incisor teeth, as by her physical health and the health of her uterus as revealed by her afterbirth.

The early determination of the fertility of females which it is desired to use for breeding purposes is of vast economical importance.

For example, Eckles[47] records that, in the herds studied, 3877 services were required for 1151 pregnancies, covering a period of 29 years. There was a total of 2726 wasted services. Among the 1151 conceptions there was a further loss through 14.6 per cent of abortions. Adding the 168 abortions to the 2726 services not resulting in observed pregnancy, there appears a total of 2894 wasted matings, or about 100 per annum during the period covered—a fair sexual load for two bulls through the entire 29 years—without having produced a calf. If the 2894 fruitless matings be estimated to have each cost the keep of a cow for 30 days, the cost of delayed conception amounts to the interesting total of 238 wasted cow years. Eckles states that 65 per cent of the sterility followed normal calving. Had the afterbirths been studied, it is safe to say that in 90 per cent of these the disease would have been apparent, and control measures available.

In modern animal husbandry the breeding is so forced, in both sexes, that the average fertility falls far below the ideal, and the breeding life is prematurely closed. Thus Eckles[47] shows that, among 1204 cows studied, infertility advanced, with unimportant vacillations, from 7.4 per cent at two years of age, to 50 per cent at 17 years. The close of breeding life is expressed or accompanied by various phenomena. In aged dairy cows the cessation of reproduction is often preceded immediately by abortion, twin pregnancy, retained afterbirth or metritis. There is thus a repetition in old age of the types of genital pathology prevailing in the very young, immature females, especially dairy heifers. There are not absolutely so many abortions and retained afterbirths in old age because there are not enough old animals to be observed.

Analogous principles apply to the sire. Errors in handling often render the male prematurely old. Fortunately the veterinarian has at his command, but unfortunately too rarely puts into use, reliable means for measuring the fertility of the male by careful study of the stained spermatozoa. This, with other means of clinical search, enables the progressive veterinarian to follow closely the fertility of valuable sires, and take timely precautions against disaster.

Ordinarily stallions and bulls retain some degree of their fertility to the end of their life. An outstanding danger is frequently present, especially in connection with famous racing stallions which are too long continued in stud service because of their fame, regardless of their low fertility and inferiority of progeny. Thus Stallion 1 in Table III, page 80, was imprudently retained in the stud after his spermatozoa, Fig. 28, indicated clearly that he was highly dangerous. Like-

wise Bull 3 in Table IV, was continued in service after his sexual disease had become a menace to the cows with which he was mated.

Lehndorf[118] relates that a Mr. Phillips purchased in London the thoroughbred stallion Sir Hercules then aged 26, and extremely emaciated and weak. Through liberal feeding and careful handling he had largely regained his vigor when he was 27 years old and was mated with 23 mares, all of which conceived, including one twin pregnancy. All the 24 foals were males! Lehndorf does not state that they were all viable or that a high ratio were of outstanding quality. Two of the 24 were winners of races.

It should be the constant aim of the breeder to concentrate reproduction in that period of the adult life of his animals during which they physiologically possess the highest degree of vigor. When old age approaches, the animal husbandman should lighten the sexual load. The principles involved are essentially the same as the advice to conserve the reproductive life of the young by postponing breeding until they shall have acquired ample growth and vigor. The forced breeding of aged animals is as unjustifiable as the premature breeding of the young. The practice is seen in its most pernicious form in aged stallions of the racing breeds. If the sire has become famous, his services are greatly in demand. He may be quite competent to beget valuable foals, if conservatively handled. But in many cases he is assigned as many mares as would constitute a maximum load for a stallion in his prime. The practice hastens senility, his pregnancies are unsafe, the ratio of abortions and of prolonged gestations increases, weak conceptions terminate unseen and leave behind important genital disease in the mares, and the viable foals are defective in quality.

The burden of reproduction may be lightened in aged females of special value by prolonging the rest interval, after the termination of a pregnancy, before resuming breeding. In dairy cows the load may be greatly lessened by reducing the period of lactation.

When senility becomes obvious, although some measure of fertility remain, the animal should be discarded, because the gestation becomes insecure and any viable young are of inferior quality. Such progeny lowers the quality of the family or breed, just as firstlings from immature dairy heifers are detrimental to the herd. The progressive animal husbandman needs select as his breeding stock the most vigorous progeny of the best parents as measured by longevity and efficiency.

Chapter VIII

THE RAPIDITY OF BREEDING

The Frequency of Coitus

COPULATION in female domestic animals is physiologically limited to the period of estrum, which occurs at varying intervals according to species. Outside these periods the female absolutely refuses the sexual advances of the male; and the male, when freely consorting with the female does not seek coitus at other times. When a confined stallion or bull accidentally escapes from his isolation he violently attempts coitus which may result in serious damage to the female as indicated in Fig. 44.

By hand breeding there is usually a single copulation, sometimes two allowed during a given estrum. Accordingly when two ideally healthy horses or cattle are mated, pregnancy follows and there occurs but a single service during the year. In pasture breeding the stallion or bull may copulate four, five or more times in rapid succession with one female and thus importantly reduce the number of females which he may serve.

Conclusions regarding the advisable number of services by a sire are commonly based upon data which lack volume and authenticity. The conditions vary with species and breed. Racing stallions are not usually placed in the stud until they have been raced two or more years, followed by a period of physical rest before the opening of the ensuing breeding season. On the contrary bulls constitute a definite economic burden until placed in breeding. Accordingly, they are ordinarily placed in service at one to two years of age, when only partly grown. It is well known that many bulls are infertile after one or two years in service, but it is not always known that they were highly fertile at the commencement of breeding. They may have been hereditarily of low fertility, as claimed by Lagerlöf;[116] they may have suffered from nutritive deficiencies as young calves, leading to genital hypoplasia;[257] or the disaster may have been the result of an excessive sexual load. All three forces may act upon a single individual. Each is of definite importance and may render the animal worthless. The evidence is conclusive, in numerous cases, that overbreeding is the chief or sole cause of serious interferences with reproduction, leading to low fertility or sterility of the sire, insecurity of his pregnancies, lowered fertility of his females and subnormal value of his viable progeny.

Most estimates of the sexual strength of sires are based upon the alleged performances of a few great individuals. If the performances of a large group of sires were assembled, the showing would be quite different. The breeder needs have a reasonable idea of what load his herd sire can safely carry, and then make liberal allowances for error and for unexpected stresses due to damaged food, unfavorable weather, disease or injury. The breeding load for the male is commonly based upon the number of females allotted, not the number of services required. The U. S. Bureau of Animal Industry[206] advises 100 cows for a mature bull, but does not indicate whether the sire is to make one or four services per cow. Yet bad weather or food may double the average number of services required. Schmaltz[178] recommends 60 to 80 cows for an adult bull when the service is distributed throughout the year, as is quite common in dairy herds; if breeding is concentrated in the spring months, 40 or 50 cows; and if confined to a very brief period, 30 to 40. Richter[168] reports that a mature bull served approximately 350 cows per annum for four years with marked success, and then suddenly broke down from testicular atrophy and virulent mange. Details are not recorded, the later fertility of the cows is not stated, and the value of the offspring not revealed.

Von Öettingen[153] reports that the famous American trotter, Hambletonian 10, served more than 100 mares annually for five successive years, with a high ratio of pregnancies, then dropped low in fertility, recovered somewhat later and continued in successful service until 25 years old. At 15 years he served 217 mares. He got numerous successful race horses, but full data regarding his progeny are wanting. Like most famous equine sires he naturally commanded the best mares in the nation, and they inferentially played an important part in the quality of his get.

Sundaram[191] reports that in the imperial dairy farm of New Delhi, India, 15 bulls are kept for serving 130 cows and heifers and that regularly a bull is not expected to make more than one service weekly. Schmaltz, citing Schwartznecker, approves of 50 to 60 mares for a stallion, during a breeding season of five months. Schmaltz cities Sand as having observed a stallion which served 271 mares in one year.

There are unquestionably males of great procreative power, but no breeder can know in advance the limitations upon a given individual. The conservative breeder who does not wish a valuable sire to become prematurely old and desires to have the highest use of

his breeding power at a time when his get will be largest, most robust and valuable, should take a lesson from the engineer who, in designing a bridge, carefully calculates the resistance of his materials and the load to be carried and then adds ample reserves of strength against wind or other forms of stress. If the breeder believes his bull capable of serving 100 cows, and he limits him to 50, it may well be that he will have more and better calves, especially should the season and food prove poor or other stresses come in to lower the fecundity of the cows or the procreative powers of the bull. The above discussion is based upon *hand breeding* in which probably but one service is permitted during a given heat. Some dairymen permit a second service immediately, thereby reducing by at least one-half the number of cows he may successfully serve. In *pasture* or *range* breeding the bull ordinarily, if not already exhausted, copulates four or five times in rapid succession and reduces his breeding power in corresponding ratio. It is not known that the probability of pregnancy, or its quality, is enhanced by the repeated coitus. In range beef cattle *pasture* breeding is economically unavoidable. This requires that a larger ratio of bulls be used than in *hand* breeding. Opinions vary widely. My limited observations are favorable to a ratio of 1:10, but many breeders use 1:25 or higher.

The attention of the breeder should not be focused upon the assumed number of pregnancies a bull may establish during a breeding season. In progressive dairying the bull is far more valuable than any one of his cows. Properly handled, he should attain the maximum of reproductive efficiency at four to five years of age and retain it until twelve to fifteen years old. If too heavily used, the security of his pregnancies is lowered; the ratio of cows seriously injured through the occurrence of abortion or retained placenta is increased; the average duration of gestation is decreased; and the size and quality of any viable progeny lowered.

The continuation of the service of a sire, in the average dairy herd, for a number of years is beset with important economical difficulties. Under the prevailing custom of breeding dairy heifers when 15 months old, only two years have elapsed since the pregnancy of her dam began, and it is not prudent to breed them to their sire. If the rate of reproduction described by Wing[271] be accepted as the standard, most of the original cows will have disappeared when the first heifers by the sire are to be bred, and his further retention is not economically justifiable. If the foundation cows produce 8.4 viable calves each, like the 17 females constituting Group I in Table X, the

outlook changes. No good reason can be given why cows should not average 8.4 viable calves or more, if their physical and genital health be intelligently conserved. Far more study should be given to the problem of the conservation of valuable sires and their increased use in adult life when their reproductive vigor should be at its zenith.

The three bulls shown in Table IV constituted a part of the sire force in a large herd of pedigreed Herefords of high class maintained for the production of breeding bulls for a commercial beef herd. Only high-class bulls were used and these were purchased as required from the best available herds. The bulls were permanently free out of doors, with abundant food and exercise. Bull 1 makes the poorest showing. Although used moderately, his ratio of calves to services was poor, and he broke down sexually in seven years. Bull 2 was specially prized, and in the anxiety to secure the maximum benefit from his services he was the most heavily used of any sire in the herd. His ratio of calves to services was poor, however. He made 3.24 times as many services as No. 3 and got a trifle more than twice as many calves. Following the general rule, the female ratio was subnormal in No. 2. Turning to the second generation, the 44 heifers of Bull No. 3 produced as many calves as the 85 of No. 2. That is, calves by the healthiest parents are the most prolific breeders. It is especially noteworthy that Bull 2 dropped in fertility following the increase of his load to more than 100 services per annum, the decline being especially marked in the year following, rather than during the heavy service.

There is evidence that the sexual load aggravates and intensifies disease which might not otherwise be marked. Thus Abelein,[1] in submitting a tabulated service record of a trichomonas-infected bull, incidentally showed that he made nine consecutive services successfully, the first six of which were distributed through 32 days, with no two in one day. The last three were in one day and the next three cows, bred some days later, became infected. Later, during several months, his infectiousness rose and fell in cadence with the frequency of coitus. So far as is known, the observation is in absolute harmony with general experience: when a breeding animal is subjected to excessive strain by those primary factors which caused it to become infected, the contagium grows in intensity, becomes more definitely a *disease* and increases in its transmissibility to other animals. Hence genital diseases advance in incidence and virulence when attempts are made to force reproduction (and lactation) beyond the inherent strength of the individual.

The Post-Partum Rest Interval of the Female Before Re-breeding

The common tendency of domestic animals is to produce young once annually. This law has been modified by selection, feeding and handling so that those having short gestation periods may give birth to young semi-annually and those with long periods, like the mare and cow, may be caused to produce young at shorter intervals than a calendar year.

The mare is ordinarily in estrum at not later than the 8th or 9th day post-partum. According to tradition this is definitely the best time to breed her and the tradition is so ancient that it is well nigh sacrosanct. The tradition is not supported by available facts. In 1926 I[245] recorded the results of 120 ninth-day breedings in a famous thoroughbred stud of high repute for fertility. Thirty per cent conceived at the ninth-day breeding; 40.8 per cent at later matings after having failed at first service; and 29.2 per cent failed. Those which conceived on the 9th day showed 11.1 per cent of aborts and non-viable foals while the pregnancies from later breedings were followed by 4.1 per cent aborts and non-viables. Those which conceived at the 9th-day breeding were presumably the healthiest mares in the stud and were penalized because of their good health.

Caslick[27] reports that of 533 mares bred on the 9th or 10th day, 36.6 per cent conceived, while of those which failed when bred at first post-partum estrum, 56.6 per cent conceived at second breeding.

Jennings[101] states that in the United States Remount Station at Front Royal, Va. during the breeding seasons of 1936-1940 inclusive 110 foaling mares were bred on the 9th day, showing, among other undesirable results:

1. An abortion ratio of 12.8%, which was four times as great as that which followed from first breeding at later dates after having foaled.
2. Dystocia occurred in 15% of mares bred on the 9th day, while no cases of dystocia followed breeding first at later dates.
3. There were 7.5% of foals dead at birth, or born non-viable following 9th day breeding, which was six times as great as that from later breedings.
4. Heavy or retained placentae occurred in 29.1% of mares bred on 9th day and in 21.0% in those bred at later periods.
5. The rate of recognizable conception from one service from 9th day breeding was 43.7% as compared with 67.3% for those first bred at later date.

At the first post-partum estrum clinical manifestations of endometritis are common, with copious genital discharges. The reproductive system has not yet had time for re-adjustment and the necessary repairs to the denuded endometrium. True, the ovum, if fertilized, does not immediately enter the unrepaired uterus but consumes 7 to 10 days in its migration through the oviduct. It is not unreasonable to assume that, after a spermatozoon swims through the genital tube filled with pathological exudate, it may be importantly damaged, and although capable of causing fertilization, its condition may imperil the viability of the ovum.

There is a wide hiatus between the fertilization of an ovum and the recognition of the fact. The extremely primitive type of placentation in the mare provides unusual opportunity for the unseen expulsion or resorption of the fertilized ovum. The inference is quite justifiable that many of the mares bred on the 9th day, but which were later in estrum, actually conceived, but that the ovum perished and disappeared. Recently I had the opportunity[262] to record studies of the breeding records of the stud at the Agricultural College, Cornell University, involving 227 gestations. Again the conception rate was comparatively low from 9th-day breedings and the ratio of aborts and non-viable foals was double that of those mares which failed on the 9th day and conceived later. The average duration of pregnancy for live foals from 9th-day breeding and for foals dead at term was 5.1 days greater, and their birth weight 7.2 lbs. less than that of pregnancies due to later breedings. Here it is to be borne in mind that in the mare many pathological gestations are prolonged, instead of being abbreviated as in the cow; the 5.1 days' greater duration of gestation signified pathological prolongation. This is well shown in the average daily weight increment for the two groups: that for the 9th-day gestations was 0.3792 lbs. per day, contrasted with 0.4071 lbs. for the pregnancies established at later dates.

High-pressure reproduction reaches its maximum in dairy husbandry, and presents some intensely interesting questions in the physiology of reproduction. It is stated that in the mare the corpus luteum of pregnancy atrophies and disappears at mid term and thereafter estrum is barred by the presence of the fetus in the uterus. After foaling, heat occurs within 8 or 9 days or earlier. The corpus luteum of the cow remains intact until after calving. Its presence presumably bars estrum and ovulation. Another element of unknown force enters into the sexual physiology. When discussing the teratology of the reproductive system it was noted in connection with Fig. 5, in which the female fetuses had teratological defects in

the uterine caruncles and teats, that the two organs have fundamentally the same functions and that when one of them was defective, the other might also be imperfect. When normal parturition occurs, correlated phenomena in the sex cycle appear to persist in the dairy cow.

J. Whitridge Williams[226] states: "If for any reason the woman does not suckle her child, the menstrual flow will probably return within eight weeks after labor." If the woman suckles her child menstruation is usually delayed until three to six months, or longer. The psychic effect of suckling her infant apparently exerts an influence upon the sex cycle comparable to pregnancy and placentation.

Authentic data for the cow are scant, but the available facts indicate that the suckling of the calf delays estrum, ovulation and menstruation. Wagner[213] states that in the Hohenheim herd there was a definite prolongation of the interval between parturition and estrum in cows which suckled their calves. But they were allowed to suckle their calves probably for but a few minutes two or three times a day, a large part of the milk was drawn by hand, and the calves suckled for four weeks only. Data are not readily available for beef cows which suckle their calves naturally to the close of lactation. The available evidence, wholly too scant, is that in dairy cows the suckling of the young delays the advent of estrum. Wagner records as the extremes 22 to 118 days, with an average of 60 to 90 days. Those cows which did not suckle their calves showed 43 per cent of the early onset of estrum while those which suckled their calves showed 20 per cent in that group.

The first heat cycles in dairy cows following the termination of pregnancy are extremely erratic, and in many cases may well be classed as pathological. This is well shown in Table XI. In a large ratio of animals the duration of the cycles up to about seventy days is chaotic, after which, if the animal is not bred, the cycles tend to become stabilized. If unsuccessfully bred at seventy days or less, the cycles may continue chaotic. This is probably due in many cases to unrecognized conception, the ovum perishing and disappearing unseen after having damaged the uterus and unbalanced ovulation. A careful study of Table XI and its summary forcibly indicates that the breeding at less than seventy days post-partum led to an orgy of disaster. Table XII dictates the same conclusion from a somewhat different viewpoint.

The chaotic heat periods occurring early post-partum, as shown in Table XI, are highly suggestive of a relationship to nymphomania

TABLE XI

THE DURATION OF THE ESTRUAL CYCLES IN A GROUP OF TWENTY-FOUR CONSECUTIVE HEIFERS FROM A BANG-NEGATIVE PUREBRED DAIRY HERD. In the "Result" columns when recognized pregnancy followed breeding, the duration of the pregnancy is recorded in days; in heat and not bred is indicated by —; and when unsuccessfully bred, by 0.

Number	Days carried in utero	Age in years at first service	Result of first service of heifer	Subsequent Estrual Cycles							
				Estrual cycle	Result	Estrual cycle	Result	Estrual cycle	Result	Estrual cycle	Result
1	275	2.0	278	31 18 20	— — —	22 19 19	0 0 0	38 18 26	— 275 275	18 27 166	— — D
2	277	2.16	277	107	—	20	Sd				
3	276	2.2	272	22 23 23	— — —	21 24 20	271 282 —	77 12 24	276 — ?	23 7	— —
4	279	2.25	274	61 65	0 0	159 8	0 0	13	—	19	283
5	275	2.0	268	59	—	20	0	89	275	82	Sd.
6	279	2.4	272	49 50	— ?	22	283	50	286	84	—
7	281	1.8	273	52 19	0 266	20 60	0 271	38 28	276 —	29 16	— —
8	276	2.9	0	Sd							
9	245	2.3	266	19 21 22 10 23	— 0 — — ?	7 22 21 22	— 268 272 —	8 8 11 21	0 — — —	53 11 18 23	0 — — —
10	275	2.27	275	23	—	83	0	41	Sd		
11	282	1.9	0	80	—	21	250				
12	278	2.2	278	38 36 19	— 0 —	19 45	* 284	** 366 74	0 —	11 20	0 —
13	285	2.2	280	20 27	— —	21 24	— —	24 21	246A Sd	136	275
14	275	2.0	0	18 42 48	0 — Sd	19 24	272 0	26 63	— 269	10 99	0 0
15	275	2.2	0	18 20 20	266A — —	74 11 17	— — ?	22 7	259 —	42 21	— —
16	279	2.2	276	22 10	— —	40 9	— —	19 17	— —	40 21	277 —

TABLE XI—*Continued*

Number	Days carried in utero	Age in years at first service	Result of first service of heifer	Subsequent Estrual Cycles							
				Estrual cycle	Result	Estrual cycle	Result	Estrual cycle	Result	Estrual cycle	Result
17	280	2.2	273	19 21 20	— — —	16 20	— —	18 20	— —	44 21	278 —
18	285	1.9	0	77 20 20	278 — ?	58 19	— —	20 20	278 —	17 20	— —
19	281	2.1	276	12 21 20	— 271 —	11 42	— —	20 24	— —	41 19	— —
20	276	2.1	271	44	Sd						
21	279	1.95	280	30 28	— —	19 23	— —	19 11	— —	19 20	275 ?
22	267	1.9	0	69 20	280 —	108	270	23	—	17	—
23	277	1.6	279	21 21	0 —	14 14	0 ?	68	0	16	0
24	276	1.5	0	197 18	267 —	35 19	— ?	18	—	18	—

In Table XI: A. (cows 13 and 15) = *abortion;* the * after cow 12 signifies that a mummified fetus was recognized and in the next column ** indicates that the cow was again in estrum and bred at 366 days after her prior conception. Sd = *sold* and D = *died*.

A study of the table indicates that the post-partum cycle is highly erratic, the variability being accentuated by service, while if left without copulation, the duration of the cycle tends to become stable.

After the termination of the first pregnancies, sixteen services were made at less than seventy days post-partum with seven recognized pregnancies, including one abortion and one mummy. The five pregnancies terminating in viable calves gave a ratio of 3.2 services per viable calf with an average duration of pregnancy of 273.2 days. Twelve services were made at from seventy to one hundred days inclusive, with eight pregnancies, all of which resulted in viable calves with an average duration of pregnancy of 275.75 days and of 1.5 copulations per live calf. Sixteen services were made at over one hundred days post-partum, with eleven viable calves, an average duration of pregnancy of 274.3 days and 1.45 copulations per viable calf.

It is interesting to note that heifer 9, born at 245 days, was herself the result of a 65 day post-partum breeding. Her three pregnancies averaged 268.7 days.

Among the twenty-four heifers in Table XI, seventeen became recognizably pregnant at the first service and produced seventeen viable calves with an average duration of pregnancy of 274.6 days. Six heifers visibly conceived after two or three services and produced five viable calves, with an average duration of pregnancy of 269.4 days and one abortion, cow 15, at 266 days.

Later, the seventeen females which conceived at first service produced twenty ad-

and may readily be classed as pathological. The nymphomania occurs almost wholly in dairy cows. I have not known an instance of nymphomania to occur in a cow or other female which was pregnant or suckling her young.

There is naturally little available information regarding the interval between calving and the first estrum in beef cows which suckle their young and are constantly at pasture. I have information on one purebred Hereford cow which became pregnant at 17 days postpartum. She was highly fed and subjected to considerable stabling.

A breeder of Hereford cattle, Mr. Edmund O. Pound, Lavina, Montana, has kindly supplied me with highly significant data from his herd. Pasture breeding is practiced, the bulls being turned with the cows on June 25th. The service dates are not accurately known. The duration of pregnancy was reckoned at 285 days. Any cow which calved later than April 25th had an opportunity to become pregnant at 60 days or less after having calved. Among 376 pluriparous pregnancies there were 111 such opportunities and 55 calves (15 per cent of the total) were born at dates signifying conception at less than 60 days after calving. Concerning this 15 per cent Mr. Pound says: "Most of the twins, most of the weak calves, and all of the monstrosities were among the 55 that probably conceived within 60 days. The cows that had their calves at about the same week each year produced more and better calves than those that jumped around in their dates. The cows that 'backed up' the most or had the least rest between pregnancies were the ones that finally lost a whole year." These data are strikingly like those available for dairy cows and for mares.

The fact that the calves are taken from dairy cows immediately after birth, and that this causes an abbreviation of the period between calving and the appearance of the first heat, has appealed to many prominent geneticists and dairy husbandmen as an opportunity to increase the yield of milk in a given unit of time and thereby increase the profits. Thus Chapman and Casida[30] report after extensive studies that: "The conclusion indicated by this investigation is that a

---

ditional viable calves, or a total of thirty-seven, an average of 2.2 viable calves per cow. One additional pregnancy, cow 12, ended in fetal mummification.

The six animals which failed to conceive visibly at first service but became pregnant after a second service (5) or a third (1) later produced four viable calves and one abort, or a total of ten viable calves, an average of 1.7 calves per animal. There is ground for the suspicion that of the six heifers which failed to become recognizably pregnant at the first service, four actually conceived, as is shown by the estrual cycle in No. 11 (80 days); 18 (77 days); 22 (69 days); and 24 (197 days). The length of the post-coital cycle is entirely too prolonged, being decisively longer than the average.

service period (the post-partum rest interval) of about 30 to 60 days is most profitable . . . only the records of normally calving cows were used." No attempt is made to record the ratio of pregnancies beginning at less than 61 days post-partum which were aborted or otherwise ended in disaster.

The usual basis of computation employed by geneticists, dairymen, and milk-testing associations appears to be the quantity of milk produced during a brief interval of time. If instead the dairying efficiency be based upon the lifetime performance, and it be assumed that first there needs be amortization of the capital invested in the cow when she first comes into lactation, a different conclusion is possible. Thus in Cow 1 of Table XII each lactation period exhausted 33.3 per cent of the capital investment; in Cow 8 the exhaustion was 7.1 per cent. When Cow 1 died from overbreeding at 5 years and 21 days, she had yielded 58 per cent more milk than had Cow 8 at the same age, but the lifetime production of 8 was about 5 times that of 1.

The exhaustion of capital investment per viable calf was 50 per cent in 1 and 7.1 per cent in 8. Seventeen years after they entered the herd, Cow 1 had two female descendants, while 8 had 14.

There is profound contrast between Cows 1 and 8. The former calved first at 2.5 years after a pregnancy of 276 days; the latter at 3 years after a gestation period of 283 days. Cow 1 quickly broke under the load of high-pressure breeding, while the stronger Cow 8, conceiving later, made an outstanding record. Cow 9 from the same herd as 8, carried the load of heavy breeding through three pregnancies, then staggered, requiring numerous breedings at irregular intervals, and finally succumbed to dystocia at the close of her 8th gestation. The data show quite well that some cows have the strength to endure high-pressure breeding for a time, but ultimately they break down, and the break comes so early in the life of the cow as to cast extreme doubt upon the prudence of such breeding, if it does not positively condemn the practice as injurious to the individual and the family. Throughout the table it is shown that, the shorter the post-partum rest, the briefer the subsequent gestation period and the lower the average fertility of the female progeny: the strength and vigor of the line is being depressed.

The four instances of twin pregnancy, Cows 4, 5, 6, and 7, each followed conception at less than 60 days post-partum. Each cow faltered. Almost all twin pregnancies in the entire herd, except a few weak primipara, and aged senile cows, have resulted from breeding too early post-partum to permit the reproductive system adequate opportunity to recover its equilibrium. This is quite reminiscent of

TABLE XII

Breeding Records of 9 Cows from Table X Showing the Effects of Rapid, Compared with Conservative Breeding. A = abortion; B = bull; H = heifer; D = died or dead. Under fertility of progeny, G = good; P = poor; VP = very poor; E = excellent; F = fair.

No. of Cow	No. of Pregnancy	Age at 1st Calving or Rest Interval between Pregnancies in Days	Result of Pregnancy	Duration of Pregnancy in Days	Fertility of Progeny
1	1	2.5 yrs.	B	278	VP
	2	37	H	276	P
	3	32	DB	277	
	4	30	DH	231	*
2	1	2.5 yrs.	B	277	
	2	46	H	279	VP
	3	94	H	279	G
	4	101, 127, 171, 194	H	284	VP
	5	62, 100, 132	B	277	P
	6	88	H	288	P
	7	27, 122	H	278	VP
	8	72, 96	B	280	
3	1	3.1 yrs.	H	275	
	2	84, 108	H	278	E
	3	37	B	277	
	4	46, 76	H	282	(1)
	5	52, 156, 237, 261, 306	B	275	
	6	44, 116	B	279	
	7	51	H	289	VP
	8	102	H	278	G
	9 * * *	54 * * * * * *	H * *	275 * *	VP * *
	10	474	B	281	
4	1	2.1 yrs.	H	280	P
	2	62	B	282	
	3	61	H	276	VP
	4	39	BB	275	
	5	107, 127, 145	B	278	
	6	76	H	282	G
5	1	2.8 yrs.	B	?	
	2	50, 125	B	283	
	3	33, 95	H	282	VP
	4	57	B	288	
	5	62, 103, 158	H	277	F
	6	53	H	277	F
	7	36, 58	BB	275	
	8 * * *	49, 73 * * * * * *	H * *	284 * *	VP * *
	9	174	B	280	
	10	177	H	281	G
	11	321	H	284	G
6	1	2.8 yrs.	H	276	D
	2	40	H	275	G
	3	42, 45	A	90	
	4	40	B	274	
	5	50	BH	224	A
	6	346	B	277	

TABLE XII—*Continued*

No. of Cow	No. of Pregnancy	Age at 1st Calving or Rest Interval between Pregnancies in Days	Result of Pregnancy	Duration of Pregnancy in Days	Fertility of Progeny
	7	76	B	282	
	8	84	A	243	
7	1	2.2 yrs.	H	276	G
	2	64	DH	276	
	3	44	B	271	
	4	51	H	279	G
	5	58	B	281	
	6	18, 42	BH	275	F
	7	49	DB	264	
	8	89, 264	A	* *	* *
	* * *	* * * * * *	* *		
	9	339	H	288	G
	10	486	H	277	G
8	1	3 yrs.	H	283	G
	2	52, 71	B	276	
	3	28, 59	H	267	G
	4	78	B	256	
	5	48, 101, 131	H	278	G
	6	74	B	279	
	7	106	B	279	
9	8	100, 162, 180	B	263	
	9	99	H	279	G
	10	82	B	286	
	* * *	* * * * * *	* *	* *	* *
	11	167	B	285	
	12	216	H	284	G
	13	238	B	285	
	14	122	H	281	(2)
	1	2.7 yrs.	H	288	F
	2	39	B	274	
	3	52	H	275	VP
	4	69	H	279	VP
	5	20, 96, 104 144, 227, 255	H	278	(3)
	6	56, 76, 113	B	278	
	7	144	B	283	
	8	48, 69, 90, 129, 172	DB	289	(4)

Notes, Table XII. * Cow 1 died from parturition. The * * * lines in table indicate the date of change in plan of breeding by which a rest interval of 180 days between calving and re-breeding was practiced. #Calf accidentally killed. (1) Heifer died young. (2) Heifer under breeding age. (3) Heifer produced no live calf. (4) Cow died from dystocia.

The 9 females presented in Table XII have been selected from the animals in Table X as representative of the early breeding of heifers and the early post-partum breeding of calving cows. They include the only twinning cow in Group I of Table X (Cow 4). The 4 twinning cows included in the table are from the foundation herd and are taken in

the contribution of Clapp[31] previously cited in connection with twinning after nymphomania; if the cow was bred at less than 35 days after the occurrence of cysts, twins were very probable.

In the entire herd of Table X, under the original plan of early post-partum breeding, 64 pregnancies occurred at less than 60 days post-partum. The average duration of gestation, excluding those classed as abortions, was 274.77 days. Under the revised plan of 180 days' rest, the last 64 pregnancies of 1936 averaged 282.2 days, an advance of 7.43 days. The latter 64 included numerous primipara, in which it is commonly believed the gestation is more brief. That conclusion appears to rest almost wholly upon data from dairy heifers, usually bred to calve at about 2 years: under the revised plan, to have heifers calve not earlier than 2.75 years, the duration of gestation in primipara was the same as for pluripara.

The 17 foundation heifers constituting Group I in Table X, purchased from a herd conservatively handled and of unusually high fertility, were bred to calve at somewhat less than three years on the average, and their first gestation periods averaged 278.8 days. The 38 other heifers, constituting Group II, emanating from less fertile herds and bred to calve first at about 2 years, show an average dura-

---

their consecutive order as they are arranged in the herd records. All four twin pregnancies followed conception at less than 70 days post-partum.

SUMMARY

Group I First pregnancies.
Group II Pregnancies occurring at less than 60 days post-partum.
Group III Cows not bred until after 60 days post-partum, or which failed at first service and conceived later.
Group IV Revised plan; cow not bred until 180 days post-partum.

Group I	9	pregnancies,	duration	275-288 days.	Average 279.1
Group II	23	pregnancies,	duration	264-289 days.	Average 271.1
Group III	37	pregnancies,	duration	256-289 days.	Average 279.2
Group IV	10	pregnancies,	duration	277-288 days.	Average 282.6

Ratio of pregnancies having a duration from 279-288 days incl.		Abortions and calves dead at birth.	
Group I	33.3%	Group I	0.0
Group II	20.0%	Group II	21.7%
Group III	48.6%	Group III	10.8%
Group IV	60.0%	Group IV	0.0

Twins. Group II, 17:100. Groups I, III and IV, 0.

tion of gestation of 271.9 days. The last 55 primipara calving under the revised plan of breeding had an average duration of pregnancy of 282.1 days.

Much is said regarding the variations in the duration of pregnancy due to differences in breed, climate, food, or family. In this instance the animals were of one family without change in climate, location, food, or of handling, except in breeding.

It is well shown in Table X that the maximum reproductive power of the different groups was immutably fixed for life at the close of the first breeding year. Disease or disaster may arise to lower or wreck the fertility of a healthy cow, but no known force can convert into highly fertile animals a group of heifers which have bred poorly as primipara.

The principle, which it has been attempted to establish, is well supported by extensive studies in other herds and the conclusion seems inescapable that conservative animal husbandry demands that a liberal rest between pregnancies be allowed. Whether early re-breeding is followed by prolonged gestation, as is common in the mare, or by abbreviated pregnancies, as is the rule with cows, the underlying logic is that the parturient female needs ample time for recovering her sexual equilibrium before resuming pregnancy. The principle involved is allied to the use of a sire producing imperfect or weak spermatozoa. Thus in Table II, Bull II, suffering from diseased seminal vesicles, got pregnancies of notably brief duration. Bacteria may have been mixed with the semen at coitus, but not of sufficient virulence to terminate gestation. The load of pregnancy with the weak ovum was too great and the gestation was terminated early in order that the diseased uterus might get relief. I have very recently studied a herd in detail in which early re-breeding was practiced. There were two sires being simultaneously used, one an aged, healthy sire, with gestation periods averaging 276.4 days and the other suffering with purulent inflammation of one seminal vesicle, whose pregnancies averaged 270.2 days. Two factors were present in the younger bull, both of which acted in the same direction.

The optimum period of post-partum rest for dairy cows is not readily determined. It is modified by food, age, method of handling and various environmental factors. The herd shown in Tables X and XII was dependent upon grazing for sustenance in a climate where the rainfall was undependable and consequently the food supply was at times defective in quality and quantity. The revised plan of 180 days' post-partum sexual rest, so that each cow might calve each 15

months, definitely advanced the reproductive efficiency. Such a plan is not economically available under most environments. The principles involved are available in all herds. Under the plan for annual calving, if 285 days be accepted as the average duration of physiological gestation, a rest interval of 80 days is available, and if cows are re-bred at the first heat after 70 days, annual calving is maintained if the animals are healthy.

It is not at all essential generally that cows should calve annually. In most dairies breeding is continued throughout the year and good dairy cows continue to yield a fair amount of milk for one or two years. Numerous progressive dairymen allow a rest of 120 to 150 days and feel that they get more and better calves in this manner.

When considering the duration of gestation the optimum was placed at 279-288 days inclusive. My studies of the herd illustrated in Tables X and XII indicate clearly that a liberal post-partum rest favors the optimum duration of pregnancy, the longevity of the cows and the vigor and fertility of the progeny. Thus in a group of 58 pregnancies under the conservative plan of re-breeding, 96.5 per cent fell within the optimum of 279-288 days. Of 64 pregnancies in the same herd, established within 60 days post-partum, 39.1 per cent fell within the optimum. In the Cornell University herd, as reported by Wing[270] 55.44 per cent of pregnancies terminated within the proposed optimum. Cow 1 of Table XII provides an illustrative breeding history. She was purchased from an ultra-fashionable herd at the highest price paid for any one of the 55 original heifers. As a primipara she dropped a bull calf when 2.5 years of age, after a pregnancy of 278 days. The calf was critically ill from umbilical infection, apparently recovered, was retained as a sire, and failed.

Despite the two danger signals of abbreviated pregnancy and a seriously diseased calf, the cow was bred and conceived 37 days post-partum, and bore a viable heifer at 276 days. She was next bred after an interval of 32 days and dropped a non-viable bull at 276 days. Thirty days later she was again bred, aborted a heifer fetus at 231 days and died from metritis at 5 years and 22 days.

Her one viable heifer had a checkered career of three dead calves and one pair of viable twins consisting of a bull and a neuter. At 7 years of age she came under the revised plan of liberal post-partum rest, after which she rallied, produced one viable bull and three viable heifers with an average duration of pregnancy of 281.2 days.

Vigorous cows not infrequently withstand early post-partum breeding for one or two periods without material evidences of serious

injury, but I fail to find definite evidence of even one cow which has successfully carried the abnormal load through three consecutive pregnancies and retained physiological fertility. Fortunately most cows do not come in heat at less than 40 days post-partum. All available data indicate clearly that the breeding of cows at a period importantly less than 80 days postpartum is definitely detrimental to the reproductive life of the animals. Individual cows of extraordinary vigor may carry the excessive load through one to several pregnancies, but ultimately serious injury results and lowers their value and that of their progeny.

Some veterinary authorities of high repute disagree with the foregoing view. Thus Elder[49] speaking for the University of Missouri, while advocating the control of abortion by the agglutination test and slaughter of reactors, states that "Bang's disease-free herds calve every 11.5 months" while those in infected herds produce only one calf each 20 months. In a personal communication Elder admits that the Missouri University herd does not produce one calf per cow each 11.5 months and that he has no definite knowledge of the existence of such a herd. Mohler, Wight and Lash[145] of the Bureau of Animal Industry, United States Department of Agriculture, state "Furthermore cows in diseased (Bang's disease) herds, on the average calve only about once every 20 months instead of once every 11½ months as in healthy herds." They fail to present evidence to indicate that any such herd is known to exist. Elsewhere Miller & Graves,[139] pursuing their investigations in the Bureau of Dairy Industry herd under the official direction of Mohler, report that after isolation, the Bang-negative fertile cows produced during a period of 4 years, 83.59% of living calves and 16.40% of abortions and dead calves. The duration of time for the production of calves is not stated. There was much sterility (or unseen death of ova) so that about 3.5 services were required for each recognized pregnancy. If the estrual cycle be placed at 21 days, there would be a delay of 52.5 days in conception. This, added to 283 days of gestation, equals 335.5 days from breeding date to calving. In order to calve each 11.5 months (350 days) it would become necessary that the cow be served at 14.5 days after calving.

I know of no herd in which there was produced an average of one living calf each 11.5 months. The Bang-free agricultural college herd described by McNutt, Walsh and Murray[131] did not produce an average of one calf each 11.5 months nor is there favorable indication that they averaged one in 20 months. The large agricultural herd studied by Henderson[86] did not produce one calf per cow for each

11.5 months. So far as may be learned from Henderson's thesis they failed to reach the despised 20 months for Bang-infected herds. Available information, which is apparently authentic, indicates that the agricultural college breeds the cows very early after calving, hoping to average one calf every 11.5 months or less. Besides, according to Henderson, a prior herd was slaughtered in order to be rid of Bang's disease, new cattle purchased, and any reactors developing in the herd promptly annihilated. Henderson further relates that of 835 gestations only 57.2 per cent came within the optimum duration of 279-288 days. It has been insisted in Chapter III that calves born outside this optimum are of inferior vigor and fertility. No herd with so unfavorable a duration of pregnancy as that described by Henderson can be profitable or self-supporting in private dairy industry.

If it were possible so to handle a herd that it might average one calf for each 11.5 months, the occurrence should be by far most frequent in the herds of agricultural colleges. They have the leaders in genetics, animal husbandry, food chemistry, and veterinary science. They have the further great advantage over private dairymen because of the available funds which enable them to replace tardy breeders. On the whole, breeding statistics of domestic animals, especially for the dairy cow, are very imperfect and extremely difficult to so prepare that they may authentically and clearly show the actual rate of reproduction.

In the preceding pages important facts have been submitted to indicate that one of the most reliable means known for the advancement of abortion, sterility, retained afterbirth, and allied phenomena consists of breeding a dairy cow or other female at the earliest possible date after parturition or abortion. The danger is most spectacular in the dairy cow which is asked to produce an artifically high amount of milk and at the same time to supply nutriment to an intra-uterine fetus. The mare is bred earlier than the cow after parturition, but she is not asked to yield an artificial milk supply. Moreover the equine ovum develops very slowly in the early stages of pregnancy, as compared with the great rapidity of development in the cow. Finally the equine fetus requires 11 months for normal development as compared with 9 for the bovine, and in the end attains approximately the same birth weight as the calf as related to the weight of the dam.

Sex Ratio and Breeding Practices

Sex ratio has been discussed at considerable length in the companion volume, *Veterinary Obstetrics*, and extended repetition is un-

warranted. Extensive data of reliable character of the sex ratio in domestic animals are wanting. A Hereford herd under my direct observation produced aproximately 5000 calves per annum as enumerated at castration-branding time. The herd was at high elevation in tropics, constantly out of doors in a salubrious climate, with abundant forage of good quality. The records show that among 94,436 calves of branding age the ratio of bulls to heifers was 102.3:100, thus closely approaching ideal sex parity. Inferentially there were some deaths of young calves, but there was not a sufficient number of these to attract attention. In *homo* the infant mortality is distinctly higher for boys than girls, but this is not known to be the case in calves; and at most, in this instance, could have played no important role in the equalization of the sexes.

The sex ratio, as stated incidentally in this, and the two preceding chapters, may be profoundly disturbed by the flagrant violation of the physiological laws of reproduction as established by evolution through eons of time. The tendency, so far as is known, is uniformly toward an excess of males. Available data appear to justify the conclusion that when the general or sexual health of either parent is depressed, the nutrition or vigor subnormal, the ratio of males is definitely increased.

More complete data are available for women. J. Whitridge Williams,[226] citing Rauber, gives the prevailing ratio as 106:100. Citing Ahlfeldt, Williams states that among primiparous women bearing their first child at 40 to 50 years, the ratio advances to 130-140:100. At such age the woman is nearing the menopause and her reproductive energy is decreasing. The ratio of boys is also excessive in aborts and premature births. Thus when either or both parents are so low in general, or sexual health that the fetus dies and is expelled, the male ratio advances. Recently the influence of the health of a parent upon the sex of the progeny has been significantly shown by Seymour and Koerner[179] who record the sex of 5840 infants born after artificial insemination with husband's semen, with a sex ratio of 157.1 boys to 100 girls; and 3649 infants following insemination with semen from donors, in which the ratio was 136.6:100. The donors were presumably more fertile than the average husband in a childless family, and therefore there occurred some decrease in the ratio of boys.

This is in harmony with the statement of Lehndorf[118] regarding the stallion, Sir Hercules. He was greatly emaciated when purchased at the age of 26 years. Owing to good feeding and care he largely recovered his vigor and at 27 yrs. served 23 mares, all of which con-

ceived, one with twins, and all 24 foals were males. It appears from available data that the progeny of parents of subnormal vigor, whether due to immaturity, senility, too rapid breeding, or to genital, or other disease, commonly show an excessive ratio of males among their progeny. It appears justifiable to conclude that an excess of males indicates reproductive weakness, with lowered resistance of the intrauterine young, the pregnant female, and the new-born to infections which may cause abortion, sterility, and death of the new-born young and of the mother.

Chapter IX

FOODS AND FEEDING

AFTER an animal has approximately completed its physical growth and a food surplus beyond its physical needs is assured, such surplus may be safely diverted to physiological reproduction. Common herbivorous domestic animals, in which mankind has its chief economic interest, subsist naturally upon grasses and cereals. Both plants and animals are readily transferred from one country into another within climatic limitations. Domestic animals thrive best upon varied vegetation. As a rule the grasses and cereals of all lands are introduced into each, in so far as climate permits, and domestic animals thus have access to a wide variety of vegetation. There is no forage plant which alone furnishes all food elements for domestic animals in such form as to assure the desired health and vigor. Domestic animals are most numerous and important in the temperate zone, in which their food is profoundly influenced in quality and quantity by temperature, moisture, and soil. In temperate climates, winter depresses or halts the growth of forage, and animals become dependent upon the naturally dried grasses, or upon the artificially preserved hay, ensilage, or grain. In whatever manner the food is supplied, it is constantly exposed to deteriorating influences, during growth or in its preserved state, which may lower or inhibit reproduction in the animals dependent upon it for nutrition. Such interferences with reproduction are as old as history, usually associated with war or famine. Fleming[61] mentions destructive epidemics of abortion in cattle in Italy as early as 232 B.C., and cites Köliser as having observed highly destructive abortion and sterility in Transylvania in 1719, following an extremely severe drought. On the other hand, up to about half a century ago, destructive sterility and abortion were frequently observed, associated with heavy infestation of grasses and cereals with ergot (*secale cornutum*) and other fungi, caused by excessive moisture during the growth of the forage. Studies have shown that these fungi have no important specific power to prevent or interrupt pregnancy. But when fungi abound the infested forage is damaged, and the energy of the animal dependent upon it is lowered and reproduction disturbed or excluded. The lowered vigor of the animal opens the way for the destructive invasion of the reproductive system by bacteria, protozoa and viruses.

There is no occasion to discuss here the necessity for an ample supply of proteins and hydrocarbons. Associated with these are other undeniably essential food elements, some of which have been grossly overemphasized in a manner which has befogged the problem of nutrition and opened the way for dangerous propaganda. Breeders are urged to believe that, by the addition of this or that substance, physiological reproduction will be restored, despite the existence of other pernicious forces over which the alleged panacea is without influence.

The Essential Mineral Constituents of Food

Certain mineral elements are essential to the health and life of domestic animals. Ordinarily they are obtained in adequate quantity, and, so far as known, of the highest quality, in the natural food and water. Most of these minerals are commonly superabundant and are in part discharged by the body, unused. Calcium phosphate is commonly regarded as one of the most essential mineral salts, constituting, as it does, an important part of the osseous skeleton, but the skeleton, once formed, is relatively permanent. With an ample supply of this salt, horses, in the presence of bone disease, sometimes throw out excessive quantities in the urine. The bone disease is not due to a defective supply of calcium phosphate, but to a wastage of that salt. In some areas it has been impressively claimed, and supported by good evidence, that the generally available food supply is deficient in this salt. The most extensive studies in this field have been made by Theiler, du Toit and their colleagues in South Africa. A good review of their studies with extended bibliography up to 1929 is submitted by du Toit and Bisschop.[46] They have shown that the forage over large areas of the veld is deficient in its phosphorus content. It is not clear that the soil is markedly defective in phosphates. The rainfall is chiefly limited to the months of December to March, and in 1926 to 1928 fell as low as 13 inches per annum. During eight months of the year, apparently little, or no rain falls. In the absence of moisture, calcium phosphates present cannot well be dissolved and made available as plant food. In some dry areas there is an extremely heavy deposit of dew, which may be available to plant life as water, but it does not materially penetrate the soil to dissolve the mineral salts. So du Toit and Bisschop relate that the phosphorus content in the forage was most deficient during the dry season. Similarly in many arid or semi-arid areas, although reserves of forage exist, the phosphates, and perhaps other equally important elements, drop below nutritive requirements during persistent drought. The South African

investigators mentioned have shown that the defects in the cattle attributed to mineral deficiencies in the forage may be overcome by the feeding of bone meal or of other forms of calcium phosphate. It is indicated also that the native Africander cattle, bred for many centuries by the Hottentots, do not suffer so severely from phosphorus deficiency as do the improved, more rapidly maturing European breeds.

The phosphorus deficiency, besides delaying physical and sexual maturity, is indicted as causing the chewing of the bones of dead animals which have been allowed to lie indefinitely upon the veld. The bones, which are infected, cause parabotulism in the cattle chewing them. The administration of large amounts of bone meal improves the general health, advances reproduction greatly and causes the cattle to cease chewing bones. It has previously been stated that the lowering of the vigor of mammalia reduces the fertility and disturbs the sex ratio, causing an excess of males. This rule proved true in the investigations in South Africa. The conclusion might be drawn that the chewing of bones by the veld cattle was an effort to obtain from them the phosphorus lacking in the forage. While this may be partly true, it is unsafe to lean too heavily upon such hypothesis.

Cattle in poor health chew and swallow all sorts of articles devoid of any bearing upon the etiology of the disease affecting them. Thus I have observed a herd of 25,000 unusually healthy cattle quite free from observed chewing of inedible articles, largely grazing upon the slopes of a great volcano; another herd upon the opposite side of the volcano, at approximately the same altitude, where the soil was derived fundamentally from lava from the same volcano, were notably in the habit of chewing large pieces of lava. The direction of the wind and hence the rainfall, and the character of the forage, differed in the two areas. They did not chew lava because of a deficit of lava in the forage. New-born animals which are in bad health have a pernicious habit of picking up and swallowing the feces of other animals, but that does not point to the absence of any specific substance from their food. Neither does the fact that the administration of large quantities of bone meal to the South African cattle was followed by marked benefit prove in itself that the low vigor was due wholly to phosphorus deficiency. In therapeutics, amelioration or cure of a disease does not prove that the malady was caused by the want of the remedy; such a philosophy would be difficult to follow in the cure of milk fever by introducing air into the udder. The evidence of phosphorus deficit is ample, and the benefit derived from

large quantities of bone meal is clear. In many, if not all lands, perverted appetite occurs in various species of animals and the tendency is that many or most individuals of a herd or flock participate in the perversion, the general vigor becomes depressed, and fertility drops or ceases. The general tendency is to attribute the pica to the absence of some food element, but the evidence is usually inconclusive. So in some areas when young are born without hair, the defect is attributed to a want of iodine in the food, and in some cases apparently the difficulty is overcome by the administration of iodine to breeding animals.

The problem is of definite importance. Unfortunately the basic causes are not clear and in many cases the need of animals for a given food element is grossly exaggerated. Thus it is claimed to be essential that cattle have a generous allowance of salt; excellent authorities place the requirements at two to four pounds per month. I have intimately observed a herd of 25,000 cattle, not one of which has tasted salt. I have not observed healthier cattle in any country, or under any allowance of salt. They were on a soil consisting fundamentally of disintegrated lava, which accordingly had no chloride of sodium. Presumably the cattle somehow obtained the necessary amount of salt, but no imaginable source existed from which the amount considered necessary might be derived. Such observation does not prove that in most areas it is not best to supply the amount of salt generally recommended.

So much has been written, and so little definitely known in this field, that it is unsafe to advise what course to pursue in a given case. Quite certain it is that it is unsafe for a breeder to invest funds in specific mineral food constituents as a panacea for low fertility. If the soil, upon which the forage or cereal is grown for animal food, is clearly defective in an essential mineral element, the question arises whether it may be best to transfer the defective land to other uses; to fertilize the soil; or to feed the missing mineral to the animals. Probably the first course is most economic in a majority of the cases. In any event the breeder should proceed cautiously in the purchase of alleged mineral panaceas for sterility.

Vitamins

During recent years much investigation has been devoted to a number of food elements designated vitamins. Many of these have been identified and specific powers attributed to them. Vitamin A is said to regulate growth; vitamin D is essential in the diet in order to exclude rickets; and vitamin E is essential to reproduction. Grant-

ing the correctness of these three hypotheses, it becomes obvious that any deficit of either of the three excludes reproduction. An animal barred from growth by want of vitamin A cannot physiologically produce young and an animal suffering from rickets owing to a deficit of vitamin D cannot be expected to breed. So with any other vitamin essential to any fundamental body function: if it is not present in an available form, the animal organism is thrown out of balance and reproduction depressed or excluded. The commercial propagandists of nostrums would have the breeder believe that an investment in vitamin E will assure ideal reproduction in the absence of vitamins A and D, as well as that of other essential food constituents or in the presence of hereditary lethal defects in the family. The vitamins are obtained from the food and it is not known that they are improved in the process of their extraction.

There is no ground for doubting the value of vitamins. The fact of greatest value is, that properly grown and preserved food, of the character to which the species of animal has been accustomed throughout the ages, contains the essential vitamins in the natural form for the animal. When a dog is fed on parsnips or a cow on fish, there may be need for the juggling of vitamins. The breeder should have profound respect for the proper variety of food for his animals, grown in a favorable environment and preserved, in case of need, in such a manner that an ample supply of vitamins, as well as of mineral salts, shall be present in the best attainable form. The problem of adding artificially produced vitamins should be approached warily.

The presence of any one, or of all vitamins, in adequate quantity and of ideal quality, assures in itself neither health nor fertility. Cow's milk is said to be the highest ideal among all foods for young children and frail persons, because it contains in the best form all essential vitamins and minerals. Nevertheless, young dairy calves, fed upon this same milk from their mothers, show the greatest incidence of disease and the highest mortality rate of any extra-uterine domestic animal. This is not an indictment of milk as a food, but is an effort to show that the mere presence of a given element in the food of an animal is but one of an infinite number of factors making for health and vigor.

As with mineral elements and other substances, the administration of vitamins in quantity or form foreign to the natural intake, has been believed to act in a highly favorable manner. Thus Moussu[147] believes that *Brucella abortus* does not destructively invade the gravid uterus of the cow except the resistance of the animal be low-

ered by a deficit in the food of vitamin E. He contends that the vitamin E normally present in the wheat germ and other foods is largely destroyed prior to assimilation and that its prophylactic power is best attained by making an oil extract and injecting it subcutaneously. By this method he claims remarkable success in 7000 cows, causing a disappearance of abortion, retained afterbirth and death of the new-born. The plan failed to control sterility. He fails to define abortion or sterility. If abortion be defined as "the observed expulsion of a fetal cadaver" and sterility as "the unseen disappearance of a fertilized ovum," which is largely, if not generally true, the sterility in the 7000 Moussu cows may have replaced the abortion rate prior to the administration of the vitamin E in oil. Moussu fails to state how many of the 7000 cows dropped viable calves, nor does he intimate the duration of gestation. The disappearance of abortion in a herd or group of herds does not unequivocally indicate ideal reproduction. There have been many remedies proposed for abortion, and enthusiastically recommended by their proponents. Men of high professional standing have proposed carbolic acid, methylene blue, killed bacteria (bacterins), living bacteria (vaccines), isolation of the infected and annihilation of the diseased for the control of intra-uterine death of young, but no one of these, nor all together, can prevent the death of the fertilized ovum doomed because of a hereditary lethal defect, or imprisoned within an unhealthy uterus.

Chapter X

LABOR, HOUSING AND CONFINEMENT

THE domestication of animals involves great modification in their environment, and each change introduced reacts upon their vigor and fertility. Ample food may be regularly supplied, thereby decreasing or obliterating the need for physical exertion by the animal in procuring nutriment. This permits more rapid growth and earlier sexual maturity. The energy naturally devoted to securing food may be partly transmuted into labor or reproduction. The relations between labor, confinement, reproduction and housing are so intimate that no one element can be wholly independent.

Labor

When under domestication, a mare is largely or wholly relieved from the responsibility of procuring food for herself. The physical energy thus conserved may be largely converted into productive labor, without recognizable danger to her physical or reproductive health. Hence the farmer who works his mares moderately and handles them carefully may breed them to foal annually and expect them to remain vigorous and fertile until twenty years old or over. The practice is often unsatisfactory because the farmer permits emergencies to arise during which the mares are overworked or otherwise improperly handled.

Race-horses are largely infertile while in training, and any efforts at breeding them during this period lowers the racing power. Commonly both sexes are trained young and are not used for breeding until after a rest following their withdrawal from the turf. The training has consumed all available energy, leaving no surplus for reproduction. Even so, the rest interval frequently fails to effect readjustment and the restoration of fertility. Many fillies fail to become recognizably pregnant during their first year in the stud. The duration of pregnancy is erratic, and both abortion and prolonged gestation are frequent. The stallions behave little or no better, and absolute sterility is not very rare.

Possibly another interesting factor enters into the question of infertility among racing stallions. During training, the reproductive system becomes atonic and the scrotum, with the contained testicles, becomes abnormally pendulous. The trainer applies a suspensorium in an effort to protect the organs. This holds the testicles firmly

against the inguinal surface and imprisons heat. It has been shown by McKenzie and Phillips[130] that the production of potent spermatozoa in the ram is readily lowered or stopped by insulating the scrotum in a manner to elevate the temperature from about 95 to 104 F. Lagerlöf,[114] in extensive and highly interesting studies, has clearly established the fact that testicular degeneration with absence of spermatozoa is readily produced by insulating the scrotum of the bull by so covering it as to definitely prevent the normal refrigeration of the testicles. The principle seems well established, for all males with testicles in the scrotum, that elevation of the temperature of the sac causes testicular degeneration and infertility. It is not impossible that the use of the suspensorium in stallions serves to elevate the scrotal temperature and interfere with spermatogenesis.

Confinement

At the opposite extreme to the severe physical trials to which racehorses and other animals are exposed in labor or sport, is the close confinement of some species and breeds. The maximum of confinement is ordinarily seen in dairy cattle. The ultimate goal of dairying is the production of the greatest amount of milk of the highest quality at the minimum cost. This is commonly attained when cows are grazed upon luxuriant pastures, and a supplement of grain added. Generally it seems to be believed that the luxuriant pasturage alone is responsible and that the associated physical exercise does not play any important part. If the pasturage is not of high class and the cow needs travel too far to obtain adequate grass, there can be little doubt that the volume and quality of milk will be lowered. The value of the physical exercise and the associated sunlight have not been duly appreciated. Denmark and New Zealand, with equable temperature and adequate rainfall, so that cows may be at pasture almost or all the year, are noted for their heavy exportation of dairy products. So far as the literature leads, the heavy exportation of dairy products from these countries is not due to spectacular records of famous individual cows, but to the general vigor, fertility and longevity of the dairy animals.

It is assumed that the burdens of pregnancy and lactation serve as a substitute for physical exercise. The hypothesis is not supported by known facts. Besides lack of physical exercise and sunlight, confinement introduces a third highly important factor—stable pollution. Many important diseases of domestic animals are definitely attributable to stable pollution. Among these stable maladies those interfering with reproduction loom large, and constitute one of the

most important reasons why genital diseases are most destructive in dairy cattle. Sterility, abortion and retained afterbirth are most prevalent in late winter and early spring, after prolonged stabling.

The hygienic principles involved in the problem of confinement are alike for all species, ages and breeds. Among dairy cattle the dry cows, the young stock and the calves are confined much the same as the cows in lactation. Dairy cattle are mostly confined in stanchions, which constitutes the closest confinement attainable. The animal is virtually limited to lying down and getting up. In the central and northern United States dairy cattle are usually closely confined from October to April inclusive. The milking cows are confined because they yield more milk, the dry cows are confined to save food, the young stock are believed to grow more rapidly confined in the stable, and the bull is confined because he is less in the way.

Cattle kept for show or sale look better, according to commonly accepted standards, when kept in warm stables during winter. During summer it is believed that the animal designed for show looks better if stabled during the day, avoiding sunlight and flies.

Vigorous exercise definitely stimulates the principal body functions—alimentary, urinary, cutaneous and respiratory—and reproduction is fundamentally based upon the vigor of these. When physical exercise is instituted after a period of quietude, one of the first observable results is defecation. If the exercise is continued, for a considerable period, body fluids are poured into the digestive tract and its contents are softened. Without such exercise the dairyman attempts to overcome the ill effects of confinement by the use of laxative foods, or by a luxuriant supply of salt, which by increasing thirst, causes the cow to drink more water and thus induce a laxative effect.

During the winter season many dairymen turn their cows into the barnyard for a very brief period each day, ostensibly for exercise. The barnyard is often shaded on one or several sides by buildings, largely excluding sunlight. The yard is often muddy, the mud being heavily mixed with feces, or it is so icy that the cows have to move with caution in order to avoid falling. Usually the yard is entirely too small to permit healthful, invigorating exercise. Consequently the alleged exercise period is largely spent by the cows in standing with arched backs, staring coat and shivering body. The value of such exercise periods may well be questioned. The exercise paddock should be of ample size and as free from dung and filth as practicable. Exercise is encouraged by placing at a distant part of the enclosure hay or other tempting food. This may be placed in an open shed,

protected from rain and against prevailing winds. The duration of the exercise period should be ample; when the weather is at all favorable, it should continue for several hours. When possible it is well to allow the cows the freedom of pasture or field where they may search for chance morsels of food.

Dry cows, calves and heifers should not, so far as avoidable, be confined in stables and stanchions. The needed protection against inclement weather is most economically provided by means of commodious sheds closed on three sides and opening into a large paddock or field, thus assuring perfect freedom.

Dairy bulls are commonly even more closely confined than cows, and if hand breeding is practised, are usually stanchioned throughout the year. A large ratio of dairy bulls show a marked decline in fertility early in life. It is not known precisely to what extent the confinement induces the low fertility but there seems to be ample reason for concluding that it constitutes a highly important factor.

Bulls would retain their fertility far better, and their keep would be much less expensive if the dairyman would fence a commodious paddock for the bull, which during the grazing season would supply ample grass for the sustenance of the animal. He should be provided with a shed which would afford protection against wind and storm, and provision made for food and water. The original investment is far greater than that of a stanchion in a dark corner of the stable, but when the plant has been established, the cost is limited to feeding and watering. This should be so arranged that it would not be necessary to enter the enclosure. Provision should also be made for attaching the staff to the nose ring, when it is desired to use the bull for service, without entering the enclosure. Such plan assures the highest degree of health and fertility and in the end the most profitable method.

In Section VII the question of the confinement of dairy calves is discussed at some length.

SECTION II

ESTRUM, COITUS AND PREGNANCY

Chapter XI

ESTRUM, OR "HEAT"

WHEN female domestic animals are born, their ovaries contain, in a primitive form, all the follicles and ova which are to be formed during life. The number of ova is very great, but only a few are destined to become mature and to function.

Physiologically all follicles are quiescent until the individual has become sexually mature. Pathologically, especially in dairy cattle, the follicles may become greatly enlarged in the fetal ovary and cause correspondingly abnormal size of the gland, as indicated in Fig. 41. At times these rupture, and hemorrhage into the crater follows, giving to the organ the general appearance of a corpus luteum. It is not known that ova form in such follicles: apparently the primitive follicles merely undergo cystic degeneration and the primitive ovum is destroyed. Similar multiple cysts of small size are met with in obstinately sterile heifers, and the evidence justifies the suspicion that this type of cystic disease of the ovaries is congenital. Many cow ovaries have some small cysts of this character, without notable interference with fertility, but when the cysts are present in great numbers obstinate sterility is apparently the rule, and is not subject to remedy in the present state of knowledge.

Upon the advent of sexual maturity, if the animal is physiologic and uniparous, a single Graffian follicle and its ovum mature. The follicle becomes distended with fluid, and in the cow, and some other species, protrudes beyond the general contour of the ovary as a hemispherical elevation. When the follicle has reached maturity, estrum appears, followed by copulation and conception. The phenomena associated with conception are best studied in the cow, in which estrum is brief and sharply defined, and the ovaries and uterus are readily and authentically palpated per rectum. The ripe Graffian follicle and the corpus luteum are readily identified.

Healthy female domestic animals, of breeding age and non-pregnant, physiologically exhibit, in regular cycles, pronounced signs of sexual desire, designated estrum or heat. As already stated on page 122 the mare is commonly in estrum at the 8th or 9th day postpartum. Thereafter if healthy and non-pregnant she is in estrum at intervals of about 21 days during the breeding season. When highly fed and carefully handled the estrum may remain comparatively regular throughout the year.

The dairy cow is ordinarily in estrum at 30 to 40 days postpartum, the period being apparently abbreviated by the customary immediate removal of the calf. It appears upon the little study accorded that if the calf is permitted to run free with the cow and suckle regularly,



FIG. 41. Multiple Ovarian Cysts in Calves, Enlarged from Fig. 5.

A, 2, Ovary from normal calf; *B*, 2, & *C*, 2, gigantic ovaries consisting largely of cysts and hemorrhages, from one fetus dead at birth and one heifer calf dead at thirty days; *B*, and *C*, 3, large uteri with abnormal uterine caruncles. *(Cornell Veterinarian)*

the postpartum interval prior to the advent of estrum is prolonged somewhat as in woman, though for a much shorter period. Pathologically the cow may be in heat at 8 to 10 days postpartum.

The estrual cycle is disturbed by disease, malnutrition and other depressing factors. Male domestic animals have no intervals of total abeyance of sexual desire. If the male regularly consorts with the

female, as in pasture breeding, he makes no sexual advances toward her during the intervals between heat periods. If the male is regularly kept apart from the female, and is suddenly brought into her presence, he attempts coitus although the female is not in estrum. Such attempts at coitus fail, except the female be forcibly restrained by the breeder.

Physiologically, estrum is linked with the maturation of a follicle, and persists until the follicle ruptures and discharges the ovum. The duration of heat varies according to species and the health of the individual. It is shortest and most clearly defined in ruminants. The duration of heat constitutes an important index to the sexual health of the individual. The heat period is shortest in the healthiest individuals of the species. In such healthy animals the maturation of the follicle is rapid and its rupture prompt. At the opposite extreme is observed the pathological follicle associated with nymphomania, especially frequent and serious in high-class dairy cows. So far as known, the ovum within the follicle of the ovary of the nymphomaniac animal does not mature, but disappears, and is supplanted by a cystic degeneration of the follicle. The follicular liquid becomes greatly increased and the walls become so dense and resistant that the cyst may not rupture for months.

Estrum is generally longer in multipara than in unipara. The cause is not fully known. It is believed, with apparently good reason, to be largely due to the numerous follicles failing to mature simultaneously. This belief is made to explain the common observation that the bitch, permitted to copulate at successive intervals with different males, may give birth to a litter of pups showing clearly the varied parentage.

The doe rabbit, the cat and some other small animals are said not to ovulate except after coitus. Should the copulation fail to occur, the estrum is prolonged, and the follicles gradually atrophy and disappear; no corpora lutea form. If the female rabbit copulates with a vasectomized male, the follicles rupture, corpora lutea develop, and pseudo-pregnancy follows. In such cases the uterus enlarges, the mammae develop and the general symptoms of pregnancy appear. Pseudo-pregnancy also occurs in the bitch, usually, if not always, following unsuccessful coitus.

In natural, or pasture breeding, coitus regular occurs promptly upon the advent of heat. It is not definitely known, but the inference seems justified, that early coitus hastens the rupture of the follicle, shortens the duration of heat and favors conception.

The state of nutrition and vigor affects the ripening of the follicles

and the occurrence of estrum. Writers upon sterility frequently emphasize the absence of estrum, or refer to a very weak expression of sexual desire as dumb estrum. The phenomenon is often attributed to the want of certain vitamins or hormones. While this may be immediately true, clinical observations indicate that the phenomenon has a deeper basis and that the hope for overcoming the defect involves the search for, and recognition of the primitive factor which denied to the animal the necessary vitamins or hormones. I once had a small group of virgin heifers for contagious abortion experiments. The heifers were reasonably mature and in fair physical condition, but failed entirely to come in heat. It developed that the ration fed the heifers consisted of a scant allowance of inferior hay, in which vitamins were scarce. A moderate addition of grain was followed promptly by heat and conception.

In a second instance my small group of experiment animals was unavoidably in charge of an incompetent caretaker for some months. Plenty of hay and grain were supplied and apparently fed in due amount. The animals were poor and did not show estrum. A change in caretaker, with no known change in quantity or quality of food, was followed promptly by heat and fertility. So far as could be determined, the difficulty had been due to water starvation, in the midst of abundant vitamins. The value, if any, of these observations is that, if ample food of good quality is provided, estrum occurs without the artificial administration of vitamins. The fundamental value of vitamins is not questioned. It is not known that, in the two cases cited, the artificial administration of vitamins or hormones might not have caused estrum. This could not have served as an acceptable substitute for prudent feeding and care. Had conception occurred under such artificial stimulus, the pregnancy could not have been physiologically maintained.

The final proof of estrum is the voluntary acceptance of the male in coitus. Sexual desire is indicated by the female, in the absence of the male, by various signs. Cows especially, and to some extent the females of other species, attempt to mount their companions, or permit other animals to mount them in mimic copulation. The confined cow usually signalizes the advent of heat by an abnormal amount of bellowing. The voice is modified, the pitch being lowered. Pathologically, in nymphomania, the pitch of the voice is lowered to that of the bull, so that the affected cows are commonly designated "bullers."

During heat the entire reproductive system is engorged. The vulva is regularly enlarged and there is discharged from it an abundance of thin, transparent, lubricant mucus. The estrual mucus of the cow

is generally quite abundant, hangs in long strings from the vulva, and adheres to the vulva and tail. Some animals show relaxation of the sacro-sciatic ligaments, faintly suggestive of the relaxation of approaching parturition or of nymphomania. Possibly the relaxation is on the borderline of nymphomania.

The female in heat is hypersensitive and erratic in behavior. She is less readily led or driven. As the estrum approaches the pathological, the animal becomes more and more difficult of handling. Some mares, on the borderline of pathology, become vicious, and kick violently at attendants or mates. It is spoken of as *nymphomania* but is essentially different from the typical nymphomania of the cow, with cystic degeneration of the Graffian follicles: these mares frequently conceive if bred, indicating that the follicle and its contained ovum are not profoundly defective. If not bred, the outbursts may increase in intensity and the mare become an outlaw, possibly redeemable by ovariectomy.

Clinical observations reveal other important evidences of heat. The mare commonly urinates frequently, especially in the presence of other horses, and in doing so opens the vulva frequently, exposing the erected clitoris. Some animals show mammary engorgement. Pathologically the female in heat may lactate. I have observed this in one sterile heifer, which yielded a gallon of milk daily. I have observed profuse lactation in a mare mule during estrum, there being incontinence of the milk, which flowed down the inside the thighs, keeping them repulsively dirty. Heifers suffering from cystic ovaries (nymphomania) occasionally lactate. W. W. Williams has observed nymphomania in a heifer less than one year old, with definite lactation. Her growth was arrested, she assumed the general appearance of an adult and soon became senile in appearance.

The introduction of the hand into the vagina of the mare or cow reveals an abundance of thin, colorless, highly lubricant mucus. The cervical canal is more or less dilated.

Rectal palpation in the cow reveals the presence of a ripe ovisac about 1 cm. in diameter, fluctuant and readily ruptured, calling for care in palpation if it is desired to breed the animal during the period involved. The uterus is engorged and erected.

Histological changes of great technical interest occur in the vulvovaginal epithelium. The facts have not yet acquired definite clinical value.

The horse breeder supplements the foregoing evidences of estrum by the process of *trying* or *teasing*. The stallion and mare are brought into close contact, with a barrier between them, and the stallion permitted to make sexual advances without opportunity for coitus.

Chapter XII

THE ARTIFICIAL INDUCTION OF ESTRUM

The Dislodgment of the Corpus Luteum

PHYSIOLOGICALLY, estrum is held in abeyance by the presence in the ovary of a corpus luteum of pregnancy or of heat and the heat cycle may be interrupted and estrum induced by its dislodgment. When occasion arises for the interruption of the heat cycle in the non-pregnant heifer or cow, the dislodgment of the corpus luteum is usually followed by heat in 3 to 4 days. If the corpus luteum be dislodged from the ovary of a pregnant cow, heat also follows in 3 to 5 days and the fetus is expelled from the uterus. The abortion is not apparently due to the absence of the corpus luteum, but rather to the sudden shock when the entire organ is removed at once. The corpus luteum of pregnancy of the cow sometimes atrophies and heat follows, but usually the fetus is not expelled.

The atrophy of the corpus luteum is held in abeyance and estrum usually excluded when a large static foreign body, such as the typical bovine mummy, is present or when the uterus is filled with pus or other liquid. Limited observation apparently indicates that when the entire endometrium has been destroyed, heat does not occur.

While it is assumed that the removal of the corpus luteum exerts similar influence in all species, anatomical considerations largely limit its application to cows. The surgical removal of the corpus luteum may be considered:

(a) When several cows in a dairy herd are simultaneously in estrum, and the service of all would cause too great a sexual strain upon the sire, some of the cows may be allowed to go unbred, and their estrual cycle interrupted later by the dislodgment of the corpus luteum at an advantageous date. Artificial insemination may be used instead.

(b) When the uterus contains a mummy, pus, lymph or other pathological substance, the corpus luteum may be profitably removed sometimes as a therapeutic operation, to cause estrum and the emptying of the uterus. This will be considered at greater length when dealing with uterine diseases.

(c) In order to terminate an undesired pregnancy. When a grossly immature heifer becomes pregnant through accident, the early ex-

pression of the corpus luteum is usually followed promptly by abortion, with less danger to the heifer's future value than permitting the pregnancy to continue. If a valued cow accidentally becomes pregnant by an inferior bull, or one of a different breed, the pregnancy may be arrested, if desired. This should only be done when the pregnancy is recognized early. After three or four months of pregnancy have elapsed it is safer and better to permit the gestation to run its course.

(d) The artificial induction of estrum has been advocated by some writers as a general cure for sterility, largely regardless of the fundamental cause. The recommendation rests upon an extremely questionable philosophy of therapeutics.

The dislodgment of the corpus luteum of the cow is effected rectovaginally. The operator introduces one hand into the rectum, locates the cervix and uterine cornua, and follows these forward and downward, finally identifying the ovary attached to the apex of the cornu by the oviduct or Fallopian tube. The ovary is grasped and drawn backward and upward, above the roof of the vagina. The other hand is then passed into the vagina. The vaginal walls are highly resistant and will withstand much compression when due care is used. The corpus luteum, having been clearly identified, is to be encompassed by the thumb and one or two fingers of the hand within the vagina, and pressure applied approximately on the line between the corpus luteum and the principal tissues of the ovary. When the yellow body becomes separated from its crater, it drops into the operator's hand. While the compression is being applied, the ovary is steadied in its location by the hand within the rectum. The compression of the ovary may be carried out by an assistant, the operator guiding and directing the compression with the aid of his hand in the rectum. Under reasonable care there is no material danger of serious injury to the rectum, vagina or ovary.

Sometimes the dislodgment of the corpus luteum as a therapeutic measure is followed by serious or fatal hemorrhage. It has been advised that hemorrhage be prevented by digital compression of the ovary for a few minutes after the dislodgment of the corpus luteum. So far as may be determined, the hemorrhage is not due to the rupture of important vessels. Thus, in the case reported by Udall and Williams[203] and illustrated in Fig. 42, no evidence was found that the fatal hemorrhage was due to the wounding of the vessels. Instead it seems that the dislodgment of the corpus luteum caused a profound shock which destroyed the vasomotor control of the utero-ovarian and uterine arteries. It appears that the compression of the ruptured

capillary ends for a brief period could not possibly have prevented the hemorrhage. The history of the cow revealed pathologic behavior in the estrual cycle over a long period. What may, in a sense, be designated as spontaneous hemorrhage from the genital organs, is not a rare phenomenon in cows. It sometimes occurs that very pro-



FIG. 42. Fatal Hemorrhage Following the Dislodgment of the Corpus Luteum. The Uterus Laid Open and Viewed Dorsally.

1, Endometrium of right cornu; 2, endometrium of left cornu; 3, right uterine artery; 3′, left uterine artery; 4, right utero-ovarian artery; 4′, left utero-ovarian artery; 5, 5, right ovary from which corpus luteum was dislodged; 6, pavilion of right oviduct; 7, right mesosalpinx; 7′, left mesosalpinx; 8, blood coagulum in right uterine artery; 9, apex of right cornu; 9′, apex of left uterine cornu. *(Cornell Veterinarian)*

fuse hemorrhage into the peritoneal cavity follows immediately upon ovulation, and the veterinarian recognizes, upon rectal palpation, a large hematoma suspended from the ovary. During an apparently physiologic gestation there not infrequently occurs a vast interplacental hemorrhage from the uterine caruncles; the ovum becomes completely detached, the blood desiccates and a mummified fetus results, as will later be described. Again after parturition, profuse, or

even fatal intrauterine hemorrhage sometimes occurs, as noted further in Chapter XXXIV. In none of these have there occurred recognizable anatomical injuries to arteries, veins or capillaries.

When the uterus contains a mummified fetus, an old-standing collection of pus or other pathologic content, the corpus luteum may have sunk so far toward the center of the ovary or have acquired a capsule so highly resistant that the yellow body cannot be dislodged by digital pressure without great peril to the integrity of the vaginal wall or to the ovarian tissues. In such cases the capsule of the corpus luteum should be incised with the aid of the ovarian scalpel, Fig. 43. The operation proceeds in the same manner as for dislodgment until the ovary has been grasped per vaginam. The most



FIG. 43. Ovarian Scalpel.

superficial face of the corpus luteum is brought against the vaginal wall, the grasping thumb and finger being in front of the ovary. The guard of the scalpel is set free so that it will automatically glide backward when the blade is pressed into the vaginal wall. The scalpel is then introduced along the volar surface of the hand into the vagina and pushed through the vagina into the capsule of the corpus luteum. The scalpel is then withdrawn and the corpus luteum is readily expressed. I have not known hemorrhage to follow the operation.

The artificial induction of estrum is sometimes highly desirable in severe pyometra, in which case it is sometimes impossible to reach the ovary per rectum. The dislodgment of the corpus luteum is then best accomplished by laparotomy. This is more fully described under pyometra.

The wholesale dislodgment of corpora lutea, as is sometimes practiced as a general cure for sterility, is without known justification. It is perilous to induce estrum for the immediate breeding of a cow or other female when she is not in a proper state for enduring pregnancy. It is not mere pregnancy which the breeder desires, but healthy pregnancy which will lead to the production of valuable young.

Experimental evidence has been submitted apparently showing that estrum may be induced by the administration of vitamin E or other vitamins. In effect, this is saying that estrum may be induced by feeding, which is not open to question. The question involved is

the value of artificially prepared or extracted vitamins instead of supplying adequate food carrying the vitamins in their natural form.

Thus in the group of heifers mentioned above, fed exclusively on the poor quality of hay and failing to come in heat, the addition of oats to their diet acted promptly. It is quite possible that the administration of vitamin E without the associated food elements in the oats would have caused estrum and the animals may have conceived. But the state of nutrition was such that, had they become pregnant, the pregnancy would not have been safe.

Similarly it has been technically shown that estrum may be induced by the administration of hormones. Some experiments have been recorded in which hormones have been used to induce estrum in cows that failed to come in heat naturally. The estrum followed and in some instances the cow became pregnant. But so far as I have been able to learn, the pregnancy was a failure. The question recurs of the value of artificially induced estrum as a remedy for non-estrual sterility. If the administration of a hormone may induce heat in an otherwise healthy animal, its value needs be recognized. If it merely induces heat without affecting the basic health of the animal it would apparently be wholly inadvisable; it would be far better to identify the fundamental cause of the failure to come in estrum and remove it.

Chapter XIII

COPULATION—COITUS

COITUS in domestic animals occurs under two distinct plans, commonly designated as pasture, and hand breeding. Under the former plan the sire regularly consorts with his group of females; in hand breeding the sire is kept apart from the females and brought into contact only when it is desired to breed an individual. In pasture breeding the male naturally recognizes estrum at its beginning and service follows promptly. The copulation is often repeated several times in rapid succession. Coital injuries to either sex are rare in natural coitus. It is not often that dangerous inequalities in size exist: as a rule the male and female belong to the same breed, or to breeds of similar size. The greatest variations in size occur in horses and dogs. A heavy draft horse may weigh 2000 to 2500 lbs., and some of the smaller ponies little more than one-tenth as much. The extremes in weight are much greater among dogs. Attempts at coitus between such extremes involves great danger and the act usually fails. The mounting of a small pony mare by a large stallion tends to break the pony down, with probability of fractures or other serious injuries. A pony mare will, however, safely copulate ordinarily with a stallion of twice her weight. Similar conditions apply to dogs, with the added peril of *hanging fast*. The dog having an extensive penial bone with a highly erectile glans and abrupt base, may be unable to withdraw the penis from the copulatory canal of a distinctly smaller female. This may prove especially dangerous in dogs running free, with probabilities of violent fighting when a number congregate, as is usual.

The young of highly-bred horses and cattle develop sexual desire when they have reached but one-fourth or one-third of their adult size. In addition to the disastrous effects of pregnancy upon very immature females, as already described, serious physical injury to the immature individual is constantly threatened. Thus I observed a six-months heifer served by a mature bull. This resulted in a fractured pelvis, and the heifer was ruined.

When a regularly confined male accidentally escapes and comes in contact with young females of the species, he attempts coitus whether the young is in heat or not. In one instance a mature stallion mounted a suckling filly, broke her down and fractured the pelvic girdle. She

survived, and, although she was a hopeless cripple, the owner had the erroneous idea that she might be a valuable breeder. I attended her in dystocia at the close of her first pregnancy and found a normal fetus in a normal presentation and position. When traction was applied to the fetus it paradoxically retreated from the vulva. The fracture of the pelvis had not healed. There had been displacement of the left sacro-iliac articulation with failure of the ligaments to reunite, and the fracture through the shaft of the right ilium which had failed to heal, as shown in Fig. 44. The non-united areas served

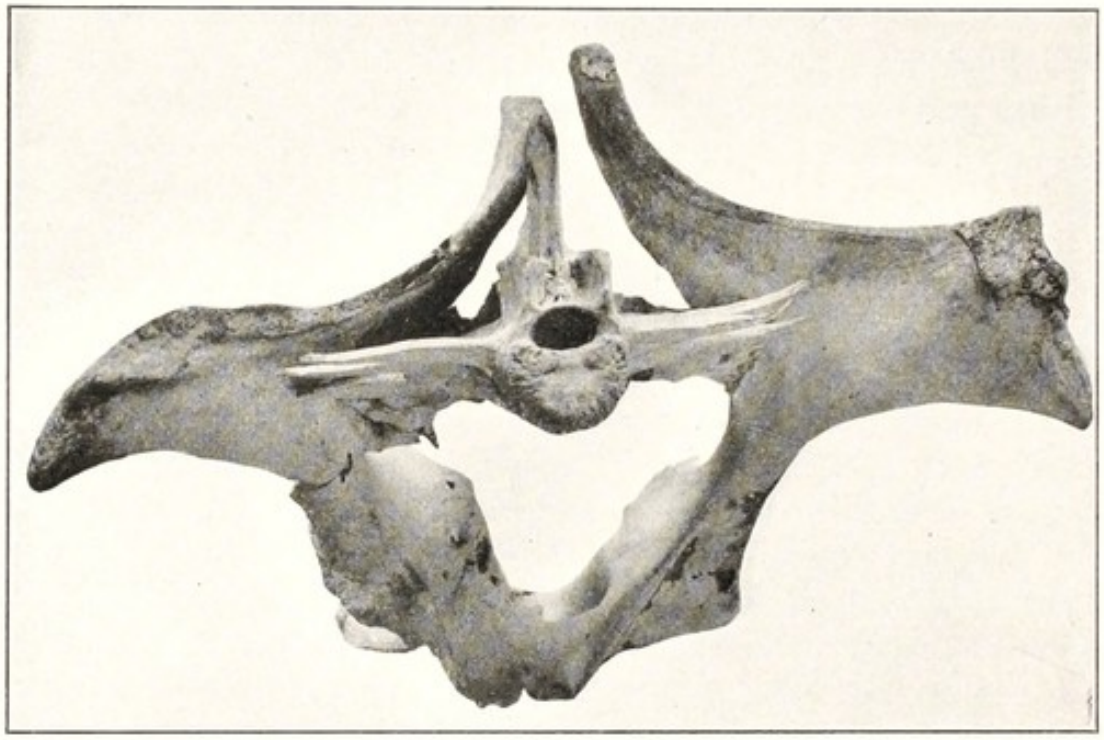


FIG. 44. Deformed Equine Pelvis, Fractured When a Foal, Due to Being Mounted by an Adult Stallion.

as hinges by which the pubis and left ilium move freely upon the left sacro-iliac joint and the unhealed fracture of the right iliac shaft. Hence, when traction was applied to the presenting parts of the fetus, the impact upon the pubic brim forced the ischia backwards and upwards toward the tail, so that the greater the traction, the smaller the birth canal.

Hand breeding has its advantages and perils. The mating is commonly restricted to a single service during a given heat, instead of several copulations in rapid succession, as in natural breeding and the sire is thus enabled to serve a larger group of females without excessive sexual load. It is the only method by which an authentic record of the date of conception and the duration of pregnancy can be

maintained. Some breeders permit a second coitus immediately, without known benefit. The practice lowers the reproductive power of the sire more than one-half. If the sire is expected to serve only a small unit of females, no known harm exists in the immediate repetition of coitus. Some horse breeders cause a second service to be made if the mare remains in heat for a period of five or six days after the first service. It is not authentically known that it does good or harm. The probabilities are that, if a mare continues in heat for a long period, it is a signal that she is in a pathologic state sexually, in which case conception to a second service is improbable and if it occurs, will usually be unsafe. If this view is correct, such repetition of coitus is inadvisable. Coitus is not a cure for sterility or for low fertility. The ideal should be to not breed the female until she is in sexual health, so that if mated with a healthy male a single service results in physiologic pregnancy.

Hand breeding is inferior to natural mating with regard to the early recognition of estrum and prompt coitus. The heat period of a healthy cow is often very brief, and may pass within a day. If closely confined, the recognition of heat calls for close attention of the dairyman. If the estrual cycle is regular and the breeder carefully records each heat period, he may well be on the alert at the date when estrum should be expected.

Hand breeding sometimes closely approaches rape, especially in mule breeding. The mare is *tried* or *teased* for the presence of estrum by means of a stallion. She is then placed in stocks and blindfolded and perhaps a twitch applied to her upper lip, after which the male ass copulates with her. When a mare or cow cannot be discovered in heat the owner sometimes causes her to be raped by the male, under the belief that the forced coitus will cause the female to promptly come into estrum. I know of no facts supporting the belief.

The Dangers of Coitus

Natural copulation between mature healthy and normal animals is not prominently associated with coital injuries. Under domestication heifers and fillies come in estrum when too immature to bear the weight of a mature male and if permitted to mate may be *broken down*. They suffer most frequently from broken pelvis.

When hand breeding is practiced, coital injuries become infinitely more probable than in natural breeding and the variety of injuries is innumerable.

Severe restraint is commonly applied in the breeding of thorough-

bred mares. The stallions are commonly regarded as highly valuable. The mares are very excitable and in many cases the recognition of estrum is not certain. This is especially true of the first estrum after foaling, when the fears of the mare for her young foal, and anger, may combine to mislead the breeder. Under such conditions the mare may behave much as if in heat. The subject has not been much studied. The probabilities are that careful investigation would reveal that rectal palpation of the uterus of the mare would authentically determine whether she is in heat. Or in most cases heat could be reliably determined if the stallion and mare were placed in adjoining enclosures with secure partition so that the sire would be safe from injury during efforts to come in contact with the mare. If in estrum, the mare will ultimately approach the stallion and clearly reveal the estrum.

Similar, but less forcible restraint is sometimes applied to cows by the dairyman. In one case a breeder thought to bring a free-martin into heat by rape. Later I found it suffering from extensive pelvic abscess. Some dairymen place small heifers in stocks so that when mounted by large bulls, they may not be broken down. When a heifer is not strong enough to stand up under the bull she is not fit to be bred.

Even when in estrum, a mare may, if unrestrained, kick violently when the stallion attempts to mount, with the danger of fracture of a limb. There is further danger that the kick may seriously injure the penis of the male. The sheath of the stallion is short and wide and the very large penis, tensely erected, protrudes from the sheath at full length, prior to mounting, and is thus freely exposed to injury. In contrast, the ruminant sheath is longer, and the penis is protruded chiefly through the elimination of the sigmoid flexure after the cow is mounted. Hence, the stallion, when jumping a fence in order to reach the mare in heat, is liable to serious injury to his protruding penis, while the bull, with his penis well within the sheath, crashes fences with comparative safety in an effort to copulate with neighboring cows in estrum.

The dangers of coital injuries to the stallion may be minimized by proper handling of both sexes, especially by having them under such control that, if an attempt is made by the mare to kick the sire, it is possible instantly to turn her head toward the stallion. As a rule a breeder relies upon restraining the mare by applying hobbles. The hobbles sometimes frighten or anger the mare and cause her to struggle violently, with danger of physical injury. Under such re-

straint, the mare is virtually helpless, and coitus occurs whether in estrum or not.

FALSE ENTRY

Harms[80] states that false coitus occurs in the mare, cow and sow, by which the penis of the male enters the rectum instead of the vagina. Apparently the accident is usually without peril in the cow or sow. In the mare it is highly dangerous and frequently results in rupture into the peritoneal cavity with prompt death. I have observed four cases, three of which ended immediately in death. The fourth case had caused rupture into the pelvic connective tissue with the formation of a large sac, which caused her death some years later from the impaction of feces in the diverticulum, producing fatal obstruction colic. The causes of this accident have not been clearly defined. The comparatively great size of the equine penis probably plays an important part. The rectum is much sacculated and is ordinarily filled with comparatively large and firm pellets of feces, not readily pushed aside. In some of my cases it appeared that an error had been made by the stallion manager and an effort made to breed a mare not in estrum. Hence it might be said that the fundamental cause lay in attempted rape upon a highly resistant mare. I have not known the accident to occur in natural coitus, where the stallion has constantly consorted with his harem of mares, and copulates only when definite heat is present. The vulva of the mare or cow may be abnormally near the horizontal if the abdomen is markedly pendulous or the superior commissure of the vulva has been lacerated during parturition. During coitus the penis tends to glide over such horizontal vulva and enter the rectum. By having the female stand with her posterior feet in a depression, the position of the vulva is improved.

The symptoms vary according to the injury. Harms says that the accident in the sow causes only unimportant tumefaction of the anus. The mare usually shows important symptoms immediately. If the rectal rupture is only into the periproctal connective tissue, there is some discharge of blood and feces and a definite expression of pain. Rectal palpation readily establishes the nature and extent of the injury. If the rupture is only into the pelvic connective tissue and the rectum is kept free from accumulations of feces by repeated enemata, recovery has been satisfactory in my experience and only slight sacculation of the rectal wall occurs. The enemata need be aseptic rather than antiseptic, because the periproctal connective tissue is very loose and antiseptics permeate it readily and cause

much irritation. Generally it is preferable to rely upon mechanical cleansing with physiological salt solution of approximately rectal temperature. If neglected, the sac becomes enlarged and tends to grow in size from the impaction of feces. At any time the fecal mass may become so great as to cause obstruction colic and death.

When the rupture penetrates the peritoneal cavity, feces immediately drop into it as soon as the penis has been withdrawn. This causes profund shock and irritation. No sooner has the stallion dismounted than the mare trembles, looks anxiously at her sides and seems on the verge of collapse. Some blood and feces escape from the anus. The pulse is rapid and extremely weak. The breathing is quickened and shallow and the abdominal muscles rigid. The diagnosis is easily made by rectal exploration, the hand passing readily through the rupture into the peritoneal cavity. Death within a few hours is inevitable.

The accident is readily preventable in the mare. Service should not be attempted if there is reason to believe that the mare is not normally in heat. It is the duty of the groom to see that the penis of the stallion enters the vulva. If the mare resists the coitus, the stallion should be withdrawn immediately and the breeding abandoned for the time. The owner of one of the mares which I attended brought legal action against the stallion owner for the death of the mare and the court decided in his favor.

LACERATION AND RUPTURE OF THE VAGINA

Laceration and rupture of the vagina of the mare by the penis of the stallion sometimes occurs. The size of the penis of the stallion does not always correspond to the size or weight of the animal. The penis is relatively larger in coarse, leggy animals. I attended one such stallion which caused lacerations of the vagina in essentially all mares which he served. In one case fatal rupture of the vagina occurred. The penis was so enormous in size as to be dangerous to mares of his own weight.

Immediately following coitus there are evidences of pain, expressed by straining, with probably some hemorrhage. This may be followed later by vulvar swelling and purulent discharge. Diagnosis should be made by manual exploration of the vagina or visually with the aid of a speculum.

If the wound penetrates the peritoneal cavity, fatal peritonitis is probable. The rupture, in such event, should be sutured and followed by douching the vagina with physiologic salt solution. If the lacera-

tions are not penetrant, the prognosis is good and demands mere cleansing of the vagina with the salt solution.

Prevention is readily attained by means of a roll or padded surcingle applied just in front of the sheath opening, the apparatus having ample diameter to prevent the penis from entering the vagina beyond a safe distance.

Occasionally a mare or cow suffers from constriction of the vagina due to adhesions from prior injuries, from severe vaginitis due to coital or parturient injuries or to imprudent douching with irritant antiseptics. When attempts are made to breed such animals there is peril to both sexes. In one instance coitus in a mare with vaginal adhesions resulted in vaginal prolapse with violent straining, which I could not control until complete chloral anaesthesia had been produced. Epidural anaesthesia would now be preferable. In the handling of vaginitis or serious injuries to the organ, any adhesions or constrictions should be noted, and if important, the female should be discarded as a breeder. Bovine twins and multiples, as heretofore mentioned, not infrequently have defective vaginae and functional ovaries. In some cases, as reported by Galli,[67] such animals come in heat and coitus results in rupture of the vagina and penetration of the peritoneal cavity. It is prudent that bovine twin or multiple females be examined before attempting to breed them.

THE ALLEGED RUPTURE OF THE BOVINE UTERUS AND BLADDER BY THE PENIS OF THE BULL

Rupture of the bovine uterus and urinary bladder by the penis of the bull has been reported. The reports seem to be wanting in authenticity. The anatomical features make such an accident extremely improbable, and suggest great care before accepting such reports. It is especially desirable that when such injury is discovered, the possibility of sadism by a sexual pervert be fully disproven.

Infrequent injuries from copulation are of endless variety. Some stallions viciously bite the neck or withers of the mare during coitus. It should be prevented by muzzling. Strangulated inguinal hernia occasionally develops in the stallion immediately after service. When violent symptoms of colic appear just after coitus, the veterinarian should at once search for hernia.

Sodomy and Sadism

Sodomy, or the carnal intercourse between man and female domestic animals is of scant interest in veterinary science because in the

larger animals no injury is caused and there are no specific venereal diseases of man transferable to domestic animals. With the moral and legal aspects the veterinarian is not commonly concerned. Sometimes important questions of diagnosis of injuries to small species of animals may arise. Guillebeau[68] relates an instance in which a series of hens died from attempted sodomy by a sex pervert. The veterinary problem offered was the recognition of the character of the mutilations and opening the way to the identification of the psychopathic man.

Sadism, so designated because of the notorious mutilation of women by the Count de Sade, is of great scientific and practical importance in veterinary medicine. The records of sadism are chiefly found in continental European veterinary literature, such as that of Guillebeau already mentioned, of Cadiot[22] and of Eggiman.[48]

Although sadism is rarely, if at all, reported in American veterinary literature, veterinarians occasionally relate cases personally. In one instance the vagina of a mare had been ruptured by the use of a broom handle, intestinal loops protruded and the mare succumbed. In another instance a renegade veterinarian, whether because of sex perversion or for other reasons, punctured the vaginae of 10 or more cows in one herd with a long iron rod, sharpened at one end, with a cross bar upon the other, which he placed against his shoulder. The mutilation was carried out under the pretext of dilating the cervix. The cows were rendered hopelessly sterile.

The published records of sadistic mutilations are largely confined to cows and goats. The lesions may involve only the mucosa of the rectum or vagina, they may penetrate the pelvic connective tissue, may traverse the urethra and rupture the bladder, or may penetrate the peritoneal cavity. The psychopathic individual may use a great variety of objects with which to cause the mutilations, largely the handle of some convenient stable tool like that of a broom or fork, or in some cases a small stick. The symptoms are as varied as the lesions. In non-penetrant lesions there usually follows swelling of the perineum and bloody discharges from anus or vulva. Penetrant wounds of the pelvic connective tissues result in painful tumefaction with obstruction in urination or defecation. Penetrant wounds of the peritoneum present the common symptoms of peritonitis, or, if large volumes of feces enter, there occur the usual symptoms of collapse.

The most important problem facing the veterinarian is the identification of the character of the mutilations. The character of the injuries is readily recognized by vaginal and rectal exploration. But the veterinarian is all too likely to be off his guard and make a false

diagnosis. When a psychopathic mutilates one animal he is quite likely to continue his activities. Accordingly a correct diagnosis is essential to the identification of the offender in order that effective measures may be taken to stop the losses. In some cases the mutilations have continued at intervals for months, involving a considerable number of animals. When the injuries are studied carefully, they cannot be attributed to coitus, they cannot well be due to accident, and they cannot be assigned to criminal action of a sane person. The offender is probably of evidently low mentality but may be of apparently sound mind, the sexual perversion being the only marked psychopathic trait, which is practiced in secret and difficult of detection.

The handling of the lesions calls for the application of the general rules of surgery. Penetrant wounds of the peritoneum are largely fatal. Some pregnant cows abort.

Chapter XIV

ARTIFICIAL INSEMINATION

THE artificial insemination of domestic mammals has been known for centuries. Schmaltz[178] states that in an Arabian book published in the fourteenth century it is related that an Arab stole semen from a famous stallion by inserting a pledget of cotton into the vagina of a recently bred mare, carried it home, inserted it into the vagina of his own mare and conception followed. Spallanzi recorded interesting experimentation in the field during the eighteenth century. Definite interest in artificial insemination, as an economically valuable factor in animal husbandry, was aroused by Ivanoff[100] in 1912 by the publication of his monograph upon the subject, with data indicating its practical use in animal husbandry in Russia.

Artificial insemination became especially popular in Russia possibly because of the communistic plan of animal husbandry which rendered it technically possible to inseminate large numbers of females from one sire. Accordingly, artificial insemination has had its widest application in Russia. Turkey established an institute for artificial insemination, which according to Beller[11] has resulted in its extensive use in that country.

The Russian workers were primarily endeavoring to extend the usefulness of a sire by dividing a given ejaculate into several parts and by artificial insemination to induce pregnancy in several or many females. The highly optimistic reports by some of the Russians awakened vivid imagination in other lands. Thus Miller[140] considers artificial insemination valuable in: (1) "transmitting semen obtained from valuable transmitting sires or strains to distant points; (2) extending the use of valuable breeding sires, through the artificial impregnation of more cows, in the same herd or in nearby herds, than could be impregnated by natural matings; (3) extending the use of sires that, because of age or crippled condition, are unable to make normal matings; and (4) preventing the spread of disease in herds served by co-operatively owned bulls, such as bull associations or rings, or in herds where the bull serves cows brought from surrounding farms."

The views of Miller are fairly representative of the general beliefs in this field. Considering the points as stated by Miller:

(1) It is technically possible under modern transportation methods to carry semen across continent and ocean and preserve the mo-

tility of the spermatozoa. The time element involved is the chief consideration. Miller and others have shown that the motility of spermatozoa may be retained by refrigeration for as long as six to eleven days. The presence of motile spermatozoa in semen does not establish its potency. Miller does not claim that bull spermatozoa have fertilizing power after 24 hours. Winters[273] records that a ewe was inseminated with semen which had been kept five days, pregnancy followed and a viable lamb was produced. How many ewes or other females have failed to conceive following the artificial use of semen held for five or more days, is not conspicuously recorded. Goetze[69] after a careful study of the problem at the Moscow institute and extensive researches at the Hannover veterinary school, concurred with the Russian authorities that it is highly desirable to perform the insemination within 30 minutes after having obtained the semen. An exception to this rule is made with bull semen, which may be held for as much as six hours. In sharp contrast, Hammond[79] states that "Recently bull sperm sent by air post from the United States has arrived in *active condition* in the Argentine." The exact point of origin or destination is not stated; the distance covered was inferentially between 5000 and 7000 miles, requiring a minimum of about 36 hours. Hammond fails to define "active condition" or to say that conception resulted. If calves were born, their quality was not recorded.

It is not definitely known for how long a period the spermatozoa remain vigorous in the ampullae of the vasa defferentia, from which region it is assumed the sperms are ejaculated during coitus. They are being rapidly formed, and excreted from the testes, and presumably disintegrate and disappear when halted in the ampullae pending coitus.

When spermatozoa leave the vasa defferentia they promptly undergo deterioration, whether deposited in the female genital tract or placed in a thermos bottle, and the longer they remain outside their physiological reservoir, the lower their vigor and potency to fertilize. The same rule must apply to spermatozoa after ejaculation as before. The fertility and the safety of any pregnancies caused by them, are dependent upon the ratio of morphologically and physiologically perfect sperms. As the spermatozoa decline in motility, they decline in fertility.

Generally in the United States of America, breeding horses and cattle of both sexes are readily transportable by automobile truck over distances of 100 to 200 miles, and within such limits breed satisfactorily. This is probably quite as economical as the transportation

of semen if expert services are required. It has been fantastically claimed, without the submission of convincing evidence, that the transportation of breeding males, especially of bulls, by truck or railway, causes testicular degeneration and sterility. In one instance a young bull, with extreme testicular hypoplasia, was shipped from Pennsylvania to California. As he was absolutely sterile, the buyer brought action against the seller. The latter contended that he had advised that the bull be shipped by express, instead of by freight, and that the vibration of the freight car caused the testicular hypoplasia and sterility. A politically prominent veterinarian testified accordingly and the seller won the verdict. Innumerable breeding males are constantly being transported by truck, train and boat, over land and sea, in the course of commerce or for showing at fairs. Not infrequently sterile young bulls are sold and arrive at their destination sterile. Bull 2 in Table II was shipped by rail 2000 miles and then by boat another 2000 miles. The table shows that he caused much loss in the herd, but it would require a spectacular flight of the imagination to attribute the abscess of his seminal vesicles, as shown in Fig. 138, to car vibration or to seasickness, when it is known that he was bred in a herd where genital disease existed in important degree. The five bulls serving the females in Table X, and the bulls in Table IV were all transported about 5000 miles by land and sea, and no known injury to their fertility resulted.

(2) The possibility of increasing the number of females which a desirable sire may fertilize, constitutes the chief, if not the sole important reason for artificial insemination. It is the general consensus of opinion of leading observers that a single ejaculate of a stallion or bull may be used for the artificial insemination of three, four, or five females, without recognizable lowering of the ratio of conception. The Russian workers, and Professor Goetze[69] maintain that the maximum success is to be had by conducting the insemination within 30 minutes after the semen has been obtained. This requires that the requisite number of females, properly in heat, be directly available. Without submitting substantial evidence, some enthusiasts claim that the number of females which may be fertilized from a single ejaculate is virtually unlimited. Winters *et al*[273] advocate the insemination of as many as twenty females from a single ejaculate. Others advocate as high as forty females. Walton[217] says "At the present moment there are no technical obstacles to prevent 1200-1500 calves being sired by a single bull during a 60 day breeding season"!

When a continent, or continents, become extremely devastated by

war, as in the present global conflict, artificial insemination may well acquire far greater economic importance, during the period of rehabilitation, than has heretofore appeared possible to most observers. Many nations, heretofore largely dependent upon domestic animals for food and labor will inevitably be almost destitute of breeding animals. Those nations not directly affected by the devastation naturally control the number of breeding males within their economic needs. They will be able to supply a relatively greater number of females to the needy people. This will quite certainly render artificial insemination a highly valuable procedure. It may prove far more expensive and less efficient than natural breeding while rendering it possible to increase the rate of reproduction until an ample supply of satisfactory sires becomes available.

Recently Cates[28] has excelled prior advocacy of artificial insemination. He states that in many southern areas the beef cattle of European ancestry fail to prosper because of the intense heat, whereas Brahmins (zebus) do splendidly. However the bulls of this breed are exceedingly expensive, but thanks to artificial insemination, one bull may be caused to answer for thousands of cows. He submits no data upon the cost of catching range beef cows and applying artificial insemination. Take, for example, the herd of 6,000 or 7,000 cows mentioned in Chapter XX, scattered over an uneven terrain of many square miles. It would require much labor by mounted men to find the cows which were in heat. Once recognized, the artificial insemination could only be performed by driving the cow into a chute, or by lassoing and casting in the open.

In order to cast and hold the cow, two expert mounted men would be necessary and another two mounted men would be needed for the insemination. If instead it were planned to confine the cow in a chute it would be necessary to drive her one to ten or more miles which would require at least four expert mounted men. Anyone who has had experience with driving a cow in estrum, with a young calf at her side, will appreciate the task. The entire herd would be kept in a constant uproar, day in and day out, month after month, with accidents unnumbered to cows and calves. Artificial insemination, by any technic yet announced, is inapplicable to range beef cows.

The estimates seem to be based upon spectacular hypotheses. Technically but one spermatozoon directly participates in the fertilization of an ovum. Hence, fantastically, if the portion of semen used in artificial insemination contains one perfect spermatozoon which may contact the ovum, that suffices. There are about 25,000,000 dairy cows in the United States of America, and it is reliably esti-

mated that there are at least 5,000,000,000 spermatozoa in a single ejaculate by a healthy, adult bull, or a minimum of 200 sperms for each milk cow in the nation. It seems undesirable to theorize too widely. Observations by conservative students in the field apparently show that a single ejaculate may be divided into four or five parts for artificial insemination without recognizable lowering of fertility. Further careful research may substantially alter this view.

The greater the number of females it is sought to inseminate from a single ejaculate, the more the semen must be damaged by handling with inevitable exposure to light, unnatural temperature, mechanical and chemical insult. It has been shown that pregnancy sometimes occurs after the use of semen held for five or more days: it has not yet been told how many failures have occurred for each success. The holding of the semen provides a conflicting problem. Aseptic semen is virtually impossible to obtain and when kept at *room* temperature, or at the temperature of the vasa defferentia or of the vagina or uterus, it quickly undergoes putrefaction. Consequently refrigeration is employed, which suspends motility and, it would seem, inevitably lowers the vigor of the male cells parallel to the duration of the refrigeration. The rapidity of the deterioration of the sperms is unknown, although it is known that some retain their fertilizing power for five or more days.

The necessity for the extension of the reproductive capacity of a great sire has been grossly exaggerated. The idea is in conflict with one of the most valuable principles of progressive breeding. Ideally when a great sire is mated with a female of equal quality, the progeny is of equal quality with the sire; when this fails, the breeding is unsuccessful. Accordingly the great sire should promptly beget abundant numbers of meritorious sons and grandsons. In normal countries there is no scarcity of satisfactory sires. Richter[169] states that there are plenty of good sires in Germany and hence no need for artificial insemination. Occasionally one encounters the statement that during the World War the supply of sires was greatly depleted. It has not been explained why the male domestic animals should be depleted by war more than the females.

It has not yet been practically demonstrated that extensive artificial insemination is more economical than the natural method of breeding. When a dairyman discovers simultaneously three or four of his cows in heat, and he desires to mate them all with one sire, (1) he may cause the bull to serve all the cows in one day; (2) he may postpone the mating of one or more of them; or, (3) he may resort to artificial insemination. My belief is that artificial insemination is

preferable to causing more than two copulations in one day. In such case the insemination would proceed in the simplest manner. One cow would be served by the bull and portions of the semen removed from the vagina and immediately transferred without cost of time or money.

(3) It is asserted that artificial insemination may render available the breeding powers of a sire incapable, because of age, disease or deformity, of copulating with a female. Such a sire inevitably loses

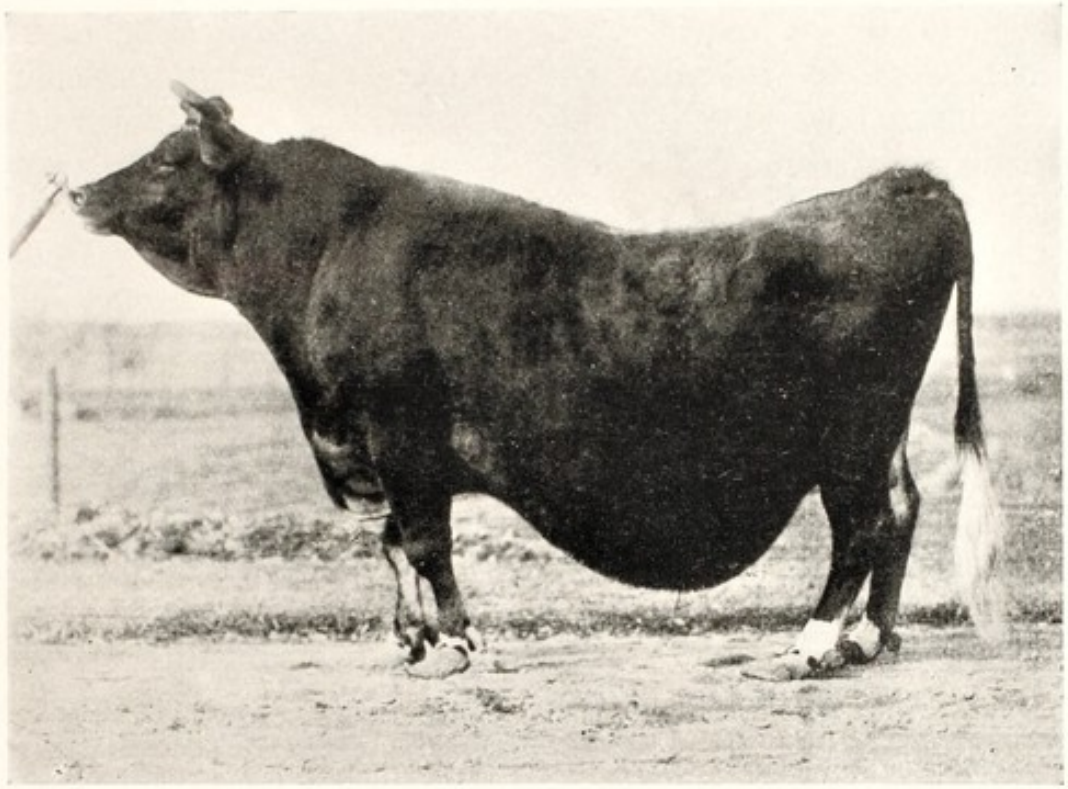


FIG. 45. Ventral Hernia in a Bull, Preventing Coitus.

vigor because unable to take necessary physical exercise. No matter how great a sire, when a male becomes too old or decrepit to copulate, he should be discarded. If a bull acquires a ventral hernia like that shown in Fig. 45, he loses his general vigor and any ova artificially fertilized by his weak spermatozoa would produce embryos or fetuses subnormal in vigor. In the case of the fashionably bred bull, mentioned on page 35 with congenital (hereditary) umbilical hernia which barred coitus, his spermatozoa were presumably abundant and vigorous. Recently a highly prized bull in a state agricultural college became unable to copulate because of articular disease in a posterior limb. The animal husbandman caused the veterinarian to resort to artificial insemination in order to retain the great (?) value of the sire. After about thirty inseminations, one calf of questionable value was obtained. Naturally no official report of the occurrence has ap-

peared or is contemplated, because it might detract from the value of artificial insemination from crippled bulls. Winters *et al*[273] relate that having a bull too monstrously short legged to serve a cow, they obtained semen from him by masturbation and bred his herd of cows successfully. Just how the pregnancies terminated is not told and no evidence is submitted to show that the calves were of value, if any were born. As a rule there is no marked want of monsters without resorting to them as sires.

(4) The claim that artificial insemination may be of material value in the direct control of coital infections should be rejected upon the available evidence. Beller[11] relates that at the station in Turkey mares have become pregnant by artificial insemination which had long been sterile under natural breeding. That is not rare in animals bred naturally: they remain sterile for a long time and then become pregnant and probably abort. Beller does not state that the semen which produced pregnancy was from the same stallion which failed to fertilize the mares by ordinary service.

The claim that artificial insemination is competent to control coital infection is amply illustrated from various angles. Bang[8] cited the observation of Poulsen, one of whose clients had 16 cows, 7 of which were bred to a bull in a herd where abortion prevailed. All 7 aborted, and had retained afterbirths and one died. The other 9 cows, bred to a different bull, all dropped healthy calves. The only logical conclusion is, that had each ejaculate of the diseased bull been divided into ten portions and used for artificial insemination, there would probably have been 70 abortions, 70 retained afterbirths and 10 dead cows. It is quite immaterial whether Bang was, or was not correct in attributing the disaster to the then recently discovered *Bacillus abortus;* whether some other infection existed in the semen of the bull; or whether his sperms were so defective and weak that the fertilized ova were non-viable, and disease forces wended their way to them as vultures to carrion. Clinically the result is the same.

Richter,[169] Fuchs,[66] and others maintain that the trichomonad infection of cattle may be controlled by artificial insemination. Küst[113] reports that by resorting to artificial insemination, trichomonad disease was effectively controlled in a group of about 500 cows in an infected commune, and that about 75% of the cows conceived at the first service and calved physiologically. Küst fails to say that any of the bulls or cows were suffering from, or had been infected with trichomonads and submits no reason for assuming that natural breeding would have caused disease. He mentions no abortions, retained afterbirths or other pathological phenomena and

leaves the reader to consider the reproduction idyllic.

The bulls responsible for the outbreaks of trichomonad disease described by Cameron, Fincher and Gilman,[23] and by Walsh, McNutt and Murray[216] entered the herds where they caused great losses, after they had been pronounced healthy by leading veterinarians. The scourge spread disaster because it passed unrecognized over a long period of time. The use of the semen from the diseased bulls before or after the diagnosis had been made would only have multiplied the disaster by the number of portions into which each ejaculate was divided.

Hammond[77] advocates artificial insemination for the control of dourine and bovine contagious abortion. He submits no evidence showing or tending to show that an outbreak of either disease has been so controlled. The outbreak of dourine in Illinois was under control within six months after official recognition by the state; the outbreaks of dourine upon the Rocky Mountain plains among wild range horses could not readily have been controlled by artificial insemination because of the expense of catching the diseased animals. Hammond also states that artificial insemination has been used in the United States for the cure of sterility of mares, but fails to designate the type of genital disease cured and cites no authority regarding the plan. About fifty years ago there was much enthusiasm over a scheme for curing sterility in valuable trotting mares by the use of an *impregnator* consisting of a heavy rubber tube so constructed that, placed in the cervix of the mare, it was expected it would remain in position during copulation and permit the stallion to ejaculate his semen directly into the uterine cavity. This would eliminate any cervical barrier to the migration of the spermatozoa, and nonmotile spermatozoa could thus be ejaculated into the uterus. Great claims for its efficiency were reported and some foals thus generated won races. The actual character of the disease, if any were present, was not revealed. Similarly there was invented and exploited an inseminating syringe with which the operator could pick up the semen from the vagina just after coitus and inject it directly into the uterus through the cervical canal. As late as 1911, Lewis of the Oklahoma Agricultural College advocated this method for the cure of sterility in mares of an unknown etiology and also declared that several mares might thus be impregnated from a single copulation. No data of results, either in the cure of sterility or from multiple impregnation from a single coitus are submitted.

There does not exist, at present, any known justification for attempting the control of any coital infection by means of artificial

insemination. If the male is infected, artificial insemination can only multiply the spread of the disease according to the number of females inseminated: if the female is infected, causing her to become pregnant by natural or artificial means is indefensible, and prolongs and intensifies the infection.

It is barely possible that artificial insemination may be justified in some types of cicatricial abnormalities of the cervix resulting from parturient lacerations after any infection has abated. But parturient lacerations rarely occur in healthy cows or other females. If utero-cervical disease is present, or if the genital system is hypoplastic, causing dystocia, artificial insemination can not go back beyond the pregnancy and repair the basic defect.

It is highly essential to dissociate conception and economic reproduction. The value of pregnancy is grossly overemphasized. If by this or that method a large ratio of females involved become pregnant, the plan is thereupon declared a success. Others pronounce the attainment of success when a large ratio of a group of females give birth to viable young. If the reader will critically study Table I it becomes obvious that, had the birth of the eighteen heifers been prevented by the sterility of their dams, or had all been aborted, the herd would have been richer and would have cost the taxpayers of the state far less. A viable calf may be either an asset or a liability. Artificial insemination of the eighteen dams of the heifers could not have made them valuable, and the artificial insemination of the heifers themselves could not have converted them from a liability into an asset.

The semen to be used for artificial insemination may be variously procured. 1. It may be obtained from the vagina of the female immediately following coitus. In the mare and cow the semen may be dipped from the vagina with the hand. In all animals a vaginal speculum may be applied and the semen collected with a syringe or other instrument. Some recommend that metal instruments should not be used unless well covered with paraffin so that the spermatozoa may not be injured by metallic contact. 2. Semen may be obtained from the stallion by applying a condom to the penis before he mounts the mare. 3. Various types of artificial vaginae have been devised for the collection of semen from the bull and other males. The bull is caused to mount the cow. When the penis is being protruded an assistant presses against the side of the sheath, deflects the penis laterally and causes it to enter the artificial vagina held at the side of the cow. 4. A dummy female is constructed and covered by the skin of an individual of the species. The dummy is fitted, with an

artificial vagina, into which the male ejaculates the semen. 5. Some males may be masturbated. A large ratio of bulls will ejaculate semen if the seminal vesicles and ampullae of the vasa defferentia be massaged per rectum. An attendant stands ready to catch the semen when expelled. In other species the massage may be applied to the prostate gland or to the penis. It does not always succeed.

The ultimate effects upon the male of the artificial methods for procuring semen have not been recorded. Under methods 3 and 4 it is unofficially known that penial injuries sometimes follow, but such injuries are not being officially published at present.

When semen is obtained from the vagina after natural coitus, the possibility of pollution from the vaginal contents of the female is present. If the female be healthy, the danger does not exist. The removal of the semen from the vagina of the cow or mare with the hand does not look well according to prevailing standards. The actual danger is dependent upon the degree of cleanliness. Ejaculated semen is not aseptic, and no method of insemination technically bars the inclusion of bacteria.

The semen, after obtained, should be kept in a glass vessel until used. A convenient container is an ordinary test tube. The tube may be placed in a thermos bottle through a hole in the cork. Most workers advise that, if the semen is to be held in storage, it be kept at 45 to 50 degrees F. Some bacteria are always present in semen and the refrigeration represses their growth.

It is commonly advised that the semen be diluted. If obtained from a healthy vagina after coitus, it is already diluted with the estrual mucus. It has not been clearly demonstrated that this natural diluent is inferior to the artificial ones recommended. If the semen be obtained by other than method 1, it is apparently best that it be diluted. The dilution increases the volume and facilitates its division into portions. Some research workers have concluded that some diluents increase the motility of the spermatozoa. The Russian workers have advised a diluent composed of glucose and the phosphates of calcium, sodium, potassium and manganese. It is not wholly clear why artificially increased motility of the spermatozoa should be regarded as either necessary or advisable. Violent or abnormal motility of animals or of individual cells may indicate disease as truly as does subnormal motility: the maintenance of physiological motility is the real goal.

It is not now known that any technic is available by which semen may be handled or stored outside the genital tract without deterioration of the spermatozoa. Such deterioration does not become recog-

nizable immediately by present means. It is evident that the spermatozoa of the bull, under moderate refrigeration, may retain considerable fertilizing power for several hours. It is the general consensus of opinion that decreased fertilizing power becomes evident within four or five hours, regardless of the continued motility of the sperms. That is, the fertilizing power of spermatozoa ceases before motility disappears. According to Miessner[137] the Russian workers hold the spermatozoa in potent condition for as long as four days. Inevitably, the longer spermatozoa are artificially stored, the higher the ratio of non-motile cells, the lower the motility of the sperms, and the greater the rate of conception is reduced. All available evidence indicates that artificial insemination should be carried out at the earliest period possible, after the semen has been procured.

The part played by coitus in reproduction is not fully known. The female rabbit and cat do not ovulate except after coitus. If the doe rabbit is mated with a vasectomized male, she ovulates and pseudopregnancy follows. It is not fully known what, if any, effect coitus exerts upon ovulation in the commercially important domestic animals. It is not impossible that coitus early in heat stimulates and hastens ovulation. If this be so, then natural breeding is quite certainly preferable to artificial insemination in so far as the individual and her young are concerned. Those females in which heat is most brief and vigorous are the best breeders. Any factor which may delay ovulation, so far as known at present, lowers the value of progeny. The long range effect of artificial insemination upon the herd, family or breed has not been determined.

The technic of insemination varies. Miller[140] advises for the cow the use of a glass tube, 18 inches long with a one-eighth inch lumen. A glass syringe is attached to this by means of a short piece of rubber tubing. The semen is drawn into the glass tube with the syringe. A speculum is placed in the vagina of the female, the distal end of the glass tube passed into the cervical canal and the semen forced out with the syringe. A simpler apparatus may be assembled by using an ordinary human urethral syringe, operated by a bulb. Attach a piece of rubber tubing of appropriate length to the nozzle of the syringe. Draw the desired amount of semen into the syringe, place the distal end of the rubber tube into or against the cervical canal and discharge the semen by compressing the bulb.

Some workers emphasize aseptic methods for introducing the inevitably contaminated semen. Some would introduce one hand into the rectum and therefrom guide the glass tube of the syringe to the os uteri. Cleanliness of the vulva immediately beneath is greatly

imperilled. Others insist upon the use of a speculum, dilating the vulva and vagina and causing a draft of stable air, and all that it contains, to flow into the widely dilated canal.

The most convenient and expeditious method of guiding the end of the tube to the cervical canal is by the operator's hand. When cleanliness is properly observed, it is probably as safe as any other method. Among dairy cows, where artificial insemination is most practiced, important vaginal infection is common. When passing from one cow or herd to another the prudent operator would naturally resort to proper disinfection.

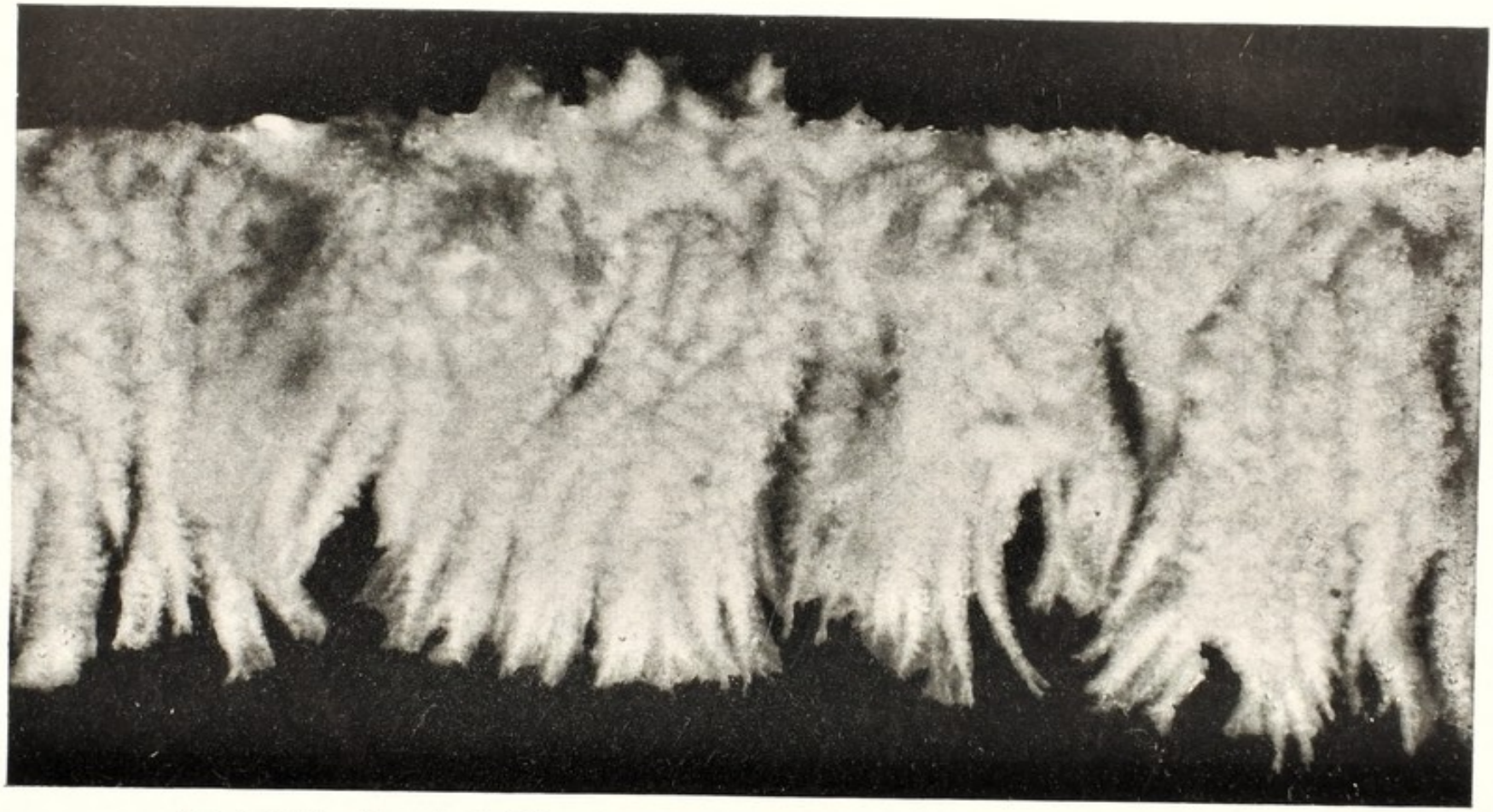


FIG. 46. Section Through the Allantochorion of Cow Showing the Placental Tufts or Villi, Enlarged.

Chapter XV

PREGNANCY

WHEN a healthy spermatozoon and ovum meet in favorable environment pregnancy follows. After coitus the spermatozoa rapidly traverse the cervix, uterus and oviduct and are presumably near the ovarian end of the latter when ovulation occurs. The uterus of the cow may be douched with an antiseptic solution within a few hours after coitus without interference with conception.

The oviduct, inferentially less frequently or dangerously infected than other portions of the genital tube, assures to the newly fertilized ovum the maximum safety. It traverses the oviduct slowly and reaches the uterine or cornual cavity several days after conception. Zietschmann[278] states that the fertilized ova of the mare and bitch require eight to ten days for their journey through the tubes. I have douched the uterus of the cow ten days post coitum without injury to the fertilized ovum still within the tube.

The fertilized ovum of the herbivorous unipara physiologically passes through the oviduct and the uterine cornu corresponding to the ovary from which it comes, to its base, where the ovum becomes arrested to remain throughout gestation. The fixation is not due, as in primates, to the ovum burrowing into the endometrium, or to the endometrium enveloping the ovum. If a pregnant cow be killed during the third month, the ovum lies free in the uterine cavity. The chorion bears over 100 cotyledons, which correspond accurately in number, form and size to the uterine caruncles or special placental areas. These chorionic cotyledons appear very early, as soon as the allantochorion has formed, and remain constant throughout gestation, except when a caruncle and the corresponding cotyledon are simultaneously destroyed by necrosis or other pathological force. The factor which maintains this contact is unknown. The contact is highly efficient and physiologically assures ample nutrition to the ovum. If the fertilized ovum be acardiac or asexual, the placental contact fails in its nutritive function and the imperfect ovum perishes unless it promptly acquires vascular contact with the chorion of a normal ovum. Later in gestation, the placental tufts of the chorion project increasingly into the crypts of the uterine caruncles of ruminants and into the endometrium of the diffuse placentae of horses and swine. When these animals are slaughtered at or near term there is some mechanical resistance to the separation of the

fetal, from the maternal placenta. Pathologically the chorionic tufts, Fig. 46, may become incarcerated within the maternal crypts to constitute retained placenta.

The Clinical Diagnosis of Pregnancy

The interest aroused in sterility during recent decades has greatly stimulated the question of the diagnosis of pregnancy, which is essential to the proper handling of the diseases of the reproductive system. If a practitioner fails to identify an existing pregnancy and attempts to douche the uterus of a cow, he may cause instrumental abortion, or the uterus may be ruptured and the fetus drop into the peritoneal cavity. If an error be made in diagnosis and the corpus luteum of pregnancy dislodged, abortion follows. When pregnancy is erroneously diagnosed in a sterile animal, delay and financial loss ensue.

The diagnosis of pregnancy is most important in the mare and cow, in which it is also most readily made because of the availability of the rectal palpation of the internal genital organs. As a rule the clinical diagnosis of pregnancy in the mare and cow is highly reliable, but exceptionally almost insurmountable obstacles are present. During the first 30 days post coitum, physical diagnosis is not dependable, and it is during this period that the highest rate of mortality of the fertilized ovum occurs, the dead ovum disappearing unseen. The signs of pregnancy are many:

(1) The cessation of the menses is regarded as strong presumptive evidence of pregnancy in woman, if the flow has been regular. No definite flow of blood occurs in domestic animals which can be identified with menstruation of woman. If a cow be killed shortly after ovulation, and has not been bred, the uterine caruncles are thickly dotted over with bright droplets of blood. Later, at about 48 hours after the termination of heat, there is sometimes a discharge of bright red blood from the vulva, coloring the tail and parts ventral to the vulva. It is commonly believed that if the cow conceives the flow of blood does not occur. Hemorrhage from coital injury, usually very slight, may occur shortly after service. Among multipara, especially the sow and bitch, slight vulvar hemorrhage may occur as a result of the engorgement of heat.

(2) The domestic mare and cow, if well fed and healthy, regularly ovluate throughout the year at frequent intervals unless pregnant. The heat cycle for the cow is about twenty-one days, with a variation in individuals of two to three days; the cycle for the mare

is said to be twenty-one to twenty-eight days. Pathologically all rules are discarded. Our knowledge of the physiological cycle is badly clouded by the difficulty of drawing a clear line between physiology and pathology.

If a fertile cow be mated with an ideally healthy bull, and she fails to come in heat twenty-one days later, she is presumably pregnant. If not in essentially perfect health, the ovum may perish at any date. If the death occurs late in the cycle, the next heat may develop after an indefinite period. If a cow is genitally diseased or is immediately on the borderline, for example, of nymphomania, coitus may aggravate the condition and may prolong and intensify the estrum. When the next estrum recurs after an abnormally long period following service, the inference is fairly justified that conception occurred followed by the death of the ovum. The value of the cessation of the heat cycle after breeding rests, therefore, upon the sexual health of the mated animals. If the female has not been regular in her heat periods, she should not be bred. If she has been bred, the failure of estrum to occur three weeks later is not a safe criterion of conception.

Some pregnant females come in heat and copulate if opportunity offers. I am not aware that such phenomenon occurs in multipara. Its appearance in unipara is variously explained. I have palpated the genital organs in a pregnant cow in estrum, and found the corpus luteum in the ovary upon the side of pregnancy in an advanced stage of atrophy, and a ripe follicle upon the ovary of the non-pregnant side. Some observers, finding a corpus luteum on one side, and a fetus in the other horn of the uterus, have concluded that the fetus had migrated, but my observation does not warrant such hypothesis. In 3000 pregnant bovine uteri examined on the killing floor, no instance was seen of the corpus luteum in one ovary and the fetus in the other cornu.

When a bovine fetus undergoes mummification the cow may not come in heat for years. If the corpus luteum atrophies, or is dislodged manually, the cow comes in estrum and expels the mummy.

THE UTERINE SEAL

The cervical canal of uniparous domestic mammals becomes occluded early in pregnancy by an accumulation of mucus differing markedly in character from the mucus secreted by the healthy non-gravid cervix. It is colorless, firm, highly resistant and extremely adhesive. It attains its maximum in the cow and constitutes a highly authentic sign of pregnancy from about forty days to the end of

gestation. It becomes increasingly prominent as gestation advances and at near term attains the size and form indicated in Fig. 47. It is readily and authentically palpable per vaginam with the finger tip. If the finger tip is gently pressed against the os uteri externum, when withdrawn, the seal adheres to the finger with definite force. At the same time the vaginal mucus changes in consistency. While moderate in volume, it is extremely adhesive. The character of the seal and

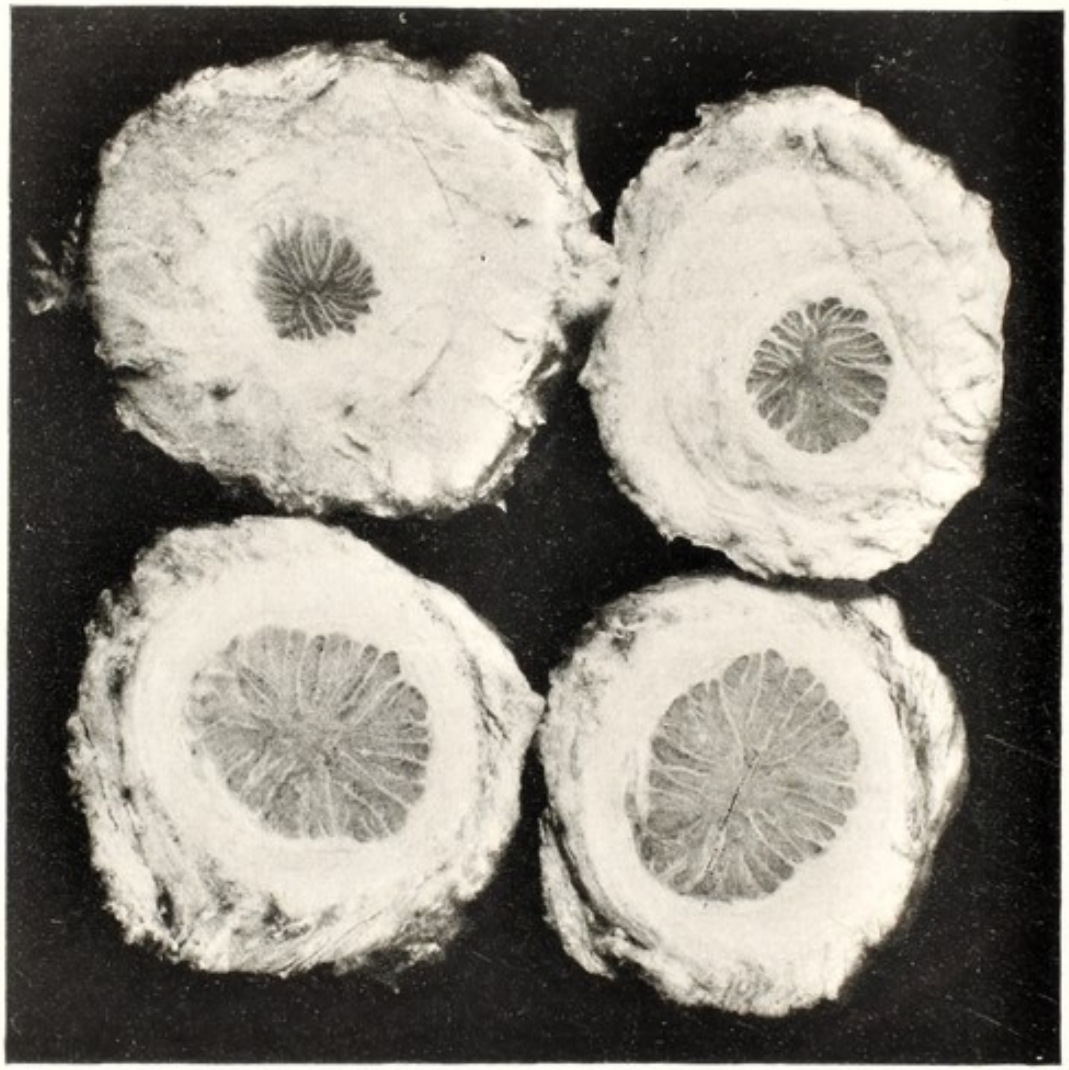


FIG. 47. Bovine Uterine or Cervical Seal.

The cervix has been divided transversely at four points, the largest seal being at the vaginal end, the smallest at the uterine end.

of the vaginal mucus are visually recognizable with the aid of the vaginal speculum.

Pathologically a false uterine seal, sometimes of gigantic volume, may form as a result, apparently, of a peculiar type of cervicitis, accompanied by a persistent corpus luteum and without estrum. The seal persists indefinitely in fetal mummification and also in the

primitive monster known as cystic mole (Fig. 48) in which the embryo perishes early or fails to form, while the vitelline sac or other membranes, corpus luteum and uterine seal persist. For a period the condition may absolutely defy identification. Repeated examination finally reveals the fact that the monster is not growing and careful examination then renders its character clear and opens the way for handling by dislodging the corpus luteum causing heat and the emptying of the uterine cavity.

A teratological projection of the cervix into the cornual cavity, with accumulation of uterine secretions, may lead to the formation

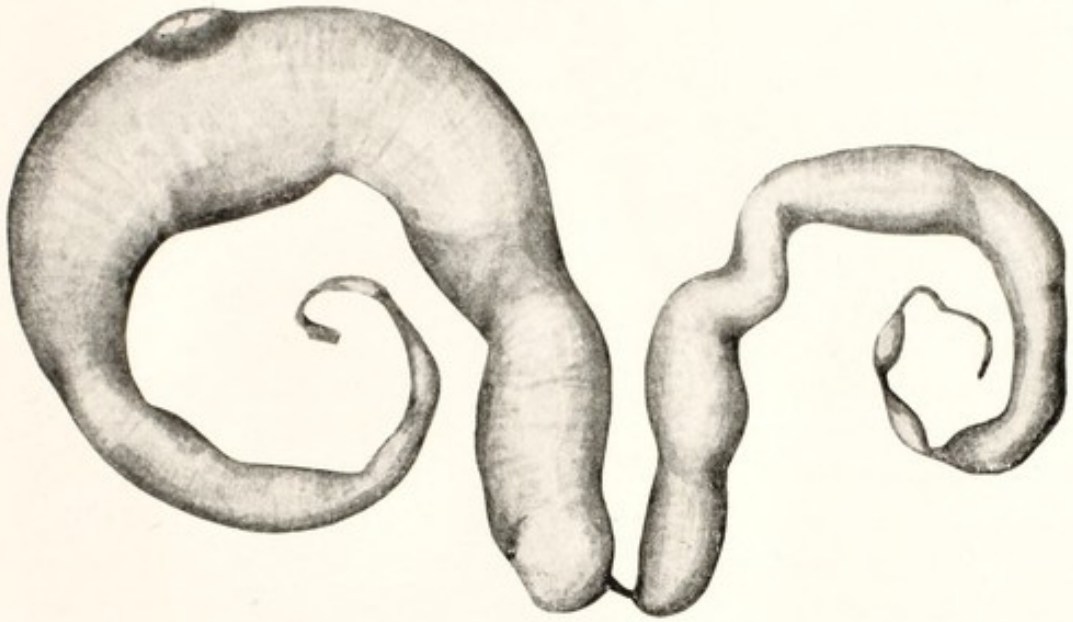


FIG. 48. Bovine Cystic Mole, Removed 200 Days After Conception.

Total length of sac forty-six inches; greatest circumference seven inches. The dark spot upon the convex border of the larger cornu apparently represents the arrested germinal area of the ovum.

of a uterine seal of large proportions, as shown in Fig. 8. The distension of the uterus with fluid, the presence of a distinct seal and of a corpus luteum tend strongly to mislead.

The trichomonad disease frequently causes fetal death and maceration, with uterine distension, without destroying the uterine seal for a considerable period, and thereby misleads the veterinarian to believe that the pregnancy is continuing.

The seal may fail to form, or be destroyed by purulent cervicitis. If the cervicitis extends to the cranial end, abortion is essentially inevitable, but in rare cases the caudal end of the cervical canal may be suppurating freely while its cranial portion remains intact. In one such case I assumed non-pregnancy because of the purulent

cervicitis, penetrated the seal in the cranial end of the cervix with a catheter, douched the uterus and caused abortion.

With a few rare exceptions such as those named, the uterine seal constitutes a highly authentic sign of pregnancy. The exceptions can be identified and a correct diagnosis made by rectal palpation.

The uterine seal is somewhat less conspicuous in the mare, but constitutes valuable evidence of pregnancy. The vaginal mucus, like that of the cow, is distinctly adhesive. The seal is not recognizably present in multipara.

THE RECTAL PALPATION OF THE REPRODUCTIVE SYSTEM

The palpation of the genital organs of the mare and cow per rectum constitutes the most valuable means for the diagnosis of pregnancy. Some writers have grossly magnified the alleged dangers of rectal palpation of the gravid uterus. Conducted with reasonable care it is virtually without danger. Certainly cows sometimes abort after digital palpation of the pregnant uterus, just as they abort without it. So in one case I assured a client that dehorning a pregnant heifer would not cause her to abort, but she aborted shortly after the operation. On the other hand, when examining a cow for pregnancy at 145 days I was badly puzzled for a time and finally diagnosed uterine torsion. The cow was cast and rolled over several times, my hand being kept in the rectum during the rolling. She was released and caused to get up, palpated more, thrown down again and rolled some more, totalling six complete revolutions. She carried her calf 280 days, calved without assistance, and both cow and calf did well.

Depending upon the size and conformation of the animal, the stage of pregnancy and the tonicity of the reproductive system, it often becomes advisable or essential to apply traction to the cervix and draw the uterus, oviducts and ovaries toward or into the pelvis in order to make accurate palpation. The traction is applied by means of Albrechtsen's forceps or other convenient instrument. The very long cervix of the cow (Fig. 49) affords ample room for grasping without material danger of important laceration. The cervix of the heifer often has so small an external os that the insertion of the forceps into the canal is impracticable, in which case the vaginal wall may be grasped at either side of the opening. The forceps may be applied by guiding them to the cervix with the hand, they may be applied by sight with the aid of a speculum, or they may be guided to the cervix by means of rectal palpation. The majority of cases do not call for the application of forceps and traction.

The application of traction in difficult cases renders the uterine walls taut and the various structures more clearly defined and brings the entire genital system nearer to the operator.

At 21 days post coitum, if the cow is pregnant and healthy, there should be present in one ovary a fully developed, normal corpus luteum, and the corresponding uterine cornu may be very slightly enlarged; it cannot be materially enlarged because at this period the fertilized ovum will have been in the cornu for but 7 or 8 days. If the ovum has not been fertilized, the corpus luteum is regularly atrophied and a ripening ovisac, probably in the opposite ovary, is present. The uterus is engorged (erected) and the two cornua are of equal size. Should fertilization have occurred and the ovum perished, the estrual cycle is quite certainly unbalanced, and heat may not recur until after an abnormally long period. In the meantime



FIG. 49. Sagittal Section Through the Cervix of a Pregnant Cow.
1, Os uteri externum; 2, os uteri internum; 3, base of second annular fold of cervix; 4, 4, the uterine seal.

the corpus luteum may be enlarged, embedded, cystic or reveal other abnormality which may delay temporarily an accurate diagnosis.

Those portions of the Muellerian ducts which form the uterine cornua of ruminants revolve upon their long axes and come to rest with the line of attachment of the broad ligaments upon their ventral, or pubic surface. With the advent of pregnancy the weight drags the uterine cornua downward and forward along the abdominal floor toward the diaphragm. This drags the cervix forward and downward and tenses and elongates the vagina. The floor or pubic wall of the pregnant uterus extends directly forward in a line with

the ventral wall of the cervix and vagina. When the cervical end of the uterus becomes enlarged and distended, the increase in the sacro-pubic diameter is upward, toward the sacrum. At thirty days post coitum there is recognizable a distinct terrace at the juncture of the uterus with the cervix. This increases as gestation advances and at near term, as shown in Frontispiece at 1′, the dorsal wall of the uterus is pushed caudalward over the roof of the cervix and the anterior portion of the vagina. The somewhat flaccid uterus of a pluriparous cow may be dragged far forward and the terrace of pregnancy eliminated. When the uterine forceps are applied to the cervix and moderate traction applied, the terrace is restored. At forty days post coitum, pregnancy is reliably diagnosable in essentially all pluriparous cows.

The uterus of the pregnant mare becomes enlarged and distended somewhat later than that of the cow. The embryonic sac is, for a time, ovoid instead of tubular as in the cow, and is located at the base of the gravid cornu, where the enlargement is palpable at about forty days. Physiologically no portion of the equine or bovine fetus, or of its amnion, invades the non-gravid cornu. The allantochorion of both species passes into, and completely fills the non-gravid cornu, but its growth is repressed. The repression in growth of the non-gravid chorion is modified by the presence of pathological conditions. A pluriparous cow which has suffered at the close of a prior pregnancy from retained afterbirth in the then gravid horn, with extensive or complete necrosis of the uterine caruncles, almost certainly becomes pregnant in the opposite cornu and the defective non-gravid cornu is definitely dwarfed. In extreme cases the allantochorion of the non-gravid cornu is a frail, thin, transparent membrane, without trace of placental structure. Sometimes, as in Colored Plate II, infection persists in the previously affected cornu, causes total necrosis of the dwarfed chorion and, almost certainly, leads to abortion.

The marked repression of the non-gravid cornu not only permits a definite diagnosis of pregnancy but, more important, reveals extensive lesions dating back to a previous gestation, lowering the safety of the existing pregnancy, and in case of viable young, foretelling probable weakness and low value. If the case be followed through parturition and the afterbirth studied, authentic record of the placental disease is found. Rarely a cow which has suffered unicornual necrosis of the uterine caruncles conceives again on the side with few or no caruncles, in which case the non-gravid horn is relatively large. I have observed but one case.

Since the uterus of the mare, Fig. 13, has a prominent "body,"

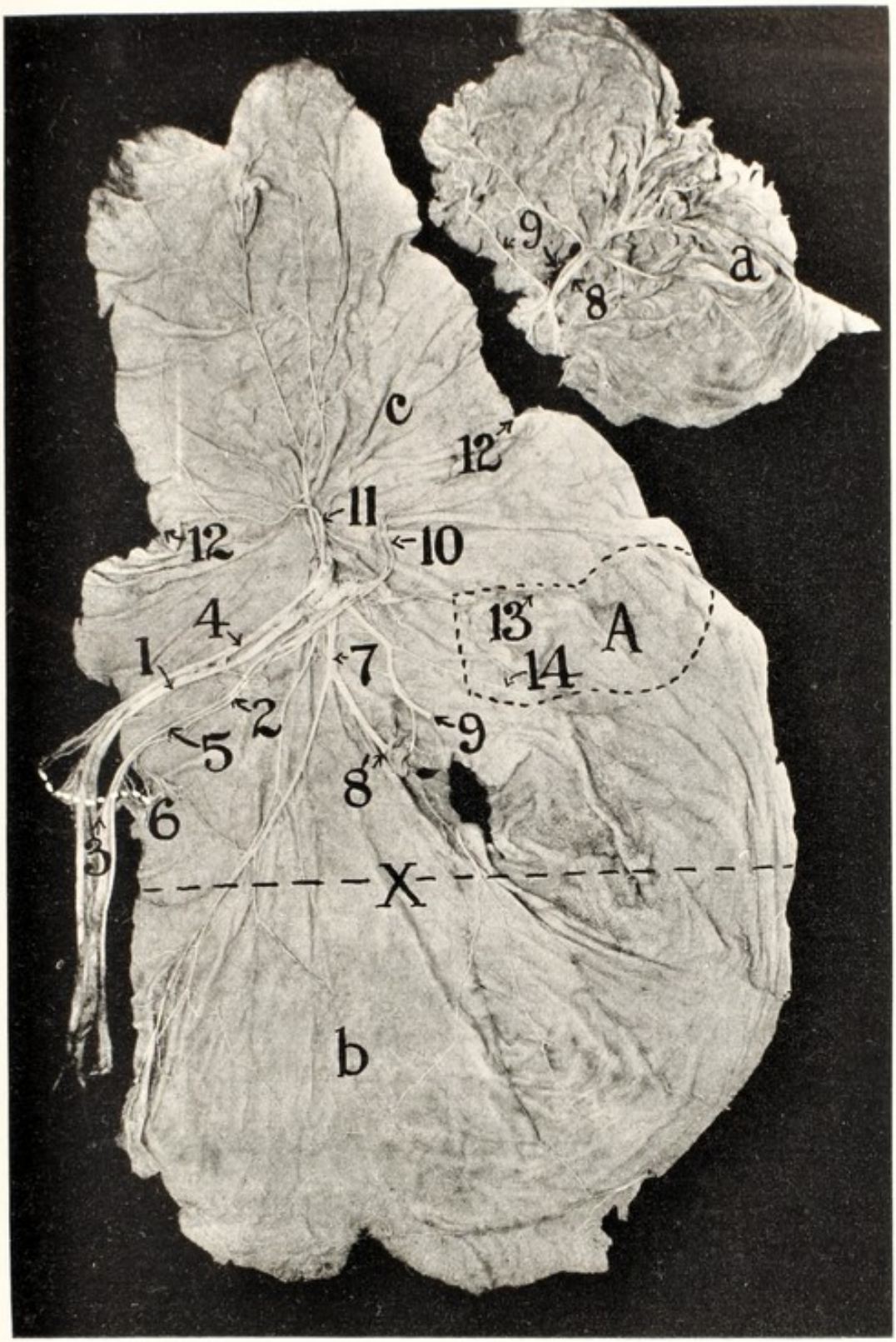


FIG. 50. The Equine Vascular Allantois.

a, excised non-pregnant horn, its location indicated by the aperture just beneath, 9; *b*, the body of the uterus; *c*, the pregnant horn; *X*, approximate line of demarcation between the body and the cornua; 1, chief umbilical vein; 2, minor umbilical artery. Slight variations in size of the arteries are common, but the variation is rarely so marked; 4, major umbilical artery; 5, minor umbilical vein; 6, vestiges of amnion; 7, chief venous trunk to the base of the pregnant horn, the body and the non-gravid horn; 8, 8′, chief branches of 7; 9, 9, chief arterial trunk to the base of the gravid horn, the body and the non-gravid horn; 10 minor allantoic trunks; 11, chief vascular trunks to ovarian pole of gravid horn; 12, 12, location of ovarian pole of amnion. *(Cornell Veterinarian)*

the fetus grows into this so that somewhat more than one-half its volume lies within the body and the other half in the base of the gravid cornu. The non-gravid horn is given off near the middle of the gravid tube, somewhat like the thumb of a mitten, as shown in Fig. 50. Pathologically the ovum, instead of projecting into the body of the uterus, may project across into the non-gravid cornu to constitute bicornual pregnancy, the body, instead of one of the horns, being non-gravid. Bicornual pregnancy should be authentically diagnosable by rectal palpation and the obstetrist be placed in position to lower the peril to the mare by inducing abortion during the first half of gestation. It is not the mere diagnosis of pregnancy which is to be sought, but when skill is applied, the character of the pregnancy is revealed and may, in some cases, serve as a guide to profitable interference.

The enlargement of the pregnant uterus is characteristic. It may become enlarged and distended from a great variety of causes, but in each case there are means by which authentic differentiation may be made. Except in the presence of twins and of bicornual pregnancy, there is a gross difference in the volume of the two cornua; when the uterus is distended with pus or other fluid the two cornua are regularly equal. During the first half of gestation in the cow the ovaries are palpable, and the corpus luteum of pregnancy stands out above the adjacent ovarian surface; when the uterus is distended with pathological fluids, the corpus is regularly central in the ovary and projects little above the general surface of the gland.

Later the fetus itself becomes palpable and may be caused to struggle by grasping an extremity. When nearing term the head of the fetus may project so far caudally, as shown in Colored Plate I, that inexperienced persons have suspected extra-uterine pregnancy. The terraced character of the pregnant uterus does not prevail in the pathologically distended organ. Diseased uterine walls are inert. The pregnant uterus is sometimes flaccid and wanting in tone, even when a live fetus is palpable. This signifies endometritis and presages abortion or pathological birth. In one case the atonic state of the uterus was so marked that my diagnosis was 'Pregnant, but unsafe.' The cow aborted some weeks later. When the uterus is distended with pus the uterine seal is usually, if not always absent. When distended with lymph of mucus, the uterine seal may be present, as shown in Fig. 8. The seal is regularly present in bovine mummification and for a considerable period during fetal maceration due to trichomonad infection.

The palpation of the uterine arteries provides information of the highest value. With the advent of pregnancy these abruptly take on

new activity and volume. There are three pairs—the utero-ovarian, uterine and vaginal trunks—of which the uterine is the most important. In the cow this artery leaves the caudal end of the posterior aorta or the iliac and passes ventrally not far from the antero-median line of the ilium, suspended in an ample fold of peritoneum, which permits it to be readily picked up and accurately palpated. In the non-pregnant cow it is 3 to 4 mm. in diameter, with clearly defined pulsation; with the advent of pregnancy it grows very rapidly and finally attains a diameter of 1 to 1.5 cm. The pulsation changes in character with the advancement of pregnancy: the intra-arterial pressure is heightened and there is a distinct thrill. Presumably similar enlargements of the arteries occur in some new growths, especially in malignant tumors, but these are almost unknown in domestic animals, and in such case the enlargement of the artery is unilateral, while in pregnancy it is bilateral, the enlargement being greater on the gravid side.

The posterior uterine or vaginal artery arises from the internal pudic and passes across the pelvic cavity within the broad ligament to the vagina and cervix. In the non-pregnant cow it is almost as large as the main uterine artery. It becomes distinctly enlarged in pregnancy, but not usually to the same degree as the main vessel. It is readily picked up and palpated.

The specific changes in the uterine arteries of pregnancy may constitute the keynote in differential diagnosis. A highly pedigreed Holstein-Friesian cow was sold as six months pregnant, the pregnancy diagnosis having been made by an unusually able and careful clinician. She failed to calve after the normal interval. A re-examination was made. Forewarned by her history, forceps were applied to the cervix and the uterus drawn as far back as practicable. The uterus was distended as at term, but the uterine walls were atonic. The uterine arteries were of non-pregnant size and type. No fetus was palpable. Although no genital discharge was evident, the cervical canal was not sealed, and the vaginal mucus was not adhesive. About five to six gallons of thin, yellowish, flocculent, odorless fluid was siphoned from her uterus. She came from an area where trichomonad disease was present. Probably she had actually conceived and the embryo had disintegrated.

The palpation of the fetus becomes feasible in the mare and cow at 120 to 130 days. Other hard masses may deceive the examiner when off his guard. One of the most confusing is the large, firm fat tumor sometimes encountered in the mesentery of the cow. Swingingly freely, it may move away from the hand much like the fetus floating in the amnio-allantoic fluids. If the veterinarian will take the

care to trace the uterus from the cervix forward, he will find that the mobile body is not in the uterus, and further palpation shows it is not a fetus.

Physiologically the fetus of the cow or other unipara lies *en arc*, with its convex, dorsal line directed toward the spinal axis of the mother. The head of the fetus is normally directed toward the cervix of the dam. The concave, ventral line of the fetal body is directed toward the line of attachment of the mesometrium to the uterus, so that the navel of the fetus faces the course of the uterine arteries. Accordingly when palpating the fetus the examiner first contacts the nose, the cranial vault or the anterior feet from the dorsal aspect. Early writers claimed that the bovine fetus lay upon its back, with its concave ventral line directed toward the convex dorsal surface of the uterus, so that an arciform body came to rest within a curved tube with the convex line resting against the concave side of the tube. The fetus may, and sometimes does, lie with its spinal column against the abdominal floor of the mother, but the uterus has revolved with it. Hence, when palpating the fetus during pregnancy, it may lie in dorso-pubic position, owing to the presence of torsion.

Displacements of neighboring organs may complicate the diagnosis of pregnancy. Rarely I have met with dislocation of the rumen in atonic cows. The organ slips downward, the ventral end gliding across the abdominal floor to the right, beneath the uterus. One then palpates in front of the pubis an immense tumor, soft and flaccid, with soft contents. By careful tracing to the left, the mass may be identified as the rumen. If an examination be made again in a day or two, perhaps the mass will have vanished.

THE DIAGNOSIS OF TWIN PREGNANCY

Most twin pregnancies in domestic unipara are bi-cornual. They are usually readily diagnosable in the cow because of the symmetrical enlargement of both cornua and the presence of a corpus luteum in each ovary. After about 130 days the fetuses may be felt. Unicornual twins are not so readily recognized. They may have either independent or fused allantochorions. Although the chorions may in part lie side by side, as indicated in Fig. 20, the fetuses must in all cases, so far as known, lie end to end. Early after mid term the combined length of the unicornual twins exceeds the distance from the maternal pelvis to the diaphragm, necessitating the folding of the gravid cornu between the contiguous ends of the fetuses, so that they come to lie side by side in the abdomen, but end to end in the allantois. The two fetuses thus come to lie approximately

parallel. If in the cornu they lie tail to tail, the two heads are directed toward the pelvis of the dam. Once the arrangement has been recognized, the clinician should expect tardy delivery of the second fetus. Besides the great distance through which it must move, it must also turn on its transverse axis through 180 degrees, the revolution involving also the ovarian half of the cornu. Acardiac or amorphous monsters may serve to complicate diagnosis in twin pregnancy. If the acardiacus is in the horn opposite to the host twin, early examination reveals twin pregnancy, which is correct, but when gestation ends the amorphus may be very small, pass unseen and perhaps be eaten by the cow with the afterbirth. The correctness of the diagnosis may then be challenged.

THE LABORATORY DIAGNOSIS OF PREGNANCY

During recent years numerous laboratory methods for the diagnosis of pregnancy have been advocated. They are chiefly based upon the presence of pituitary, ovarian or placental hormones in the blood or urine of the pregnant female. Among the earliest students of the problem was Aberhalden[2] in 1912, followed by many others, including Aschheim and Zondek[6] in 1927. In 1928 Helm and Zühdi[85] reviewed the literature quite fully and made extensive researches according to the methods of Lüttge and Mertz and of Dahmen-Wollersheim. With each method they concurred with Lüttge and Mertz: "Wir sind auf dem Wege—nicht am Ziel." (We are on the way—not at the goal!) The laboratory diagnosis of pregnancy was originally advocated for the early diagnosis of pregnancy in woman, where palpation is far less readily applied than in mares and cows. There is apparently much conflict of opinion regarding its efficacy by prominent medical writers. Thus J. Whitridge Williams[226] observes that the technic is too complex for the clinician and that the findings are wanting in reliability.

Writers upon veterinary obstetrics generally regard the clinical diagnosis of pregnancy in the mare and cow as definitely more economical and reliable than the laboratory methods. Among the smaller domestic animals the early diagnosis of pregnancy is of minor interest. Neither is the prudent clinical veterinarian profoundly interested in the very early diagnosis of pregnancy, such as the 7th or 8th day post coitum diagnosis in the mare while the ovum is yet in the tube and may not reach the uterus alive. It is quite common in dairy herds to observe an average of 2 to 3 services per recognized pregnancy, and it is highly probable that most of the failures are owing to tubal death of a weak, fertilized ovum. When a cow does not visibly conceive until after three services the clinician

knows that the ovum or the spermatozoon was defective, and a diagnosis of pregnancy or non-pregnancy before 20 to 30 days is certainly of very scant, if any value.

Schmaltz[178] admits that the laboratory tests are technically interesting, but observes that the advocates of the methods disagree among themselves. Stoss[189] believes the laboratory test definitely inferior to the clinical examination. Keller[105] regards the laboratory methods as impracticable and unreliable. After 30 days, clinical diagnosis of pregnancy in the mare and cow is far more trustworthy than laboratory methods at present known.

There are sundry other methods for diagnosing pregnancy, such as the change in the behavior of the animal, the changes in the form of the abdomen, ballottement, and the development of the mammae, all of which have some value but are open to error.

The value of the diagnosis of pregnancy when taken alone has been overestimated. If a sire becomes wholly sterile, his infertility is promptly exposed by the females with which he is mated, and this is readily verified by the study of his stained spermatozoa. When an individual female fails to conceive, it becomes a question whether she or the sire is responsible. If the female is not regular and physiological in her estrual cycles, she should not be bred, in which case no question of pregnancy arises. If pregnancy be diagnosed by laboratory methods earlier than would be possible clinically, and the ovum later perishes, the effect is confusing and misleading.

The clinical examination of the reproductive system of the mare and cow has not yet reached, or closely approached its potential value. When a cystic mole, like that in Fig. 48, is present, the condition may persist indefinitely and may not be discovered by any laboratory method; if studied clinically, it is identifiable and remediable. Bicornual pregnancy in the mare, which quite uniformly involves the death of the fetus with imminent peril to the life of the mare, is diagnosable clinically in early gestation, when the abnormality may be overcome with reasonable safety by inducing abortion.

The desired goal is not therefore the mere diagnosis of pregnancy or non-pregnancy. When pregnancy is present, the character of the pregnancy may be determined by clinical examination. If not pregnant, the obstacles to conception may be recognized and properly handled. When laboratory diagnosis of pregnancy is applied, it may reveal only the one fact, and for a clear conception of the character of the pregnancy resort must yet be made to clinical examination; if non-pregnancy be diagnosed, clinical examination must determine the cause.

SECTION III

TUMORS OF THE REPRODUCTIVE SYSTEM

Chapter XVI

MALIGNANT TUMORS

MALIGNANT new-growths of the genital organs of domestic animals are apparently less common than in man. The impression may be quite erroneous. Most domestic animals primarily grown for meat, are slaughtered before reaching an age conducive to the formation of cancer. Such tumors occur in all species of domestic animals and in each of the organs, as is well shown by Kitt[107] and other authorities in the field of pathological anatomy.

Malignant Tumors of the Testicles

I have observed two clinical cases of suspected malignant testicular tumors, neither of which was verified by histological examination. One was in a young boar, presented for cryptorchid castration. The abdominal testicle was firmly adherent to the contiguous viscera and was deeply injected. The other case occurred in an aged draft stallion. The owner refused to permit the removal of the enlarged gland. Autopsy was not available. Malignant tumors of the testicle, whether scrotal or abdominal, are difficult to diagnose clinically. Invasion of the gland, especially in the bull and boar, by tubercle bacilli or by *Bacillus abortus* may induce similar symptoms. The application of the tuberculin test in the former, and the agglutination test in the latter may aid in diagnosis. When these possibilities have been eliminated, the removal of the testicle is justified. In valuable breeding males the loss of one gland does not seriously interfere with fertility.

Since cryptorchid testes in man apparently offer important peril of cancer their removal is recommended as a prophylactic measure. This tendency is not recognizable in domestic animals, and castration is practised for economic reasons aside from any tendency to disease.

Malignant Ovarian Tumors

I have observed three ovarian tumors having the general appearance of malignancy and the diagnosis was verified by laboratory examination. One occurring in an infertile Holstein-Friesian heifer, was discovered by rectal palpation. The ovary, Fig. 51, was four inches in diameter and very firm. A colleague diagnosed it as an abscess of the broad ligament and I believed it an ovarian cyst. We were both in error, perhaps largely because, owing to the rarity of malignant tumors of the gland, we were not on guard. Had we paid



FIG. 51. Ovarian Angio-Sarcoma from Heifer.

1, Left cornu; 2, enlarged right ovarian artery; 3, right ovary. Below, a sagittal section of the tumor.

due heed to the grossly enlarged uterine artery, 2, Fig. 51, a correct diagnosis would have been probable. The laboratory diagnosis was angio-sarcoma.

A six-inch ovarian tumor, removed by laparotomy from an aged Guernsey cow, was diagnosed in the laboratory as carcinoma.

The third instance occurred in a sow of about 300 pounds, of which



FIG. 52. Ovarian Cyst-Adenoma from Sow, Weighing Thirty-Eight Pounds. 1, Necrotic area; 2, 2, cystic areas.

the tumor, Fig. 52, represented 38 pounds. It was not clinically diagnosable. The sow was destroyed because of great abdominal enlargement. The laboratory diagnosis was cyst-adenoma.

Malignant Tumors of the Uterus, Cervix and Vagina

I have observed but one case of cancer of the uterus, Fig. 53. It occurred in an aged bitch and caused a profuse, fetid discharge.

Malignant tumors of the uterus and of other derivatives of the ducts of Mueller are too infrequent to be of material economic concern. I have observed one cancer of the vagina in a cow. I had opened one of Gartner's ducts in order to remove an accumulation of calculi. She was in a district where cancer of the eye was common. The cancer developed at the point of incision into the duct of Gartner.



FIG. 53. Sarcoma of Canine Genital Tract.

1, Rectum; 2, left oviduct; 3, 3, oviducts; 4, uterus; 5, vagina; 6, urinary bladder.

Cancer of the vulva is seen and is to be differentiated from tuberculosis and actinomycosis.

Malignant Tumors of the Penis

I have not observed malignant neoplasms of the penis of uncastrated male animals and recall but one case in a gelding. Veterinary literature indicates that such tumors are rare.

Although malignant tumors of the genital organs are infrequent in domestic animals, the clinician needs bear in mind that they may and do occur in all organs of all species. Malignant tumors of the ovaries and testicles may be successfully removed if recognized early. Cancer of the uterus is not remediable in breeding animals, because their removal usually obliterates the genital canal and renders the animal sterile. When the penis is the seat of the tumor, and it is confined to the glans, timely removal is favorable for the continuance of fertility in valuable sires. Both stallion and bull copulate readily after the removal of the glans. The technic of amputation is presented under *benign tumors*.

Chapter XVII

BENIGN TUMORS

Benign Tumors of the Ovaries and Testicles. Par-ovarian Cysts

SOME of the benign tumors of the gonads are known to be teratological and the cause and date of origin of the others are largely unknown. Dermoid cysts of the ovaries and testicles of horses are not rare; in other species of domestic animals they are almost unknown. The notable limitation of dermoid cysts of the gonads to equidae is in virtual harmony with the incidence of dermoid cysts and dental anomalies as a whole: the horse also shows a high incidence of odontomes in and about the alveoli and of dermoid and dental tumors in the squamous temporal bone. While odontomes occur in all species, they are far more common in the horse than in all other domestic animals combined, as I have elsewhere attempted to show.[229, 230, 231] The dermoid cysts extending from the margin of the concha, and often ending in a large tooth in the squamous temporal bone, are not known to occur in other species of domestic



FIG. 54. Dermoid Cyst of Cryptorchid Equine Testicle.

1, Glandular tissue; 2, osseous or dental tissue; 3, cavity in osseous tissue; 4, epididymis; 5, spermatic cord.

animals. The dermoid cysts of the gonads, the odontomes and the "ear teeth" accordingly constitute a highly interesting group of congenital defects almost exclusively occurring in a single genus.

The dermoid cysts of the equine testicles are generally, if not always associated with cryptorchidy. They are usually much enlarged and firm and contain long matted masses of hair, frequently accompanied by bone or tooth tissue or both, as is indicated in Fig.

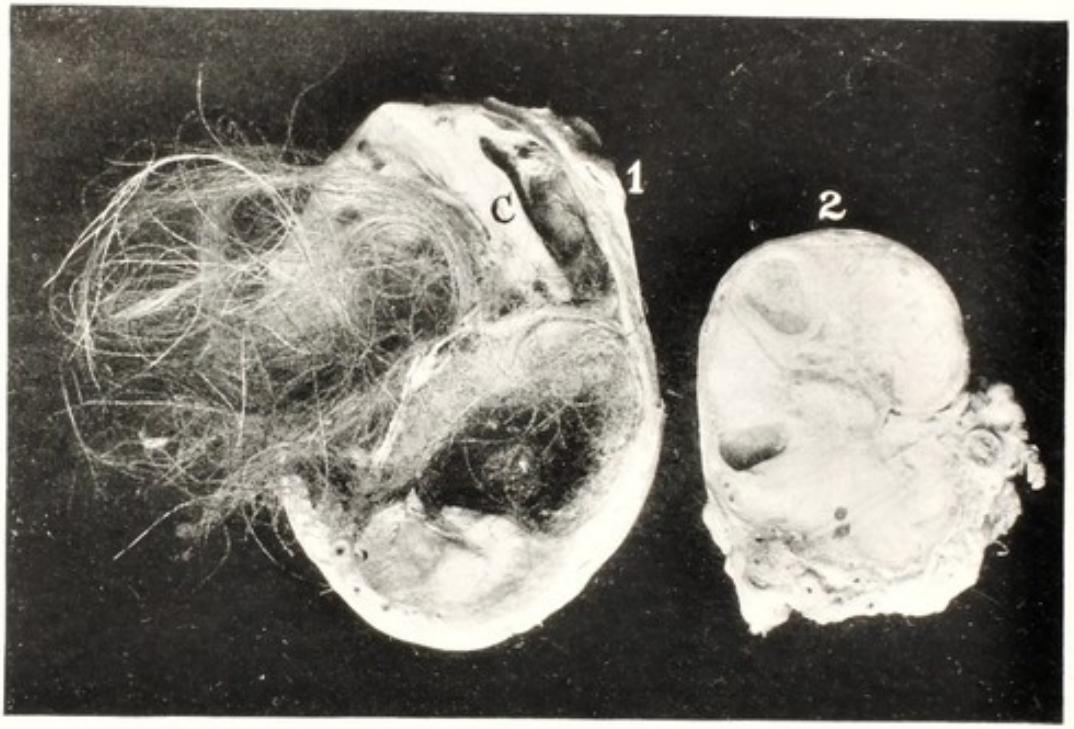


FIG. 55. Ovarian Dermoid Cyst of Mare.

1, The cystic ovary, containing long, matted hairs and at, *C*, a mass of cartilage; 2, normal ovary from same mare.

54. They are usually first recognized after surgical removal, because of cryptorchidy.

As a rule, ovarian dermoid cysts, Fig. 55, are not clinically identifiable. They produce no symptoms generally which might suggest their presence. When suspected, a provisional diagnosis may be made by rectal palpation. They have no known tendency to become malignant, or to induce nymphomania. As a rule the involvement of the gland excludes reproduction by the defective organ, but is not known to interfere with the function of the opposite gland. It is possible that both gonads may be alike involved, as in the case I have recorded[251] of a foal having dermoid invaginations from the margin of each conchal cartilage, leading to an "ear tooth" in each squamous temporal bone.

Human obstetrists state that in woman dermoid cysts of the

ovaries may become markedly pedunculated and drop into the pelvis during labor, to cause dystocia. During the dystocia the dermoid cyst may become ruptured by the pressure of the fetal head, causing the contents of hair and epithelial debris to escape into the peritoneal cavity and lead to fatal peritonitis. Such result is virtually impossible in domestic animals, the quadrupedal position and the location of the ovaries causing the enlarged gland to drop away

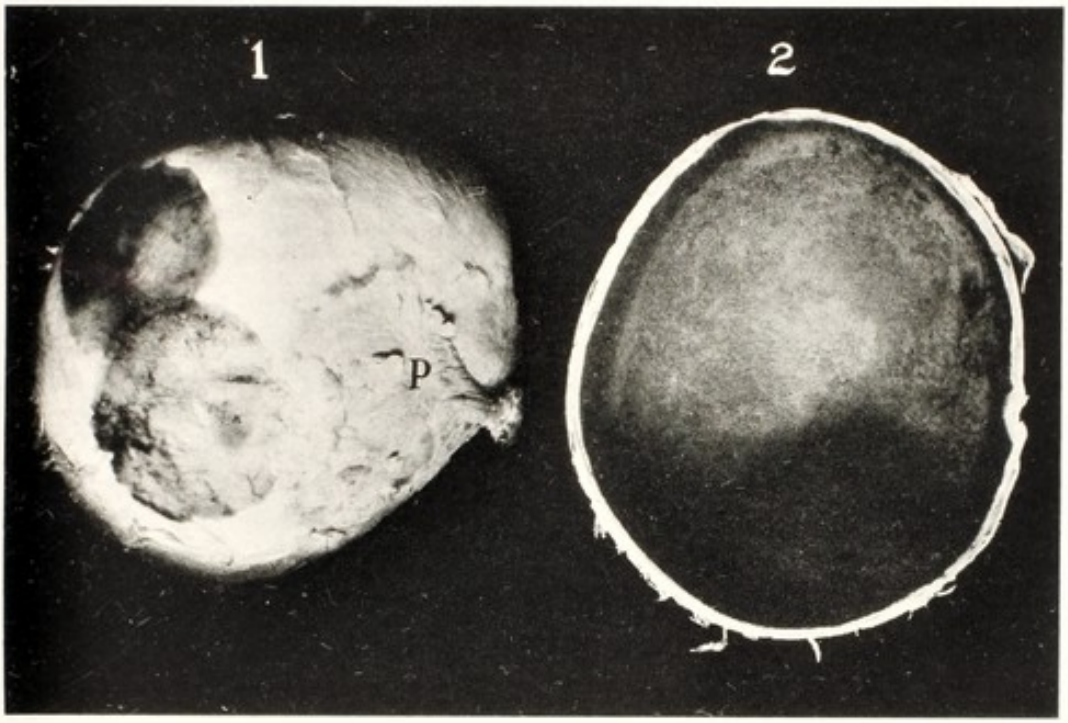


FIG. 56. Ovarian Cyst from Mare.

1, Exterior of cyst, showing, at *P*, the pavilion of the oviduct flattened against the cyst; 2, the interior of the cyst showing the dense walls.

from the pelvis. The enlarged ovary is more liable to lead to incarceration of the rectum, such as is observed in pedunculated parovarian cysts or cystic ovaries as described in the next paragraph.

Large ovarian cysts sometimes occur in the mare and cow, and presumably in other species, which, owing to the weight of the tumors, associated with the quadrupedal position of the animal, cause the formation of long pedicles. Their date and cause or origin are undetermined; they are probably largely congenital. They logically destroy, as a rule, the function of the involved gland but do not, so far as known, cause nymphomania or other evidence of ovarian pathology. They are discovered sometimes in the course of search for obstacles to reproduction. Most frequently they reveal their presence by incarcerating the rectum by means of their long pedicles,

causing violent obstruction colic, or the animal dies from the intestinal incarceration and its nature is recognized postmortem.

The rarity of this class of ovarian tumors tends to cause confusion in diagnosis and renders it important that the clinician acquaint himself with their occurrence. The closely associated parovarian cysts have a very similar significance to the clinician. It is important that the practioner remember that in very early embryonic life, when the two sexes are attaining their sexual characteristics, certain of the tubules of the Wolffian ducts, from which

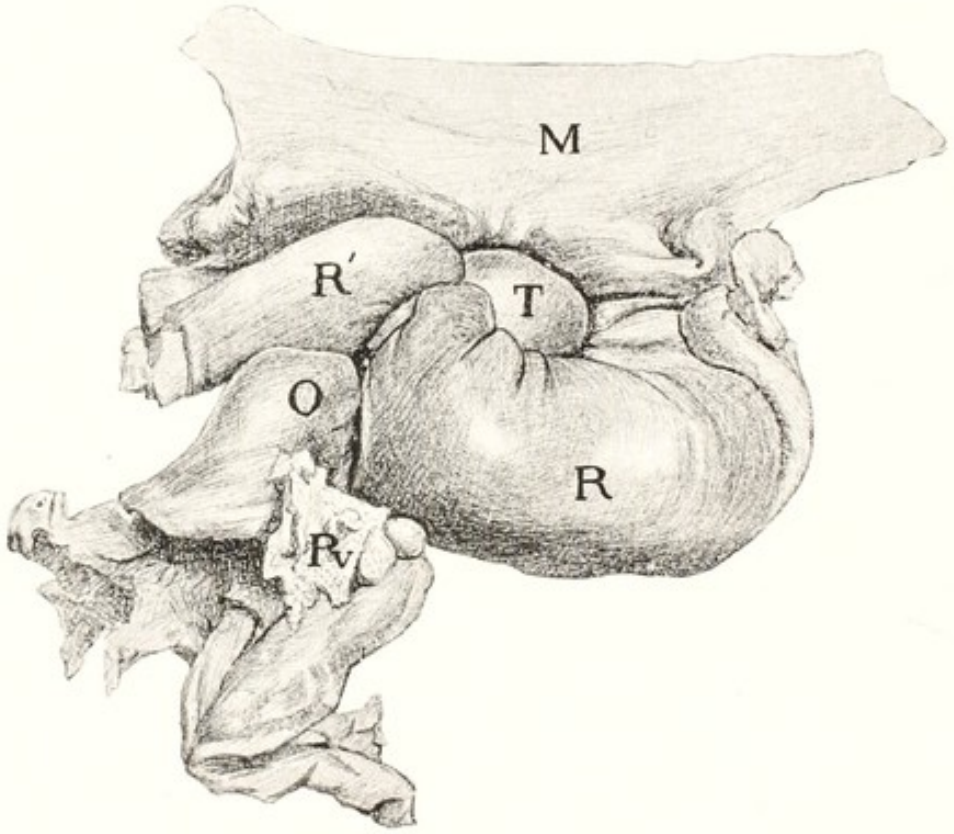


FIG. 57. Par-Ovarian Cyst from Mare Leading to Fatal Incarceration of the Rectum.

M, Right side of mesentery; *T*, tumor covered by the mesentery; *R*, the incarcerated portion of the rectum; *R'*, the rectum upon the anal side of incarceration; *O*, ovary; *Pv*, pavilion of the oviduct near origin of cyst.

the epididymis of the male arises, persist in the female as the parovarium or organ of Rosenmueller. In some species these tubular remnants become distended with fluid to constitute hydatids or cysts which, after attaining material size, acquire long pedicles because of their weight.

These hydatids are apparently common in woman, although as a rule too small to be of clinical interest. Occasionally they become so large that in the upright position of woman they drop into the

pelvis, as already noted in the case of dermoid cysts, and cause serious interference during labor. Such cysts are apparently quite rare in domestic mammals: I have observed but one case. It occurred in a mare. Veterinary literature does not indicate that it is often observed in any species.

The ovarian cyst depicted in Fig. 56 was removed from a mare in the college clinic, entered with a history of repeated violent colics which disappeared as suddenly as they had appeared. Upon rectal

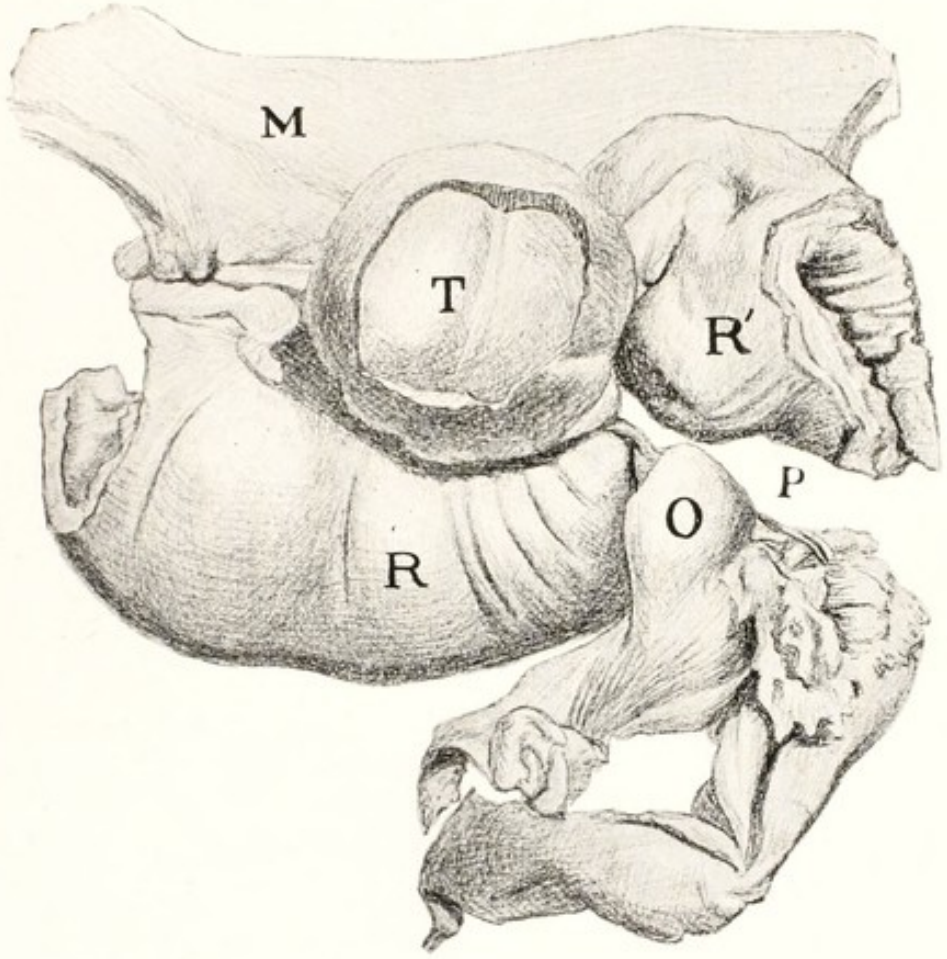


FIG. 58. Same as Fig. 57, Viewed from the Left Side.

M, Mesentery; *T*, cyst after portions of mesentery have been removed; *R*, the incarcerated segment of rectum; *R′*, the rectum upon the anal side of incarceration; *O*, ovary; *P*, the pedicle of the cyst.

palpation the large cyst, 20 cm. in diameter, was readily recognized. Laparotomy was performed, the cyst grasped and held firmly against the laparotomy incision while the liquid was allowed to escape externally through a trocar. The collapsed cyst was then removed through a much smaller incision than would have been necessary otherwise. Recovery was uneventful.

The par-ovarian tumor illustrated in Figs. 57-59 caused severe

obstruction colic, for which the mare was entered in our clinic after several hours of suffering. I could pass my hand into the rectum freely for about 18 inches, where the gut abruptly constricted to such a degree that only the index finger could be passed. There was no swelling or other evidences of disease of the intestine. The rectum gave the tactile sensation of being tightly corded. Without knowledge of the occurrence of such cases, I failed to make a diagnosis and the mare promptly succumbed.

The autopsy revealed a large par-ovarian cyst with a long, slender, cordiform pedicle. The cyst had fallen across the rectum from the right, pushing the mesentery before it. The long pedicle had

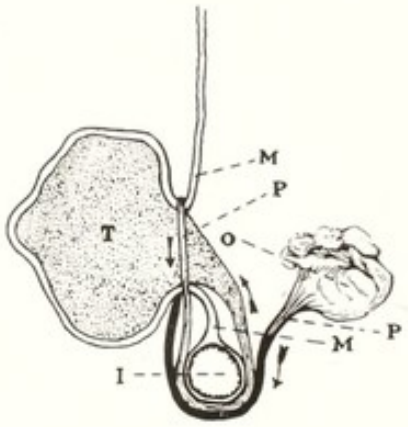


FIG. 59. Schematic Illustration of the Method of Incarceration of the Rectum in Figs. 57, 58.

I, Rectum; *M*, *M*, mesentery; *P*, pedicle of tumor at its origin near the ovary, *O*; *T*, par-ovarian tumor or cyst.

passed beneath the rectum, dropped over the cyst, covered by the mesentery, and had become looped about it in the form of a "half-hitch." In a brief space of time this caused distension of the gut upon the cranial side of the cord, drawing the loop more tightly.

There is no known limit to the size which ovarian or par-ovarian cysts or solid tumors may attain. Richmond[165] reports a pedunculated multilocular cyst in a mare, attached "a few inches anterior to the ovary" (par-ovarian cyst?) which weighed 150 pounds.

The most outstanding clinical expression of the presence of large ovarian, or par-ovarian tumors is the sudden advent of violent pain, like that of strangulated hernia. Should the incarceration not be too fixed in character, like that in the first case mentioned above, the incarceration may spontaneously cease and the animal suddenly recover, with the probability of repetition. When the incarceration is not promptly overcome, spontaneously or surgically, death occurs early.

I have not observed this species of intestinal incarceration in the cow, and it is probably very rare, but is manifestly possible with a large ovarian cyst, such as that shown in Fig. 60.

Rectal palpation is a prudent measure in all cases of severe abdominal pain not fully understood otherwise. When, in addition, the

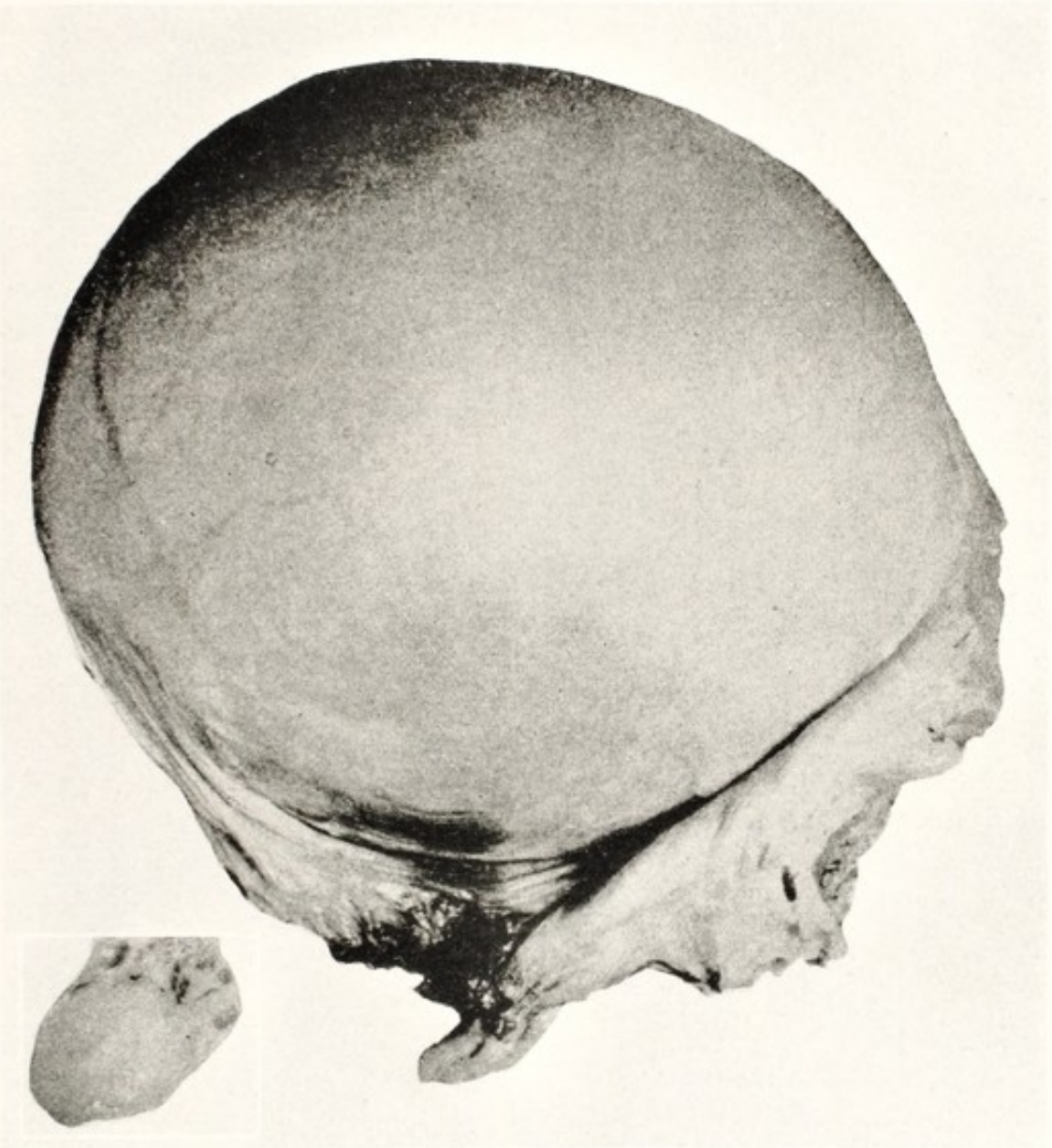


FIG. 60. Large Ovarian Cyst from Pregnant Cow, with a Normal Ovary (below), Containing a Corpus Luteum.

patient is a female, and the cause of the pain seems obscure, rectal palpation is definitely advisable. In cases of incarceration of the rectum by pedunculated tumors of the ovaries or by par-ovarian cysts, the first and most significant finding is the abrupt incarceration of the rectum giving the tactile sense of closure of the gut by an encircling cord. There is no swelling of the rectal walls and no evi-

dences of adhesions or of compression of the rectum by pelvic abscesses. Turning then to the palpation of the genital tract, and carefully tracing each cornu to its respective ovary, the nature of the obstruction is authentically determined. If an ovarian tumor or cyst is at fault, the gland itself is directly involved; if a par-ovarian cyst be present, the ovary is in its normal position and not materially involved in the incarceration, but in the immediate vicinity of the ovary, between it and the oviduct, is given off the telltale cordiform pedicle of the cyst, which can be followed around the rectum.

With a history of recurrent colics in the female, the possibility of ovarian or par-ovarian tumors should always be considered in the examination of the patient. If an ovarian tumor is present, it is probably hanging far forward in the abdomen and it may be necessary to draw it into the pelvis through traction upon the cervix or cornu.

When in the course of the examination of the genital organs of breeding mares or cows, such tumors are discovered, their timely removal is indicated. If neglected they may at any time cause fatal injury. They are not liable to cause peril in a pregnant female. They are not known to incarcerate other viscera than the rectum, and in advanced pregnancy the tumor moves ventralwards and anteriorly away from that viscus.

It is not absolutely essential that the diagnosis be technically complete in all details. When it is authentically determined that an ovarian or par-ovarian cyst or tumor is present which because of its size and peduncular type may imperil the health of the animal, surgical interference is indicated. The type of operation needs careful consideration. Any attempt to rupture the angio-sarcoma of the ovary illustrated in Fig. 51 would have been doubly perilous from hemorrhage and from the emptying of malignant tissues into the peritoneal cavity. The peril would be avoided by paying due heed to the greatly enlarged uterine artery and limiting the operation to the removal by free laparotomy and adequate ligation of all the blood vessels. As a rule laparotomy is the safe approach in the entire group. Only in cases like that shown in Fig. 59 is it advisable to attempt the rupture or puncture of the cyst within the abdominal cavity, and it is extremely difficult to make certain that one is dealing with so simple a cyst. Prudence dictates that care be taken not to permit the escape of the contents of the tumor into the peritoneal cavity.

Benign Tumors of the Oviducts, Uterus and Cervix

Benign tumors of the derivatives from Mueller's duct seem to be extremely rare in the larger domestic animals. There are no ex-

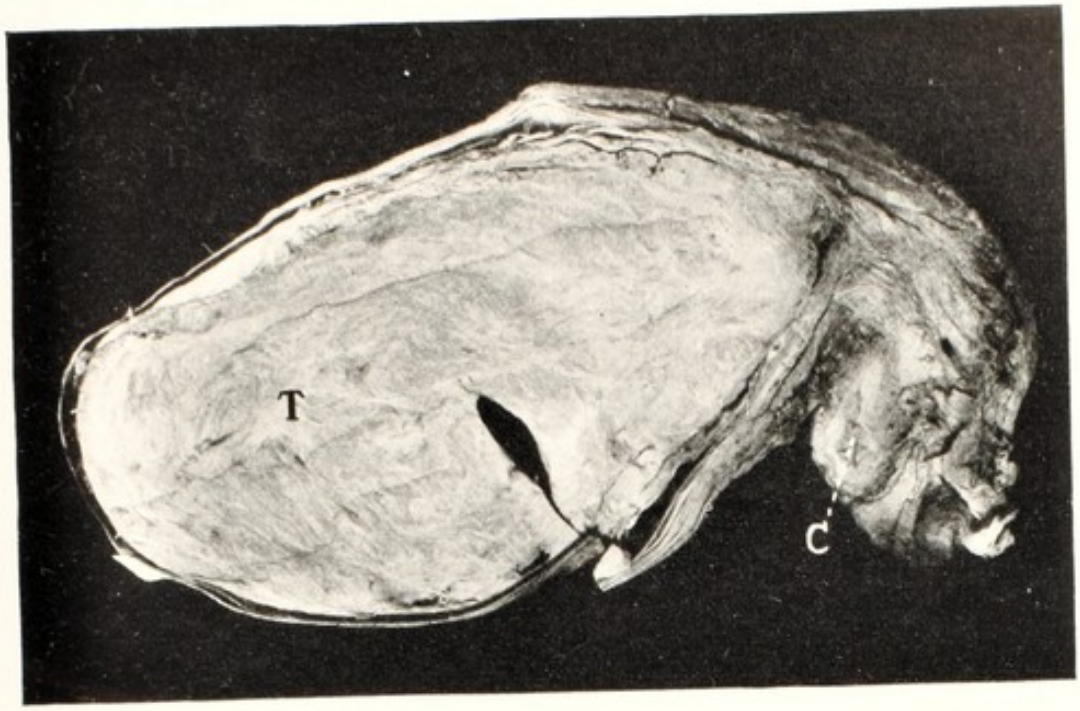


FIG. 61. Uterine Fibroma from Cow.
C, Cervix; T, tumor.

amples in the college collection of benign tumors of the oviducts or of the cervix, and but two of the uterus (cornua included). The fibroma from the uterus of a cow, shown in Fig. 61, is from the abattoir and hence without clinical history. It is oblong, 4 x 7 inches, and is located near the ovarian pole of the affected tube. The neighboring uterine walls show no indications of changes due to its presence.

The myom shown in Fig. 62 is from a mare which had appeared



FIG. 62. Equine Uterine Fibroma Causing Prolapse of the Uterus and Death of the Mare.
The dark periphery is due to hemorrhage, presumably caused by the uterine prolapse.

well up to the day of her death. She was at work when without warning she exhibited severe pain with violent expulsive efforts which forced the eleven-pound tumor out through the vulva. Entered in the clinic of Professor Frost, the tumor was removed, the resulting incision in the uterus sutured, and the organ replaced. The mare died a few hours later owing to hemorrhage from the uterine artery which had been ruptured when the uterus was prolapsed.

A cow, in which a uterine fibroid of large size was diagnosed clinically, produced several calves from the other cornu and then became sterile. It was not determined what, if any relation the tumor bore to the sterility.

Benign uterine tumors in the mare and cow, where rectal palpation is freely available, may generally be diagnosed clinically. Ordinarily the tumor grows slowly, causes no discharge or recognizable irritation and is not painful upon manipulation. It may, or may not bar fertility in a mechanical way, according to volume and location. Rarely a very small fetus macerates and the small bones lie embedded in desiccated pus, in such a manner as to closely resemble a uterine tumor. I have met one such case in a heifer. The leg bones were about 1 cm. long. Had the fetal cadaver been larger, the bones would probably have been palpable. Rarely, confusion in diagnosis might arise in instances of desiccated hematom in the uterus.

When clinically identified in valuable breeding animals, the tumor may be removed in those cases in which the patency of the uterine cavity may be retained. If but one cornu is involved, it, with the corresponding oviduct and ovary, may be removed and the female continue to breed from the remaining horn.

Fibroid vulvo-vaginal tumors, usually pedunculated, are not extremely rare in heifers. Their point of attachment is generally at or near the hymeneal ring. They usually protrude from the vulva when the animal is recumbent. They may offer some obstruction in labor and are very liable to become torn during calving and invite infection. They should be removed by incarcerating the pedicle in a ligature. It is best to use a needle with a strong suture of silk or linen, pass the needle through the base of the tumor and ligate the pedicle tightly. The tumor may then be excised at a safe distance from the ligature.

The pedicle of the tumor involves the vaginal walls in such a manner that its removal by simple excision or by ecrasement, invites hernia through the weakened area.

There appear occasionally in heifers epithelial tumors of the vulva which clinically suggest contagion. Thus in a group of 26 unbred heifers, 15 developed vulvar tumors. The new growths were upon

the vulvar muscosa, largely at the superior commissure or about the meatus urinarius. They varied in size up to four inches in diameter, and some of the larger ones protrude from the vulva when the heifer was recumbent. They caused no visible distress or injury. A few of the largest ones were removed after ligating their bases. The tumors had appeared during winter, while the heifers were in stable, and those not removed disappeared spontaneously after the animals were turned to pasture.

The suggestion of contagiousness is apparently supported by the occurrence of the definitely transmissible venereal tumors of dogs, described in Chapter XXV.

Benign Tumors of the Penis, Prepuce and Sheath

Benign tumors of the penis, prepuce and sheath are comparatively common in horses and cattle. They sometimes interfere with coitus.

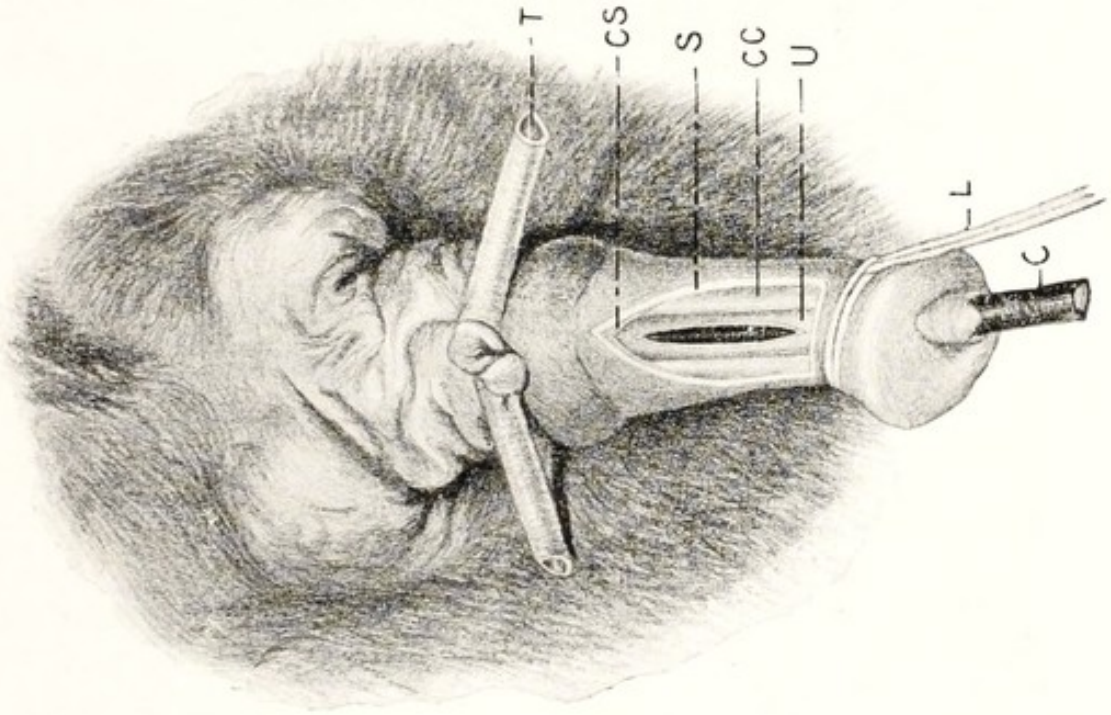


FIG. 63. Amputation of the Penis of the Horse, I.

T, Elastic ligature; *CS*, corpus spongiosum; *S*, skin; *CC*, corpus cavernosum; *U*, urethra; *L*, ligature; *C*, catheter.

They usually appear in young animals and are of the epithelial type. They sometimes bleed freely, especially after attempted coitus. In the horse, with the secretion of much sebum within the prepuce, its accumulation may be favored by the presence of the tumor and a large collection of fetid sebum with pus follow. Such tumors rarely attain a volume sufficient to cause phimosis or paraphimosis.

Their early removal is advisable. When small they may be safely

excised. If large, or if so located that excision will probably be followed by cicatricial deformity of the penis, amputation should be used instead. The technic of amputation illustrated in Figs. 63-65 has been satisfactory.

The operation is performed upon the recumbent animal, under general or local anaesthesia. The upper hind limb is so secured that it cannot obstruct access to the field of operation. The point of amputation is to be determined by the location and extent of the tumor. The amputation of the entire glans of the bull or stallion does not interfere materially with breeding. The operation is to be conducted under the usual rules of surgical cleanliness.

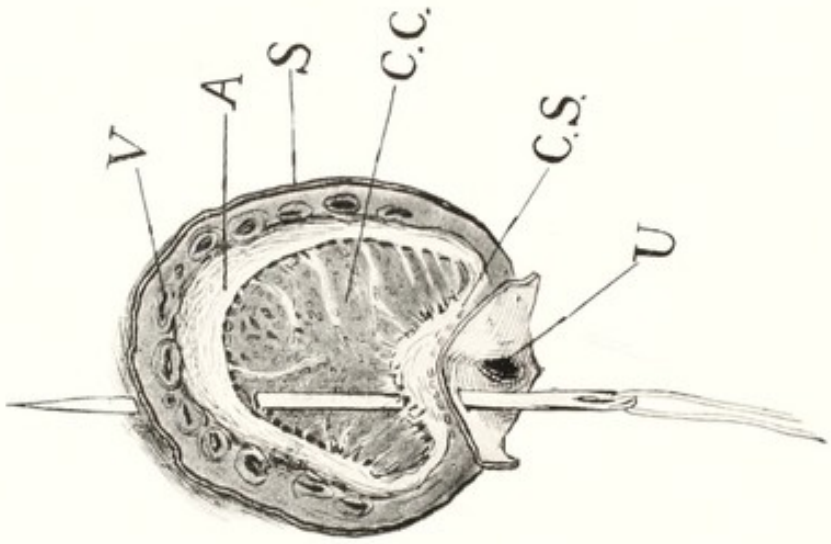


FIG. 64. Amputation of the Penis of the Horse, II.

Showing the method of suturing the stump. V, Dorsal vessels; *A*, fibrous tunic of the corpus cavernosum; *S*, skin; *CC*, corpus cavernosum; *CS*, corpus spongiosum; *U*, urethra.

The penis of the horse is to be drawn out of the sheath and prepuce with the hand and grasped by an assistant in the vicinity of the preputial ring, a catheter is introduced well beyond the selected point of amputation, as indicated at T, Fig. 63, and a piece of tape or cord looped about the distal end of the penis, as shown at L. A triangular incision is then made upon the ventral surface of the organ, about 4 cm. long by 3 cm. wide, the base of the triangle being directed toward the terminal extremity of the penis, as indicated in Fig. 63. The incision is carried through the skin, S, the corpus spongiosum, CS, and along the corpus cavernosum, CC, down to the urethra, U. The tissues within the triangle are dissected away down to, but not including, the urethra. A longitudinal incision through the urethra is then made along the median line from the apex of the triangle, and

a series of interrupted sutures is inserted as shown in Fig. 65. The sutures are passed through the urethral wall and the skin, but not through the corpus cavernosum. The wounded surfaces are completely covered by the urethral mucosa and the skin.

The catheter is then withdrawn and the penis excised obliquely, the line of excision extending from the urethral or ventral side toward the dorsum of the organ. A straight needle, carrying a strong silk suture, is passed through the urethral wall and the fibrous capsule of the corpus cavernosum, and then across, not through the cavernous

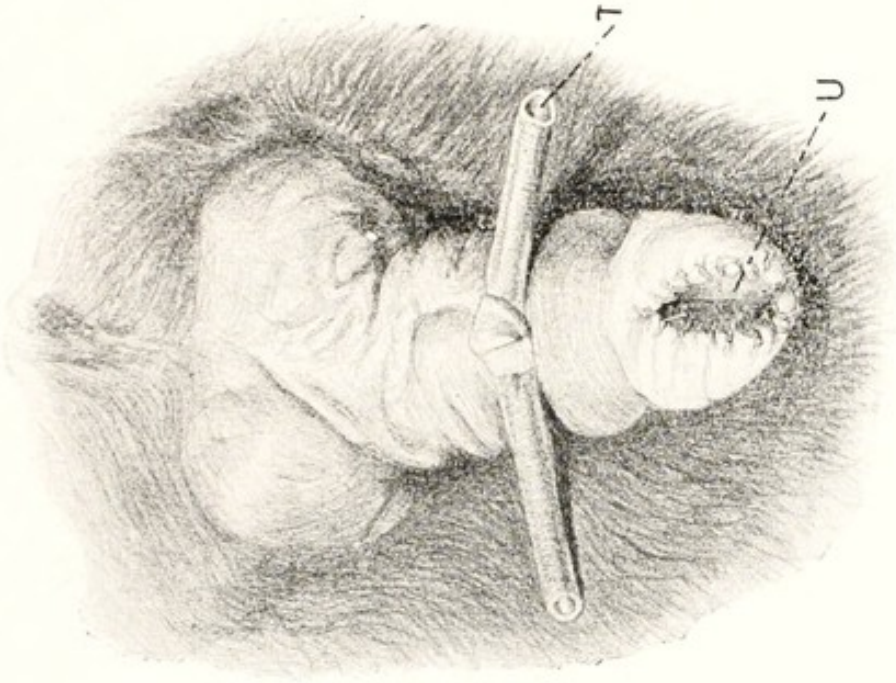


FIG. 65. Amputation of the Penis of the Horse, III. Completed operation. *T*, Elastic ligature; *U*, urethra.

tissue, to the dorsal portion of the fibrous capsule, and passed through it and the skin. When the sutures are tied firmly, the urethral mucosa and the skin are brought into appositon and the cavernous tissue so compressed that hemorrhage is barred.

The principles laid down for the horse are readily applied to amputation of the penis of the bull. The exposure of the bull penis for operation is best attained by grasping the organ, covered by skin, at the point of insertion of the retractor penis muscle and exerting firm traction upon the organ until the resistance of the retractor is overcome and the sigmoid flexture of the penis eliminated, freely exposing the organ.

SECTION IV

SYSTEMIC AND ORGANIC INFECTIONS WHICH MAY INVADE THE GENITAL SYSTEM AND LEAD TO INTERFERENCES WITH REPRODUCTION AS COMPLICATIONS OF THE PRIMARY DISEASE

Chapter XVIII

ANIMAL PLAGUES

ANY infections involving organs adjacent to the genital system may extend to the reproductive organs by continuity and interfere with reproduction. Some infections, like bursatte and actinomycosis, to be described below, may invade the genital organs primarily and endanger the breeding life of the animal. General systemic diseases invade all organs of the animal body through the blood stream and may interfere definitely with reproduction. Such invasions of the reproductive system are not usually extensively studied in connection with the great animal plagues. The chief attention is riveted upon the imminent peril to the life of the adult.

In the preceding pages it has been held that reproduction is a luxus function, over which the physical life of the individual holds precedence. All highly dangerous contagious diseases are notably perilous for pregnant females, because of the extra burden which they carry in the sustenance of the intra-uterine young. Hence in virulent animal plagues the mortality is definitely higher in pregnant, than in non-pregnant females. In such rapidly mortal diseases as anthrax, death of the pregnant female occurs so swiftly that it cannot readily be determined whether the intra-uterine young succumbed prior to that of the mother. In less virulent diseases the intra-uterine young often perish and are expelled, after which the mother may enjoy increased hope of recovery because the burden of pregnancy has been removed.

Taussig[193] states that pregnant women, suffering from typhoid fever, cholera, scarlet fever, smallpox, and similar maladies abort in a ratio of about 50 per cent. He emphasizes the great peril to pregnant women in the virulent influenza epidemic of 1918. The Taussig list includes a large ratio of virus diseases.

Such animal plagues as hog cholera, foot-and-mouth disease, equine contagious pneumonia (Brustseuche), and especially the equine malady designated influenza, epizootic cellulitis or "pink eye" are notable for the high ratio of abortion in affected pregnant females. The abortion rate in rinderpest and in sheep pox is high. These, like the list of Taussig, are largely virus diseases. If the aborting female survive, the injury to the uterus may result in subsequent sterility.

It is not known to be essential that the infection penetrate the uterus and attack the placenta or fetus. The disease may so depress

the resistance that other microorganisms are enabled to cause the death of the fetus, or the fetal death may result from toxins caused by the disease.

Any general consideration of the effect of animal plagues upon reproduction does not fall within the scope of this treatise. There are, however, some diseases not primarily genital, which merit discussion.

It should be noted here that especially to American readers, the significance of "animal plagues" especially regarding cattle, has undergone great change during the past seventy years. Thus in 1879, Walley[214] wrote an extended treatise upon "The Four Bovine Scourges" which included pleuro-pneumonia, foot-and-mouth disease, rinderpest and tuberculosis. The first three are now virtually unknown and the fourth has become largely controlled in North America; numerous writers at this time place as cattle plague No. 1 the so-called contagious abortion.

Chapter XIX

GENITAL TUBERCULOSIS

GENITAL tuberculosis of dairy cattle constitutes a highly important phase of that malady, and is one of the most dangerous types from the standpoint of the spread of the disease. It causes, or is linked with much sterility and abortion. Plum[157] states that in Denmark in a series of 834 successive fetal membranes examined he found tuberculous abortion in 1.79 per cent. In the abattoir in Denmark uterine tuberculosis shows a ratio of 2 per cent. Plum studied a total of 212 tuberculous abortions. He found an abundance of tubercle bacilli in the fetal membranes which were recognizable by staining, culture, and inoculation of laboratory animals. The macroscopic appearances of the fetal membranes and of the contents of the uterochorionic space (the exudate of contagious abortion) did not visibly differ from those appearing in the presence of *Bacillus abortus*. Most of the tuberculous abortions, like abortions in general, were observed at 250 to 273 days.

Animal inoculations were made in 162 cases, of which the animals died of sepsis in 10. One hundred and forty-five showed conclusive evidence of mammalian tuberculosis, 6 were of the avian type, and one uncertain. The uterine lesions differed markedly in the two types. Those of the mammalian type were essentially like those illustrated in Figs. 78-85. The avian type caused much less injury to the genital tract, and its lesions were confined generally to pea-sized abscesses in the uterine submucosa. Hence mammalian uterine tuberculosis early renders the cow hopelessly sterile, while the cow affected with uterine tuberculosis of the avian type retains her power of conception but aborts the pregnancy, or, abortion failing, the calf may be born viable but having tuberculosis.

Nieberle[149] describes congenital tuberculosis in a series of 56 calves which showed conclusive evidence, by primary lesions of the portal lymphatics and the liver, that the infection had occurred during fetal life through the umbilical vein.

From my clinical observations I may relate two cases which serve to illustrate the significance of genital tuberculosis in the spread of the disease. A purebred Holstein-Friesian cow, represented as negative to repeated subcutaneous tuberculin tests, and infertile for a considerable period, was sold into a highly valued herd, believed to be tuberculosis-free. After she had been mated repeatedly and un-

successfully with the herd bull I was asked to examine her because of sterility, and diagnosed advanced uterine tuberculosis. The herd bull, in good general health, was unable to copulate owing to phimosis. Several tumors were readily identified alongside the penis, as shown in Figs. 72-73. Incision of one of these revealed macroscopic evidence of tuberculosis, and laboratory search verified the clinical diagnosis. The bull responded to the subcutaneous tuberculin test. Some years later there were apparently good reasons for believing that the test had been made by an unreliable officer, and that had the cow been properly tested during the earlier stages of the disease, she would have reacted. At the date when I observed her, the disease was so advanced that reaction to the subcutaneous tuberculin test was highly improbable.

In a second instance I was asked to make a survey of a small, highly valued Guernsey herd because of defective reproduction. The herd had a history of a high ratio of positive reactors to the semi-annual, subcutaneous tuberculin test, regardless of the fact that all reactors were promptly eliminated and the premises disinfected. An aged cow revealed extreme uterine tuberculosis, as illustrated in Figs. 78-79. Each of the two cows was unusually dangerous as a spreader and neither was diagnosable by the subcutaneous tuberculin test. Both cows discharged from the genital tract important quantities of presumably highly virulent tuberculous matter.

Genital tuberculosis of cows carries an unusual peril for the herd bull. Prior to the excellent work of the federal and state authorities in the elimination of tuberculosis, primary penial (coital) tuberculosis in herd bulls was a common occurrence in concentrated dairying regions. The danger is greater for the bull than for the cow, because the cow is not hindered in copulating, while the bull, in many but not all cases becomes incompetent to copulate because of tuberculous phimosis. The subcutaneous tuberculin test has proven one of the most if not the most valuable diagnostic procedure known to veterinary science. Unfortunately this has led some veterinarians to disregard the yet valuable procedure of clinical examination, and although the failures of the tuberculin test are comparatively few, they occur usually in the most dangerous cases. Competent clinical search of the genital organs becomes of unusual value in the control and elimination of the disease.

Prior to the awakening of an important interest in the pathology of reproduction, veterinary literature regarding genital tuberculosis was scant and vague. Writers had accorded genital tuberculosis only a minor place. Its occurrence was admitted, but directly or inferen-

tially it was regarded as rare. A familiar statement was that it caused nymphomania, but nymphomania is increasingly abundant, largely in tuberculosis-free herds. The veterinarian does not usually search for genital tuberculosis. Clinically it is chiefly when he concentrates his attention upon genital diseases in connection with sterility that he recognizes the importance of genital tuberculosis.

Tuberculosis of the testicles and epididymes is not common, but occasional cases arise in various species of domestic animals. In comparatively limited observations in the field I have recognized no case of tuberculosis of the epididymis, and but one instance of testicular tuberculosis. That occurred in a boar (Fig. 69).

Among the cases of orchitis in bulls studied by Richter[168] were 7 due to tuberculosis, or 12 per cent of the total. It is difficult of clinical differentiation from orchitis due to other causes. Richter finds that the tuberculin test is unreliable, as I have found in genital tuberculosis of cows. Apparently testicular, like uterine tuberculosis, is largely dependent upon a saturation of the system by the infection in that degree which inhibits the typical reaction to tuberculin. As a rule, the clinical diagnosis of tuberculous orchitis is not of great importance. When definite orchitis is present the animal is ordinarily doomed as a sire and the gland may be removed surgically or at autopsy and studied histologically.

Richter relates an instance in which the bull was continued in service after the advent of the orchitis, which during the next year was followed by numerous cases of uterine tuberculosis among his cows, with extensive peritoneal adhesions. It is always perilous to use a sire suffering from orchitis, regardless of the underlying cause, and it becomes especially dangerous when due to tuberculosis. Fortunately, as Richter observes, in many cases of orchitis and epididymitis no spermatozoa or other secretions or excretions from the testicles are ejaculated: the semen consists of the secretions from the accessory glands, seminal vesicles and prostate.

The rule is, without known exception, that orchitis is bilateral, regardless of the cause. The orchitis may not be clinically evident in one of the glands, but histological study reveals that the disease is bi-glandular. Hence the danger cannot be eliminated by the removal of the clinically diseased organ upon the assumption that the other is healthy. The sire should be promptly discarded.

Tuberculosis of the vas deferens, seminal vesicle or prostate occurs but rarely. Each would tend to interfere with the ejaculation of semen. The diagnosis would be based upon rectal palpation, plus the tuberculin test. Slaughter is indicated.

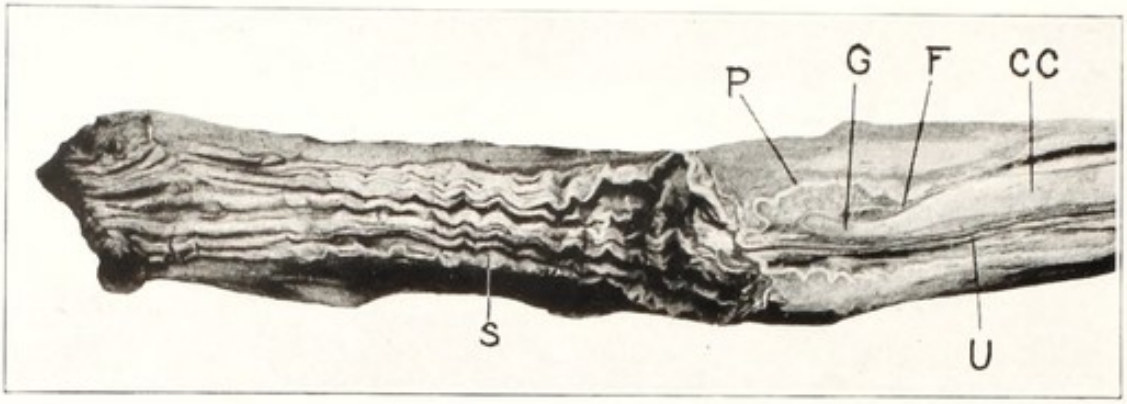


FIG. 66. Penis of Bull Calf with the Preputial Sac Unopened.

S, Sheath; *U*, urethra; *P*, borderline between prepuce and glans; *G*, glans; *F*, point at which fornix is located when the prepuce separates from the glans; *CC*, corpus cavernosum.

Penial tuberculosis is comparatively common in the bull and may involve the corpus cavernosum, corpus spongiosum, urethra, prepuce, sheath, and especially the lymph nodes along the course of the penis. The anatomy of the copulatory organs of the bull, and the mechanism of copulation, invite injuries to the epithelial layer of the prepuce. The protrusion of the penis from the sheath and prepuce during

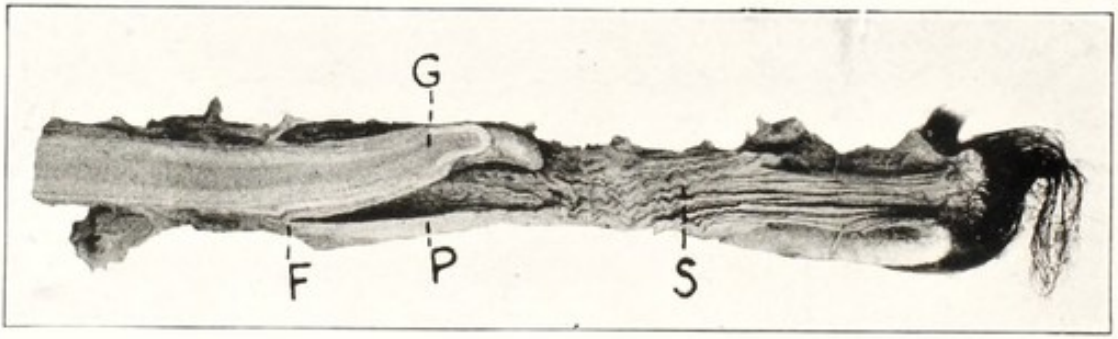


FIG. 67. Penis, Sheath and Opened Prepuce of Bull Calf.

F, Fornix; *G*, glans; *P*, prepuce; *S*, sheath.

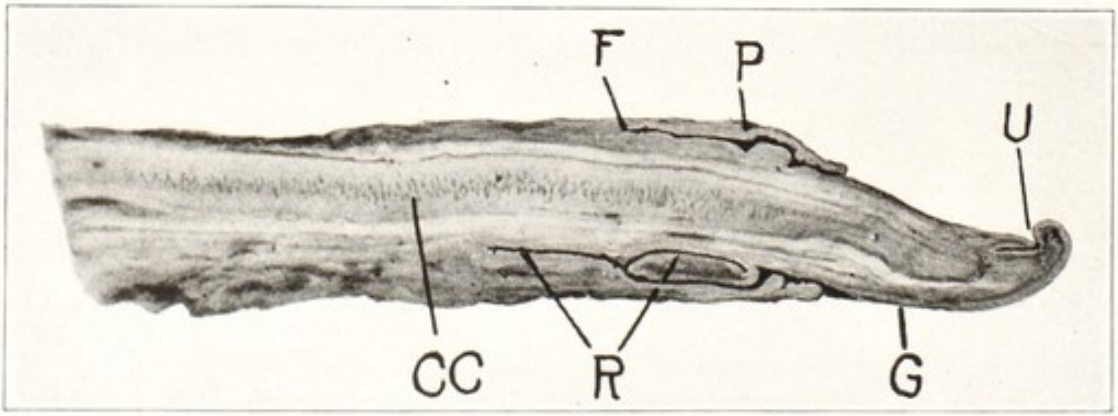


FIG. 68. Coital Rupture of the Prepuce of the Bull.

CC, Corpus cavernosum; *F*, fornix; *P*, prepuce; *G*, glans; *U*, urethra; *R*, rupture of the prepuce, the ruptured end being doubled back at the right.

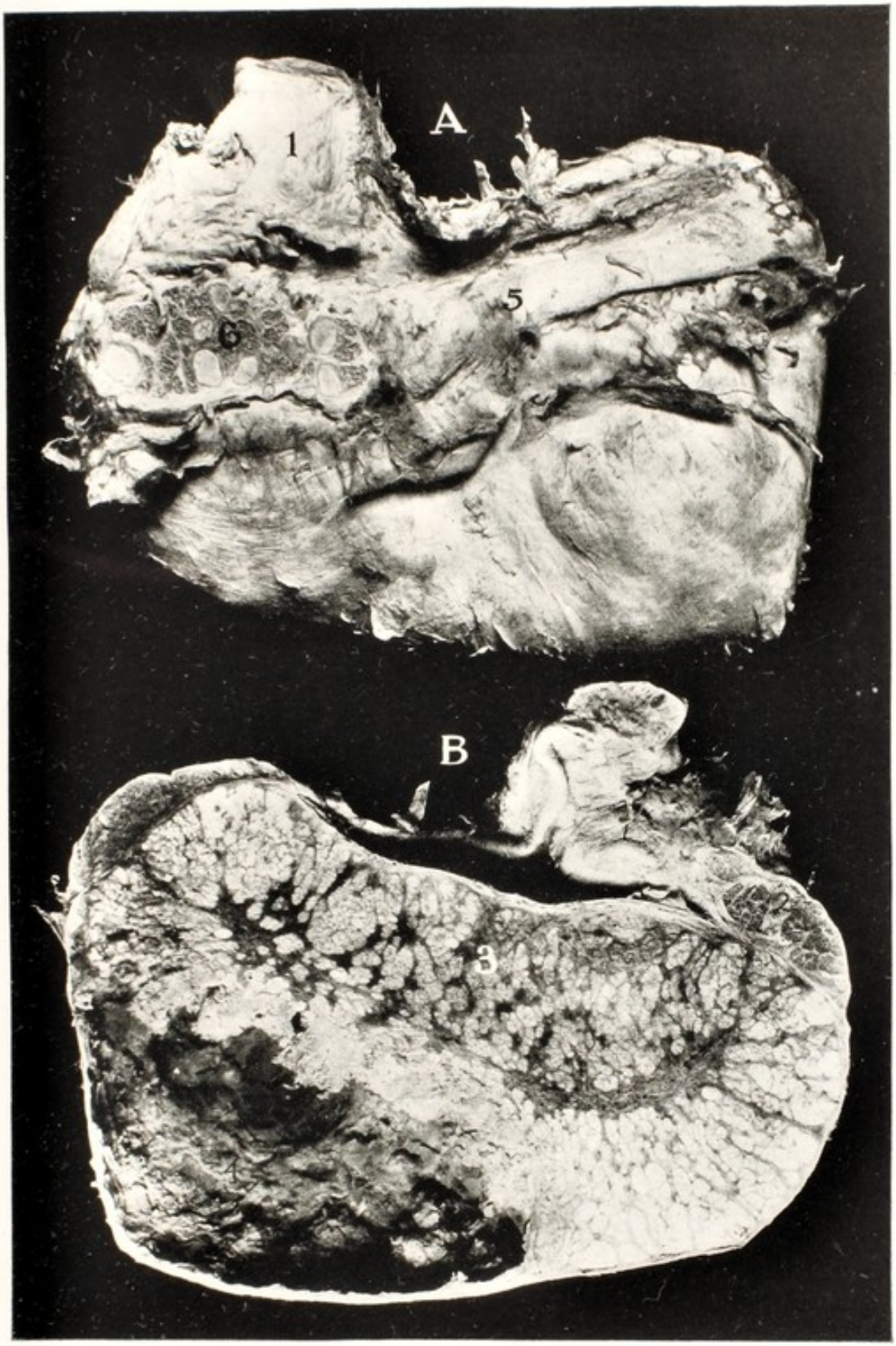


FIG. 69. Tuberculous Testicle in Sagittal Section. Boar.

A, The exterior of the gland; *B*, the sectioned surface. 1, Spermatic cord; 2 (at the right in *B*), head of epididymis; 3, tubercular parenchyma; 4, tubercular abscess; 5, body of epididymis; 6, section through epididymis showing tuberculous masses.

coitus is chiefly accomplished through the elimination of the sigmoid flexure by the momentary overcoming of the retractor penis muscle, RP, Fig. 74. The sheath (Figs. 66-67) exists as an independent organ at birth, while in ruminants and swine, the prepuce is a closed sac, firmly adherent to the glans, so that the penis can not be protruded at the time of birth. Later, as sexual maturity approaches, the parietal layer of the prepuce separates from the visceral and the glans penis becomes free. If the male is castrated very early, the opening of the prepuce is barred and the castrate unavoidably urinates in his sheath throughout life.

The coital thrust of the bull is so violent and the epithelial covering of the glans so delicate that there is constant danger of epithelial injury. This is rendered more probable by the usual presence upon the glans and the parietal layer of the prepuce of many nodules of the nodular venereal disease, Chapter XXVI, which is essentially universal in ruminants and swine. The summits of such elevations readily suffer abrasion. Epithelial injuries are also invited when the vulvar lips of the cow have adhering to them dried crusts of feces or of vulvar discharges. Owing to the violence of the copulatory thrust, perhaps most frequently when serving virgin heifers, in some of which there is marked vulvar hypoplasia, the prepuce is not rarely ruptured at the fornix, Fig. 68. If the cow has tuberculous genital disease, infection of the penis or prepuce is invited. Such primary tuberculous infection may involve any part of the copulatory apparatus.

I have clinically observed two cases of tuberculous infection of the glans and have seen others in the abattoir. A large Holstein-Friesian bull was entered in the clinic because for some months he had been

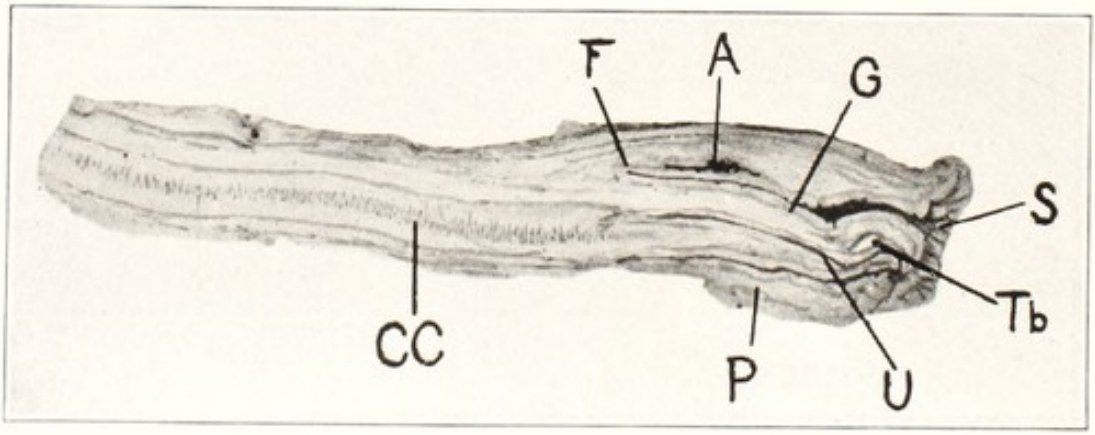


FIG. 70. Tuberculosis of the Glans and Prepuce of Bull.

F, Fornix; *A*, tuberculous abscess in prepuce; *G*, glans; *S*, sheath; *Tb*, tubercle at the tip of the glans; *U*, urethra; *P*, prepuce; *CC*, corpus cavernosum.

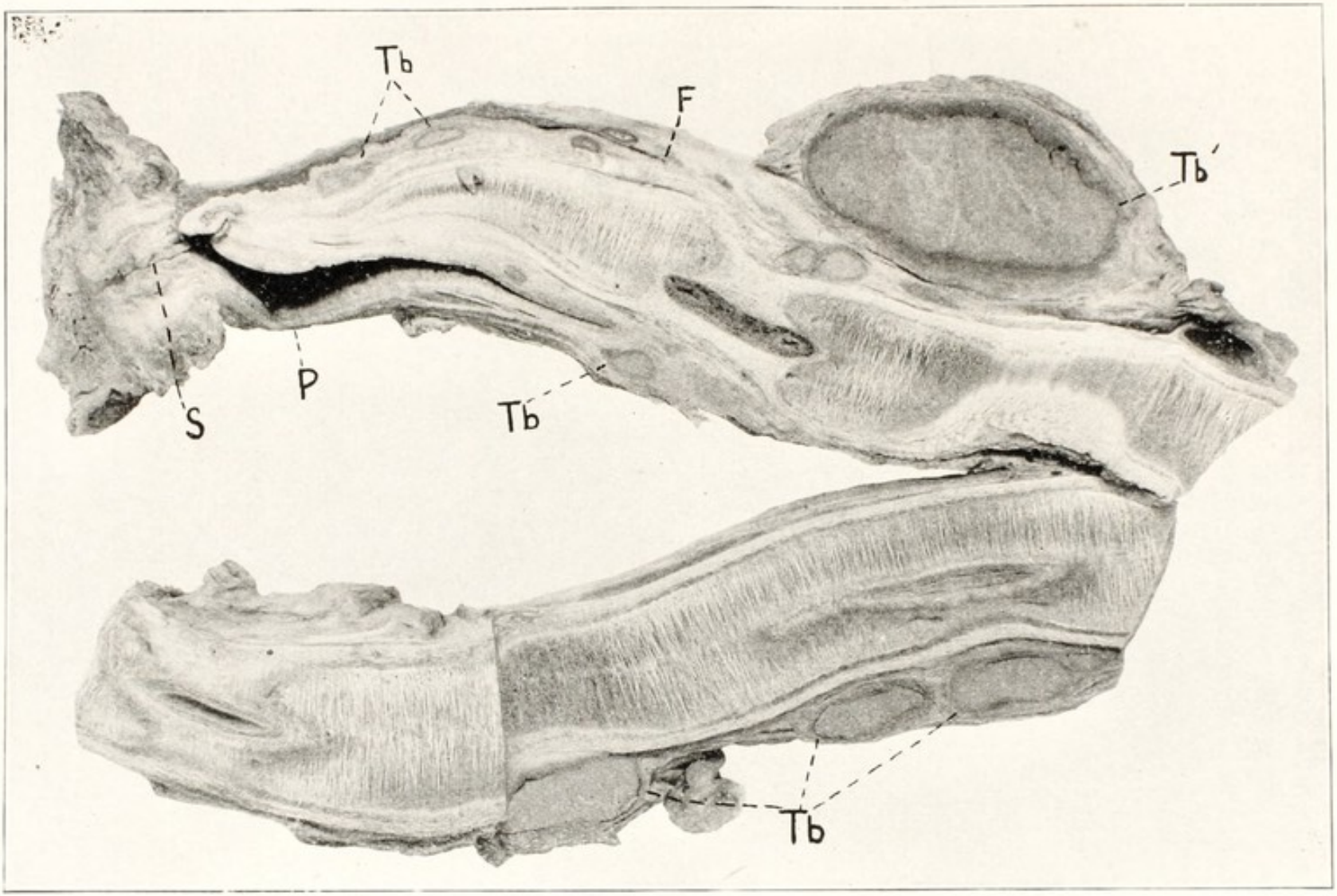


FIG. 71. Primary Penial Tuberculosis in Bull.
S, Sheath; *P*, prepuce; *F*, fornix; *Tb*, *Tb*, tuberculous areas.

unable to copulate with small females though quite able to serve large cows. Attempts at copulation were followed by hemorrhage. When the penis was exposed by forcibly effacing the sigmoid flexure, it was seen that the apex of the glans was deeply inflamed for a distance of about three inches, was much enlarged and bled readily upon touch. The epithelium was largely destroyed and the surface

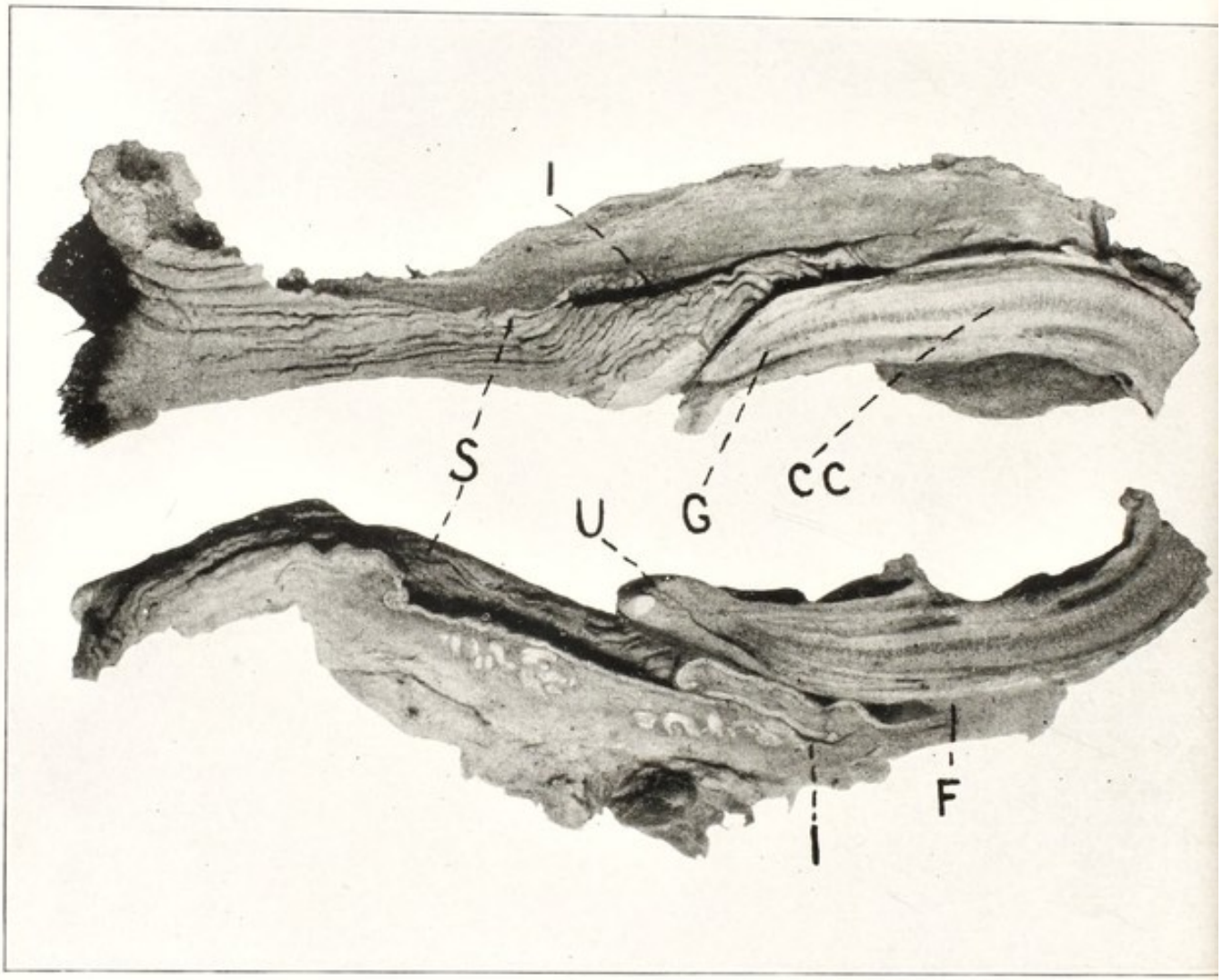


FIG. 72. Tuberculosis of the Sheath and Prepuce.

S, Sheath; *U*, urethra; *G*, glans; *F*, fornix; *CC*, corpus cavernosum; *I*, *I*, fissure into tuberculous mass.

covered by purulent exudate. The diseased tip of the glans was amputated and the laboratory diagnosis was that of tuberculosis.

The incident was prior to efforts by the state to eliminate tuberculosis, and the breeder was more interested in covering up the disease than in its control. I was advised that tuberculosis was rampant in the herd. The attitude of the owner barred any investigation regarding the transmission of the infection to cows served. So far as I have observed, he was apparently the most dangerous bull I have seen. An

interesting abattoir specimen is shown in Fig. 70 with tuberculous lesions at the tip of the glans. Submucous tuberculosis of the glans is shown in Fig. 71. The disease was brought to a crisis by the large tuberculous lesions about the prepuce and glans which caused phimosis.

I have one well defined specimen of tuberculosis of the sheath, shown in Fig. 72. An abattoir case, without clinical history, the specimen suggests that phimosis ensued early and caused the discard of the bull. Clinically the condition could probably not have been readily

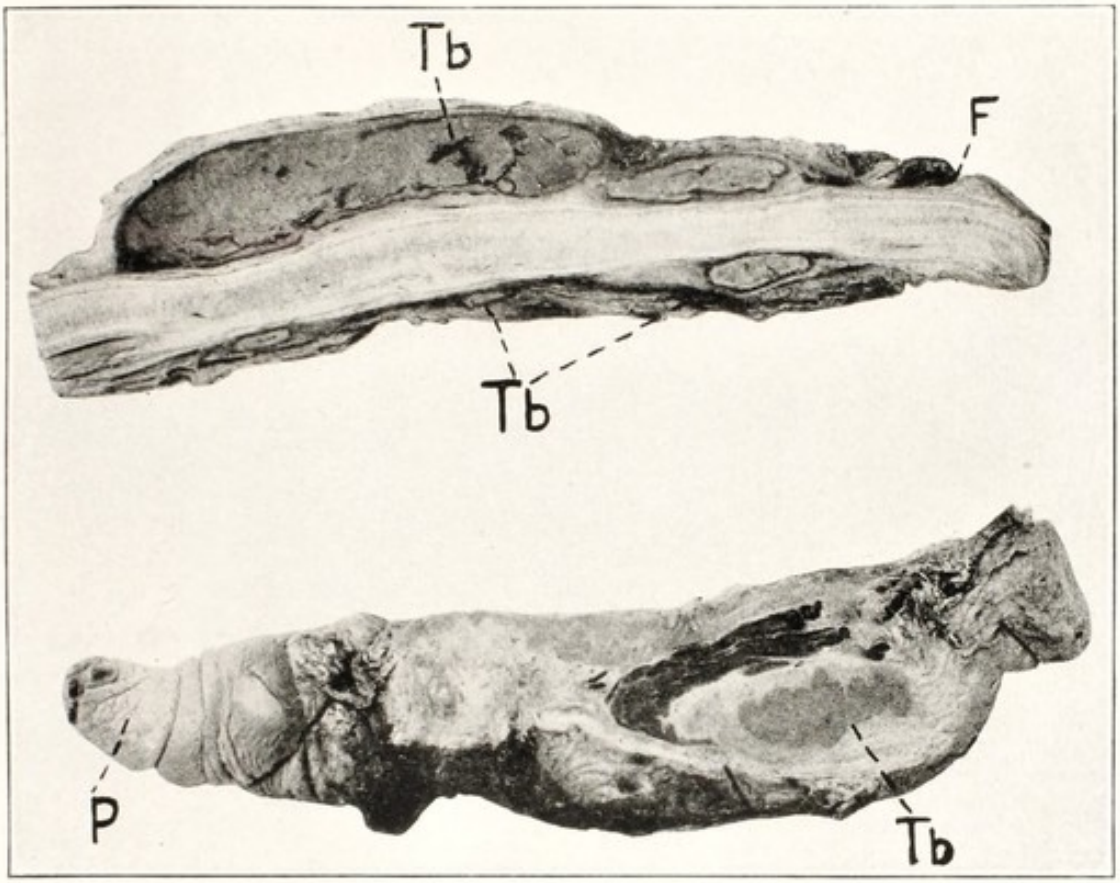


FIG. 73. Tuberculosis of Glans Penis and Prepuce of Bull.
P, Prepuce; *F*, fornix; *Tb*, extensive tuberculous masses.

differentiated from actinomycosis or other type of infection. It should have aroused suspicion and the tuberculin test been applied.

So long as a bull, with open lesions of the glans or prepuce, can protrude the penis and copulate he constitutes a serious menace. Accordingly it is highly important that care be exercised in making a diagnosis when disease of the penial sheath is involved.

Preputial tuberculosis is not common, despite the fact that the membrane is delicate and exposed to coital injury. Apparently the infection tends to become located beneath the mucosa, as in Fig. 73. Injuries to the prepuce apparently serve rather as an avenue of in-

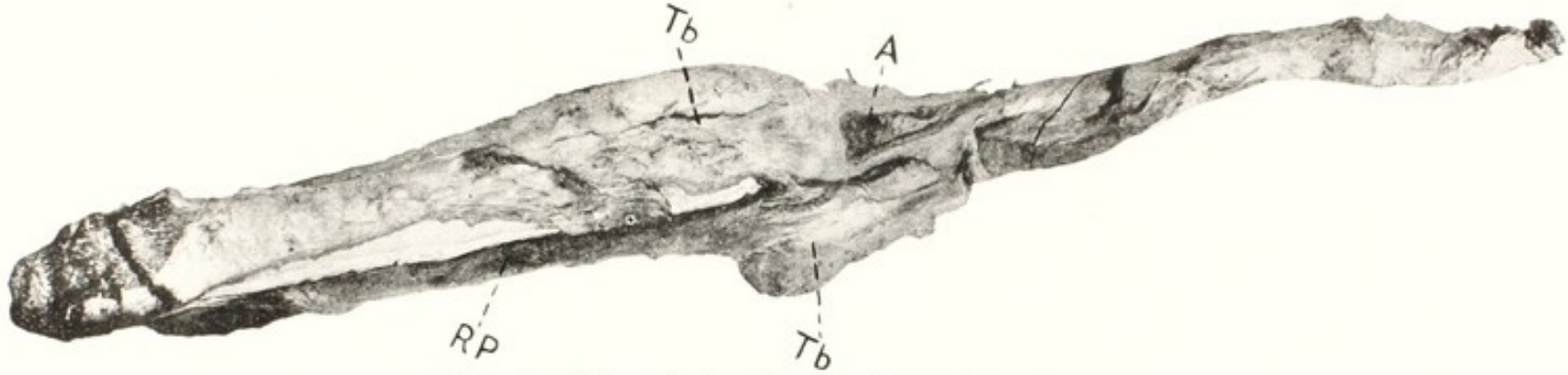


FIG. 74. Tuberculosis of Peripenial Lymph Glands.

G, Glans; *F*, fornix; *RP*, retractor penis muscle; *Tb*, *Tb*, tuberculous lymph glands concentrated about the sigmoid flexure and preventing protrusion of the penis.

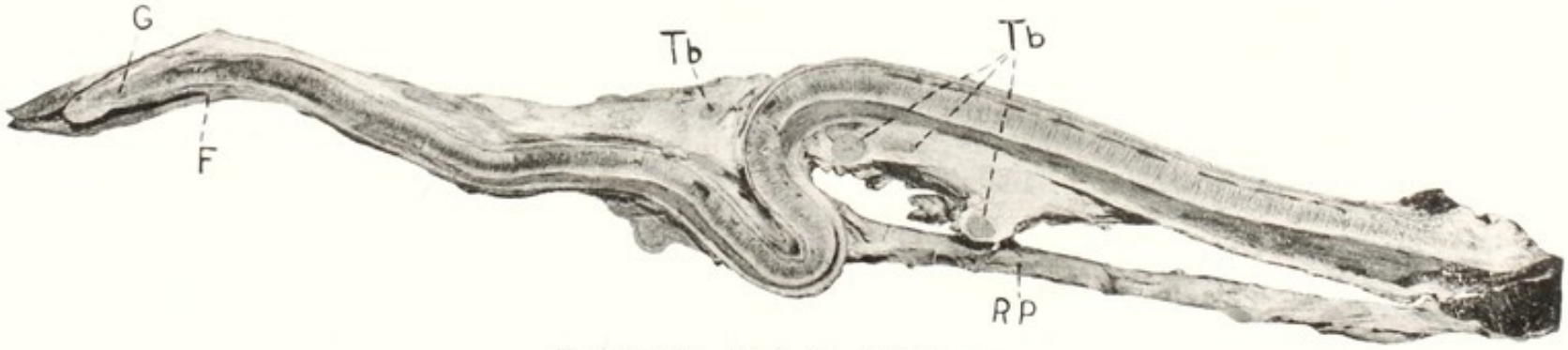


FIG. 75. Fig. 74 in Sagittal Section.

vasion to deeper tissues than as a seat of tuberculous lesions. When infection occurs and inflammation is established, the loose areolar tissue between the prepuce and the external dartoid sheath becomes involved, adhesions develop, and the penis is incarcerated. Clinically it is probably difficult to identify the character of the disease at first. When the disease is moderately advanced, copulation is liable to aggravate it greatly and give at first the appearance of a purely mechanical injury. In the presence of suspicious symptoms, the tuberculin test is indicated.

The most common form of genital tuberculosis in bulls is the tuber-

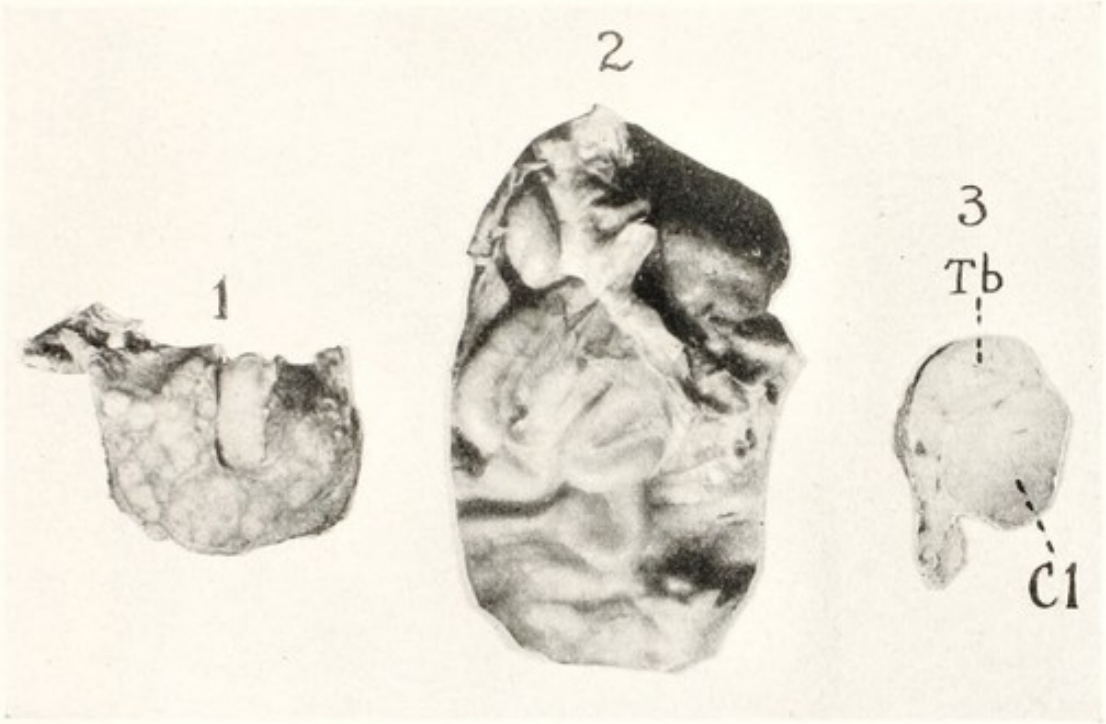


FIG. 76. Tuberculosis of Bovine Ovary and Oviduct.

1, Section through the oviduct; 2, oviduct thrown into coils by elongation due to tuberculosis; 3, ovary showing a corpus luteum, *Cl*, and a tuberculous mass, *Tb*, at the site of a ruptured ovisac.

culous invasion of the penial lymph glands, scattered from the margin of the sheath to or beyond the sigmoid flexure. The swellings of these glands are soon identifiable. They are usually small, as indicated in Figs. 71, 74, and 75. They are firm, not recognizably painful, and contain thick pus. Inferentially such infections of the lymphatics may be caused by various invaders, but in my observations all have been tuberculous. Hence in the presence of such lesions, a provisional diagnosis of tuberculosis is to be made and the tuberculin test applied.

Tuberculosis of the ovary is probably more frequent than that of

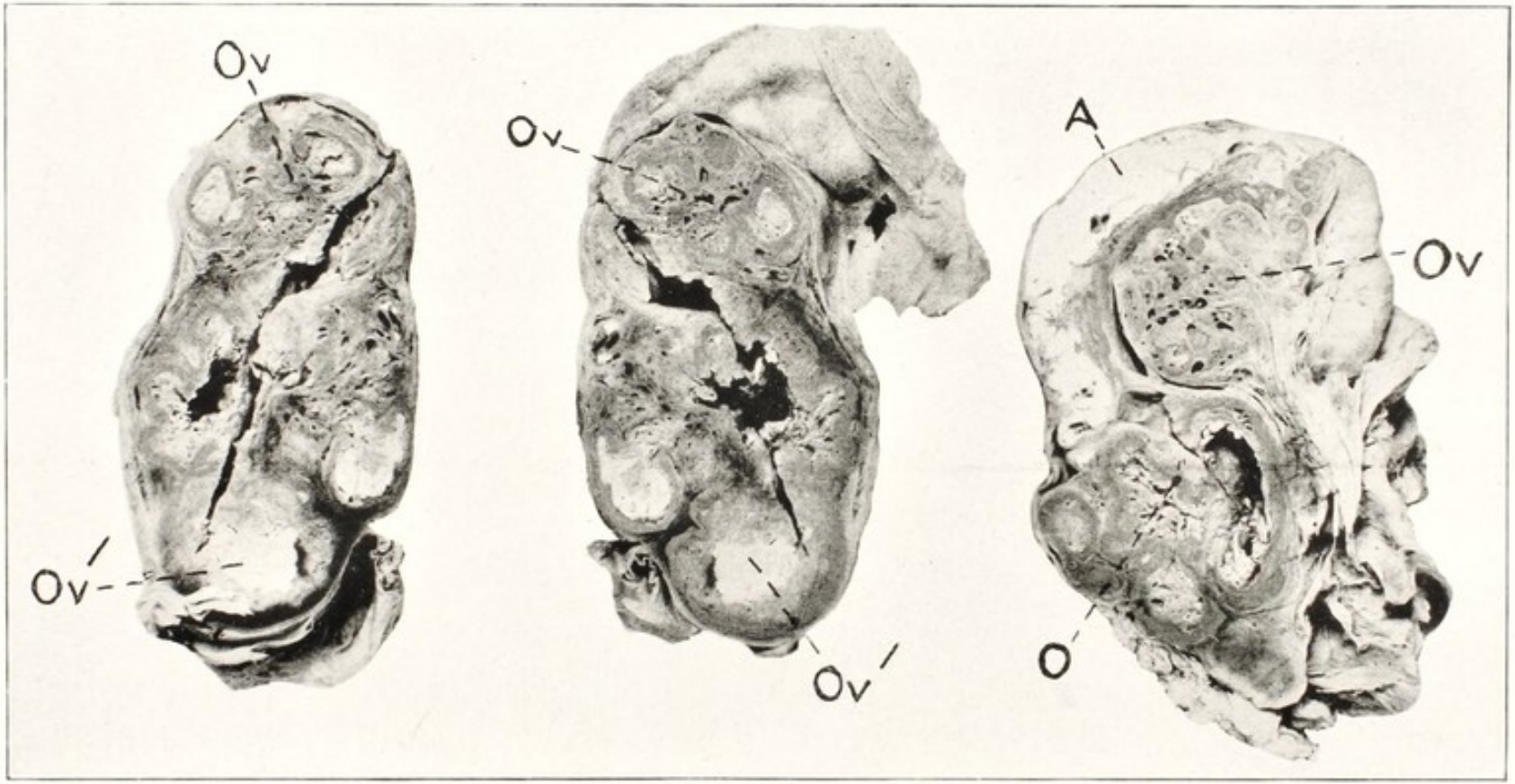


FIG. 77. Serial sections through ovary showing advanced tuberculosis.

Ov, Oviduct; *Ov'*, section through greatly enlarged abscessed pavillion of the oviduct; *O*, ovary; *A*, parovarian adipose tissue.

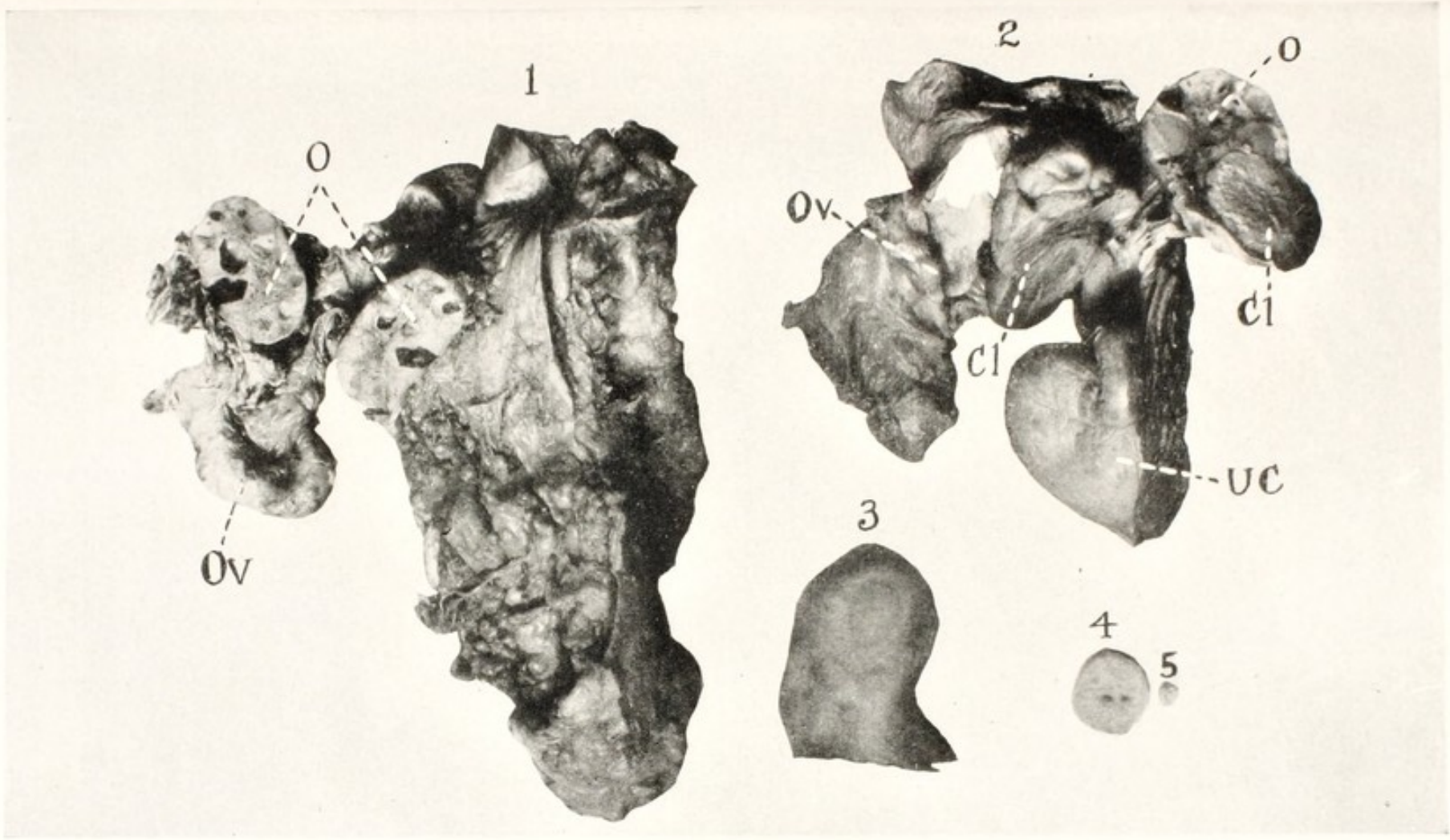


FIG. 78. Advanced Tuberculosis of Oviducts and Cornua.

1, *O*, Ovary in sagittal section; *Ov*, greatly enlarged oviduct; 2, *O*, ovary; *Cl*, corpus luteum; *Ov*, oviduct; *UC*, uterine cornu; 3, cross section of horn; 4, cross section of tuberculous cornu; 5, cross section of normal oviduct.

the testicle. The ovary is directly exposed when tuberculosis exists in other organs within the peritoneal cavity. The specimen shown in Fig. 76 suggests that the avenue of invasion was through the crater of the ruptured ovisac. The periodical rupture of the follicles serves as a wound through which the infection may readily enter. Fig. 77 illustrates advanced tubercular disease of oviducts and ovaries. Generally the oviducts are adherent and upon rectal palpation the tubercles are readily distinguishable. Ovarian tuberculosis is rarely

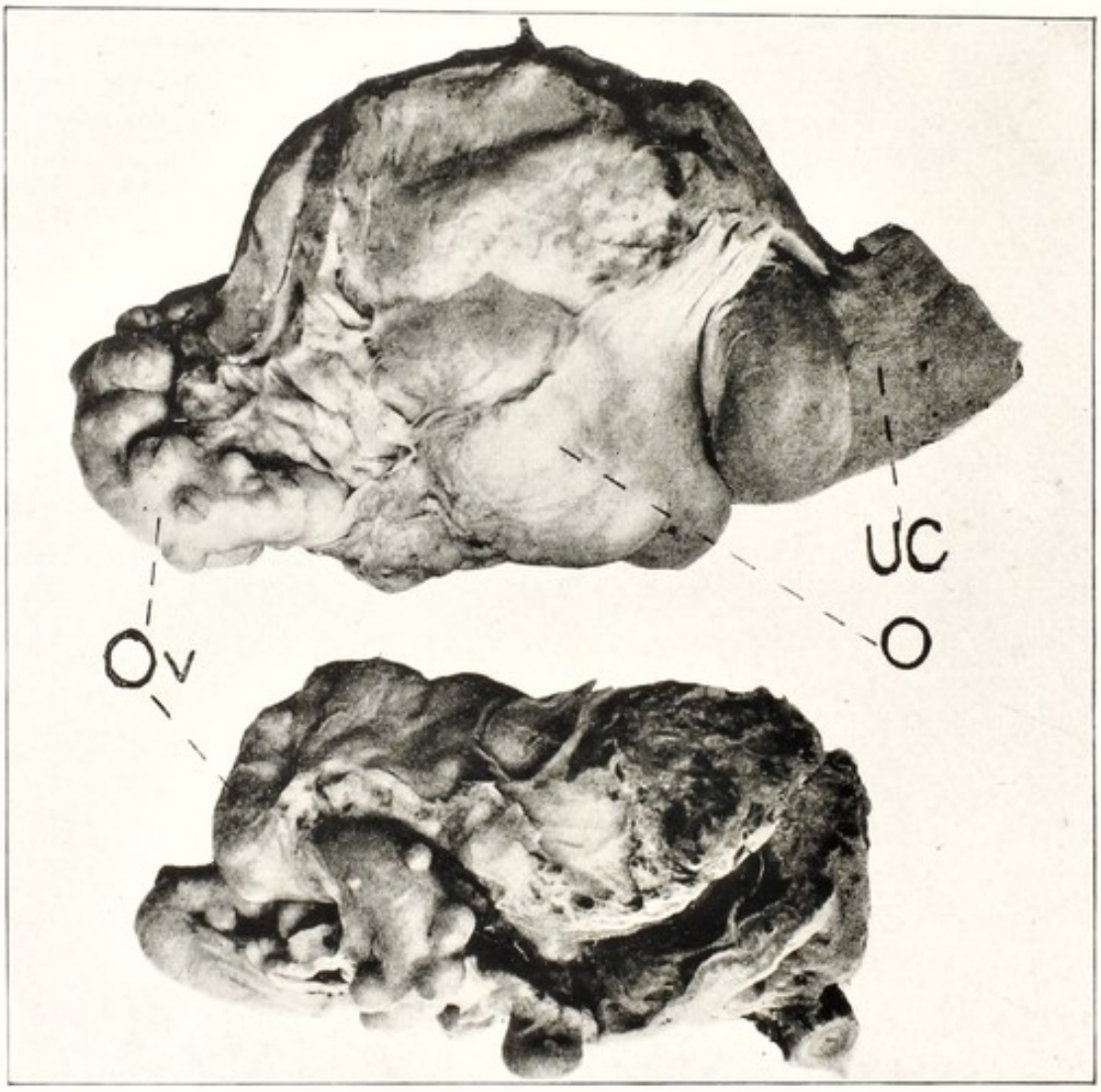


FIG. 79. Advanced Tuberculosis of Ovary and Oviduct.

The oviducts are much enlarged and nodular and the ovaries adherent within the pavilion of the tube. Ov, Oviducts; *O,* ovary; *UC,* uterine cornu.

solitary, but is accompanied almost always with tubal and uterine disease (Fig. 78), the presence of which aids in differentiation. Some veterinary writers have claimed that ovarian tuberculosis causes nymphomania. This I have not observed and have seen no tuberculous ovaries in the abattoir suggestive of nymphomania.

Tubal tuberculosis is not rare. The oviducts are enlarged, firm, and

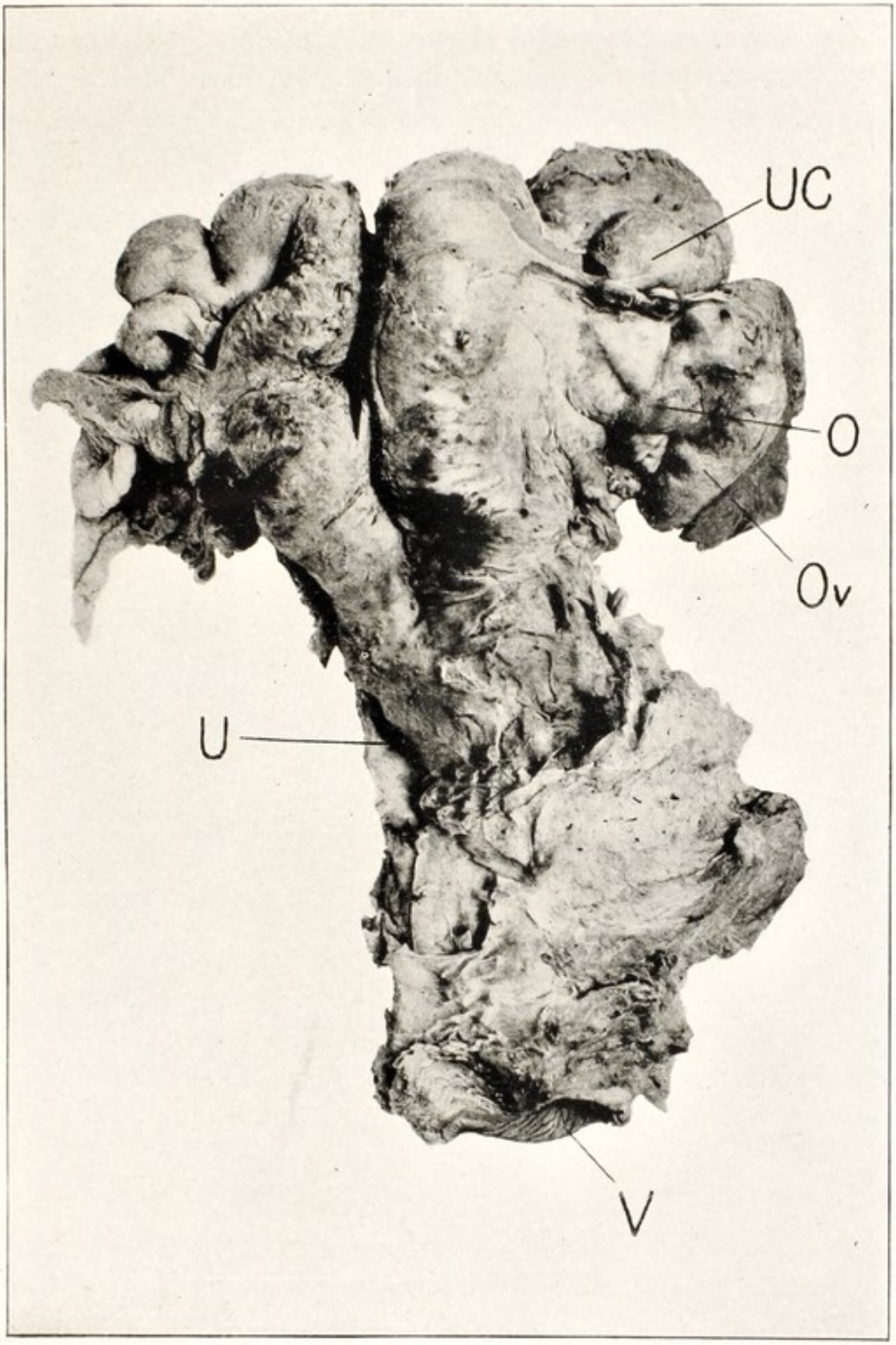


FIG. 80. Generalized Tuberculosis of the Reproductive System, Viewed Dorsally. *V*, Vagina; *U*, base of uterine cornu; *Ov*, oviduct; *O*, ovary.

definitely nodular upon palpation. The tubes may be ¼ to ½ inch in diameter. This is well shown in Figs. 79-82. Pyosalpinx and hydrosalpinx may sometimes lead to confusion in diagnosis. They are of even contour, however, as shown in Figs. 150, 153, 155, 156.

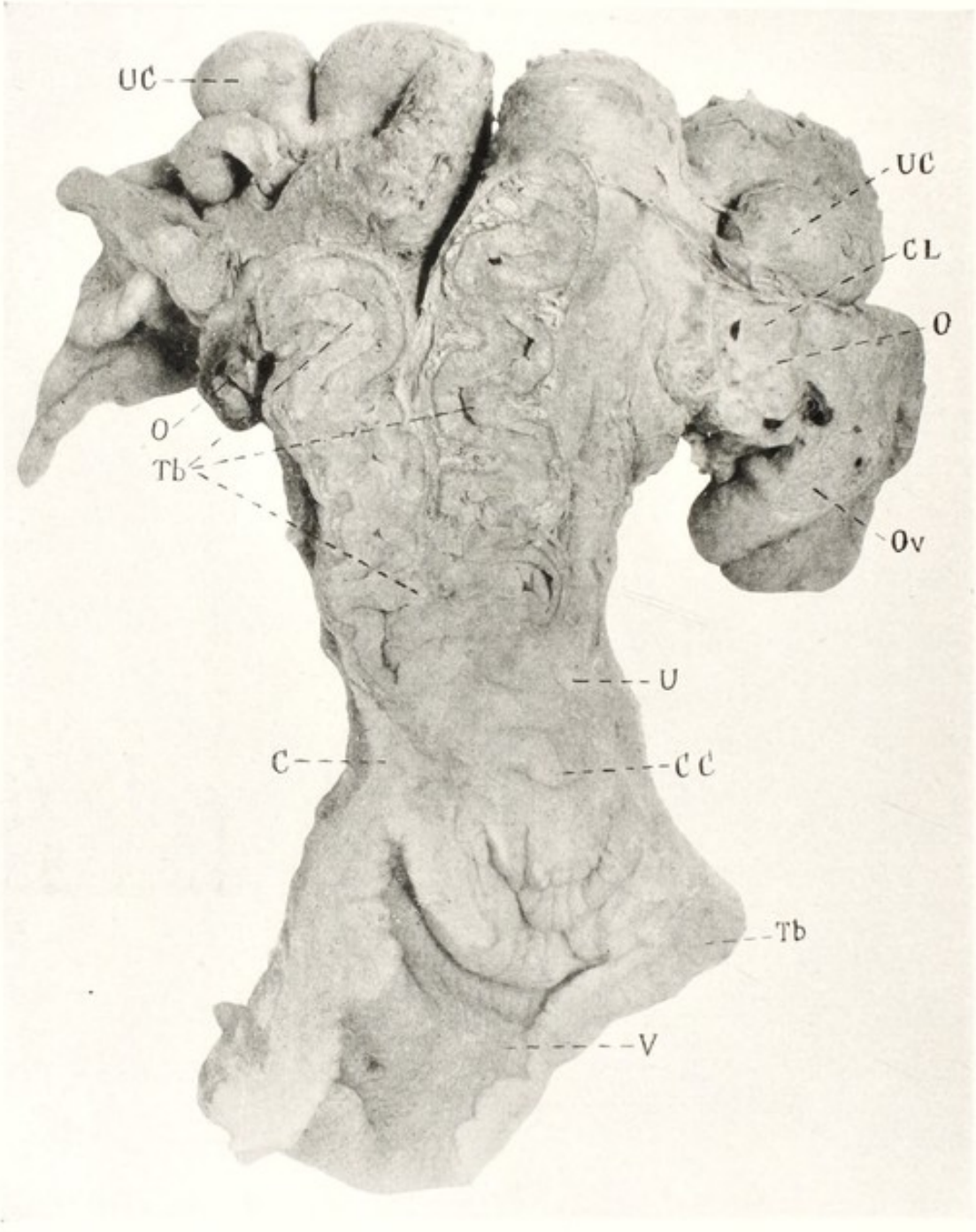


FIG. 81. Horizontal Section of Fig. 80.

V, Vagina; *Tb* (below), tubercles in right vaginal wall; *C*, cervical wall; *CC*, cervical canal; *U*, base of uterine cornua; *Tb* (above), tubercles in cornua; *O*, *O*, ovaries; *CL*, corpus luteum; *Ov*, tuberculous oviduct greatly enlarged and pushed out of normal position; *UC*, uterine cornua.

Uterine tuberculosis is the most dangerous type of tuberculous genital disease, because of the voluminous discharges and the coital peril to the herd sire. In some advanced cases the ovaries, oviducts,

and cornua constitute a large adherent mass, as in Figs. 80-81. The tubercles are usually readily palpable. Adhesions occur also after severe metritis, with which there is usually a history of prior disease. Perhaps most confusing is the rare occurrence of genital actinomycosis as shown in Fig. 90. Figs. 80-81 are from a cow entered in the clinic for sterility. Slight uterine catarrh was present, but no palpable



FIG. 82. Advanced Tuberculosis of the Reproductive System of a Cow Negative to Tuberculin Test.

Sectioned horizontally. 1, Cervix; 2, uterine cornua; 3, 3, 3, 3, oviducts; 4, 4, ovaries.

lesions of genital tuberculosis. After apparent recovery she was bred and soon broke down so that genital tuberculosis was evident. With limited infection of the genitalia, coitus possibly aggravated the disease. More probably she became pregnant, the ovum perished from tuberculosis and the decomposing embryo set the disease aflame. At least it is not rarely observed that an apparently healthy cow, carrying tuberculous infection, aborts, or, going to term, has retained fetal membranes and goes down with acute tuberculosis.

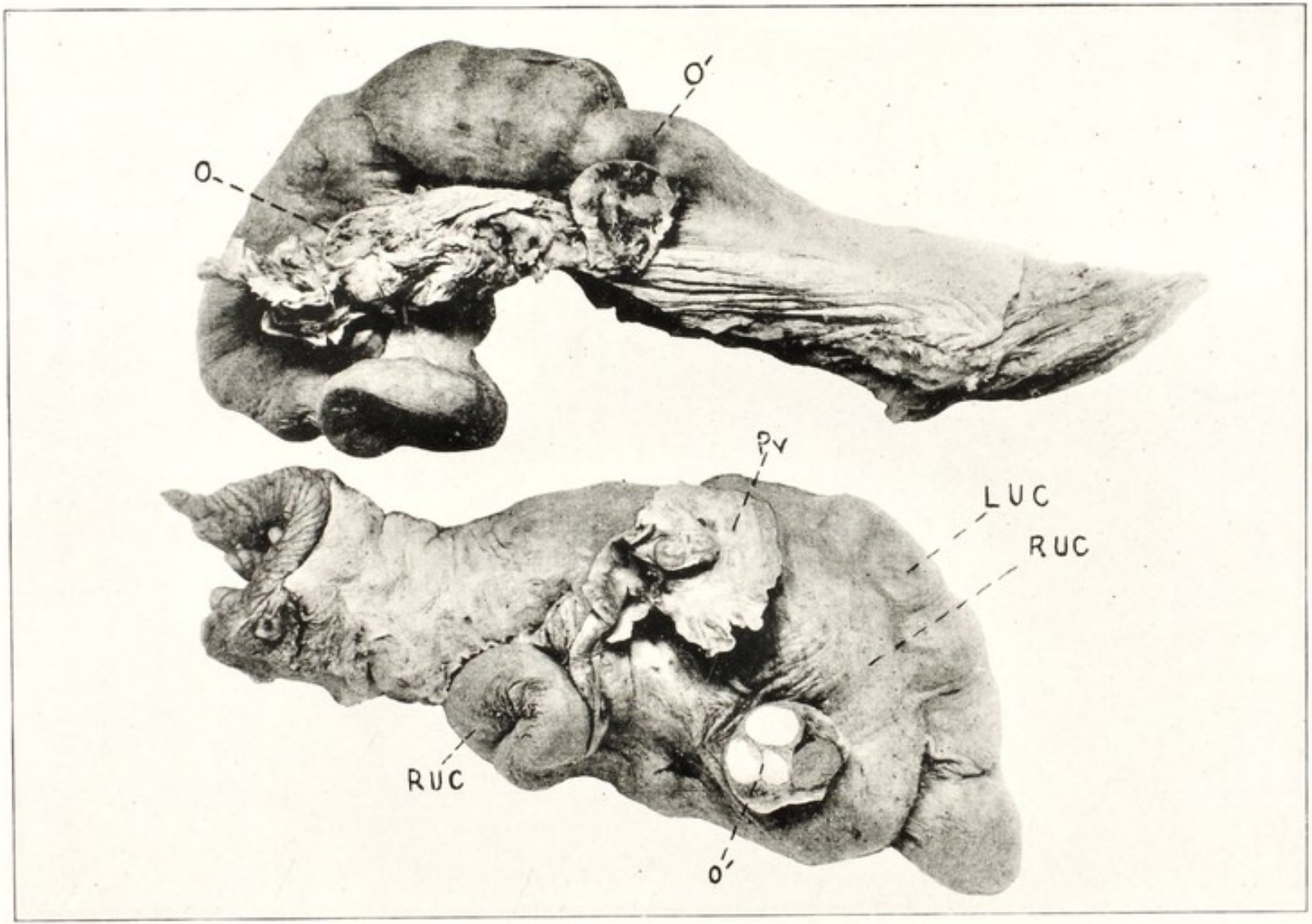


FIG. 83. Advanced Genital Tuberculosis without Peritoneal Adhesions.

O, Ovary; *O'*, *O'*, excised portions of ovaries; *Pv*, pavilion of right oviduct spread over the attached half of the ovary; *LUC*, left uterine cornu; *RUC*, right uterine cornu.

According to Plum[157] when avian tuberculosis invades the bovine genital tract, the organs do not become adherent, the lesions, at least for a time, being limited to many small abscesses, the size of a pea, such as are shown in Fig. 88. It is not readily diagnosed by palpation and the animal does not react to mammalian tuberculin. She will generally react to avian tuberculin, but such reaction also occurs in



FIG. 84. Sagittal Section of Fig. 83.
CC, Cervical canal; 1, pus in base of cornu; 2, pus at cornual apex.

the presence of Johne's disease. Some uteri diseased from mammalian tuberculosis also fail to become adherent, as indicated in Figs. 83-85, in which, however, the tubercular masses may be definitely recognized by palpation.

Discharges from uterine tuberculosis are usually non-fetid, while discharges due to maceration of a fetus and from some types of pyogenic infection are usually fetid. Tuberculous uterine disease is almost always symmetrical, while in the case of a very small macerating fetus one cornu is much larger than the other.

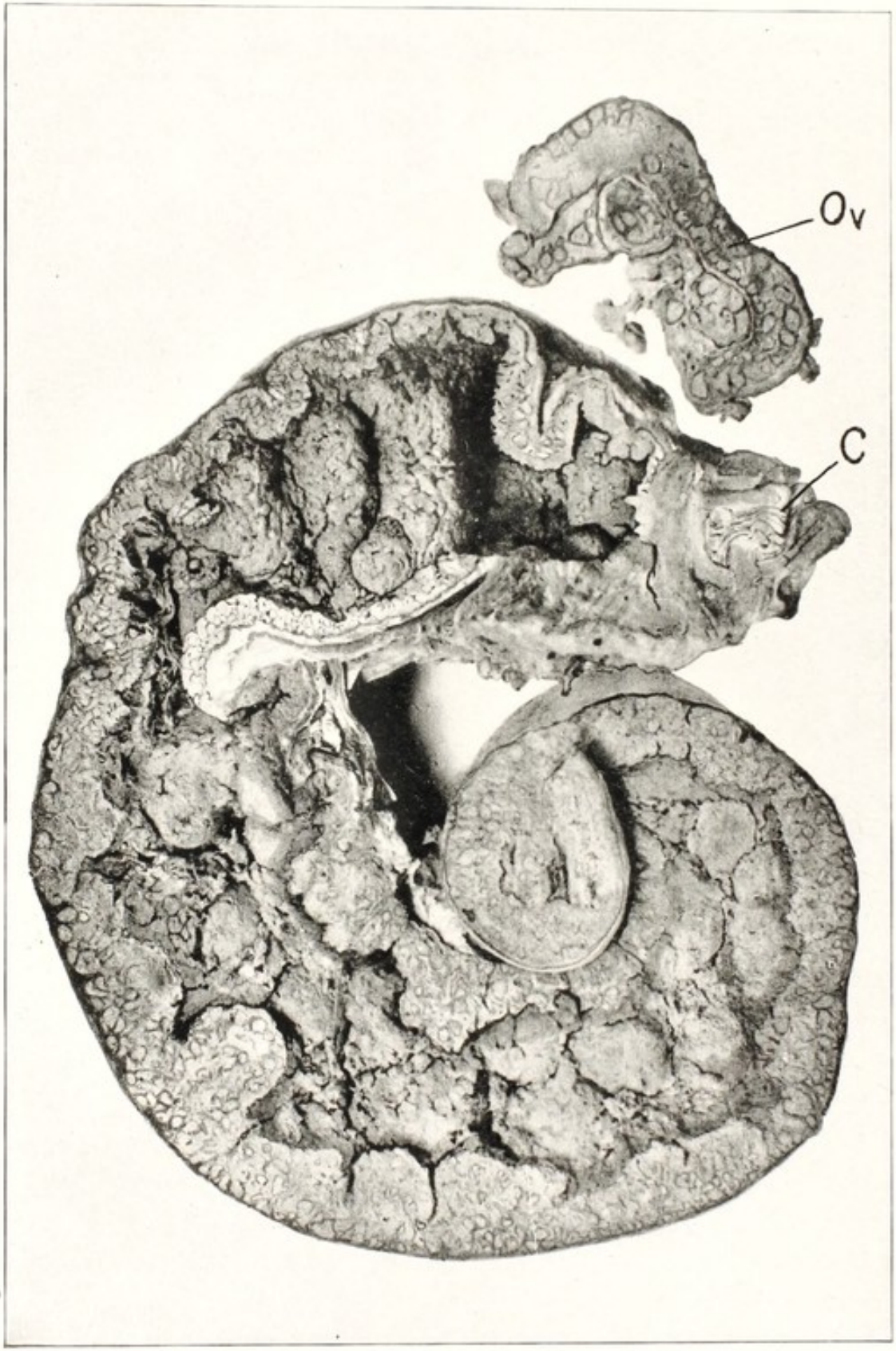


FIG. 85. Advanced Tuberculosis with Profuse Pyometra.
Ov, Oviduct, greatly enlarged; *C*, cervical canal.

When the uterus and oviducts are extensively adherent the breeding life of the cow is almost always closed regardless of the species of infection causing or accompanying the disease, and accordingly she should be discarded. It is important, however, that a definite diagnosis be made, post mortem if necessary, in order that spread of the disease may be controlled.

Tuberculosis of the cervix is not common and when present is usu-

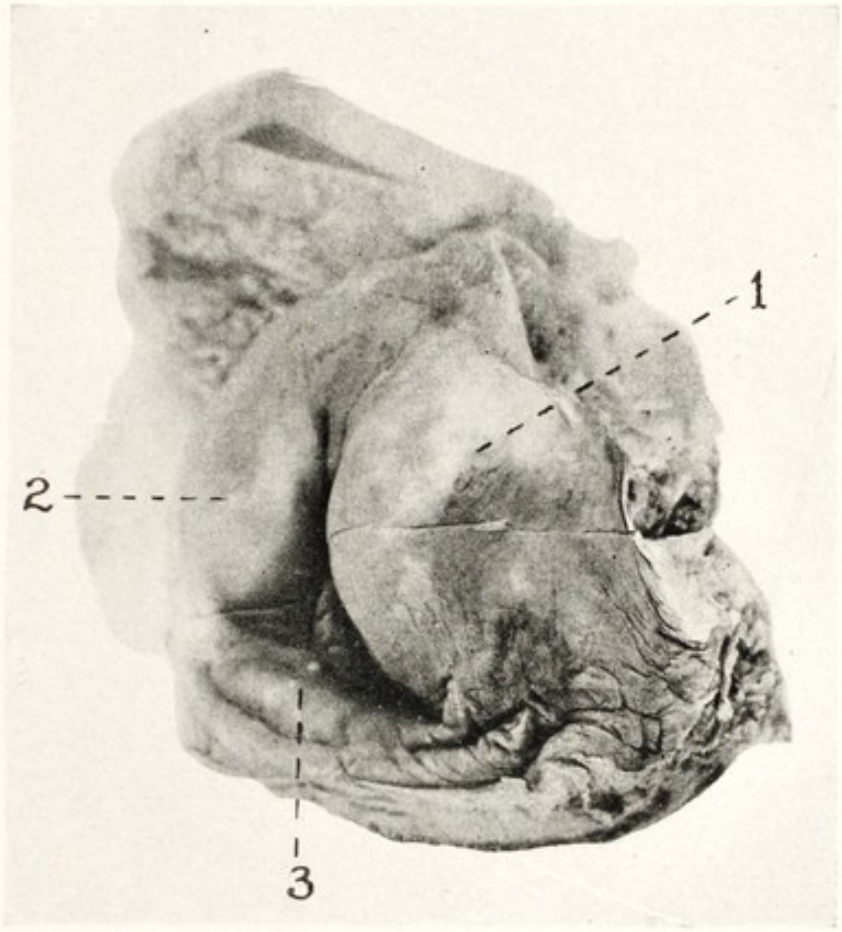


FIG. 86. Tuberculosis of the Cervix.

1, Greatly enlarged cervical lip; 2, a second, lesser tuberculous enlargement; 3, healthy area in first cervical fold.

ally accompanied by uterine and tubal disease. Its general appearance is illustrated in Figs. 86-87.

The vaginal walls are at times the seat of tuberculous lesions as indicated in Fig. 81. I have observed two cases of vulvar tuberculosis. In one I opened what I assumed was an ordinary cyst of Bartholin's gland. At the next examination tuberculosis was evident, with involvement of neighboring lymph glands. Usually the diagnosis is probably easy. Vulvar tuberculosis is evidently highly dangerous for the bull.

The importance of genital tuberculosis should command the ear-

nest attention of the clinician, especially in those areas where tuberculosis has not yet been brought under satisfactory control. It sometimes occurs that in advanced tuberculosis the tuberculin test fails and the clinical recognition of the disease becomes of very great importance.

The clinical diagnosis of genital tuberculosis of the internal portions of the reproductive system of swine is apparently impossible in

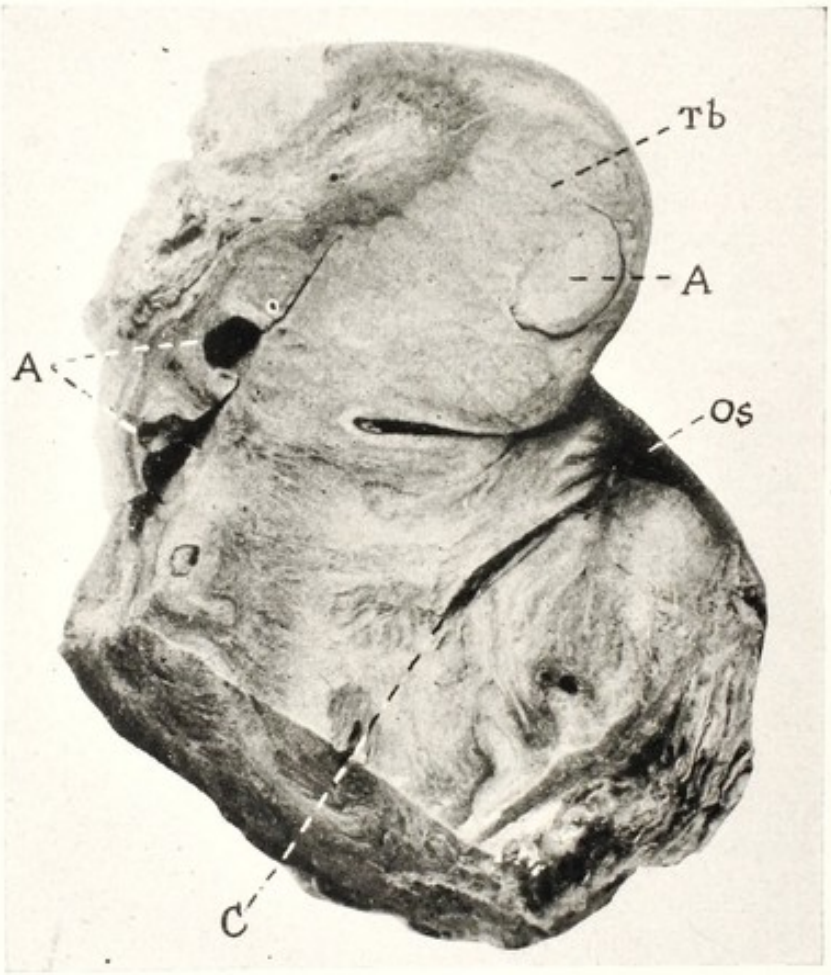


FIG. 87. Sagittal Section of Fig. 86.
Os, Os uteri externum; *C*, cervical canal; *Tb*, tubercles; *A*, abscess

the present state of knowledge. Sometimes the testicles are the seat of extensive lesions, as shown in Fig. 69. With such enlargement of the testicle, tuberculosis is to be suspected. Swine tuberculosis is said to be largely of the avian type. When the testicle of a breeding male has been grossly enlarged over a considerable period of time, the recovery of its function is virtually hopeless. If but one is involved its removal opens the way to diagnosis.

Genital Actinomycosis

Actinomycosis and some other closely allied infections rarely invade the reproductive system and repress or bar reproduction. So far as known, genital actinomycosis is referable to primary infection rather than secondary invasion from other organs. I have observed actinomycosis in a Galloway bull. The primary lesions occurred in

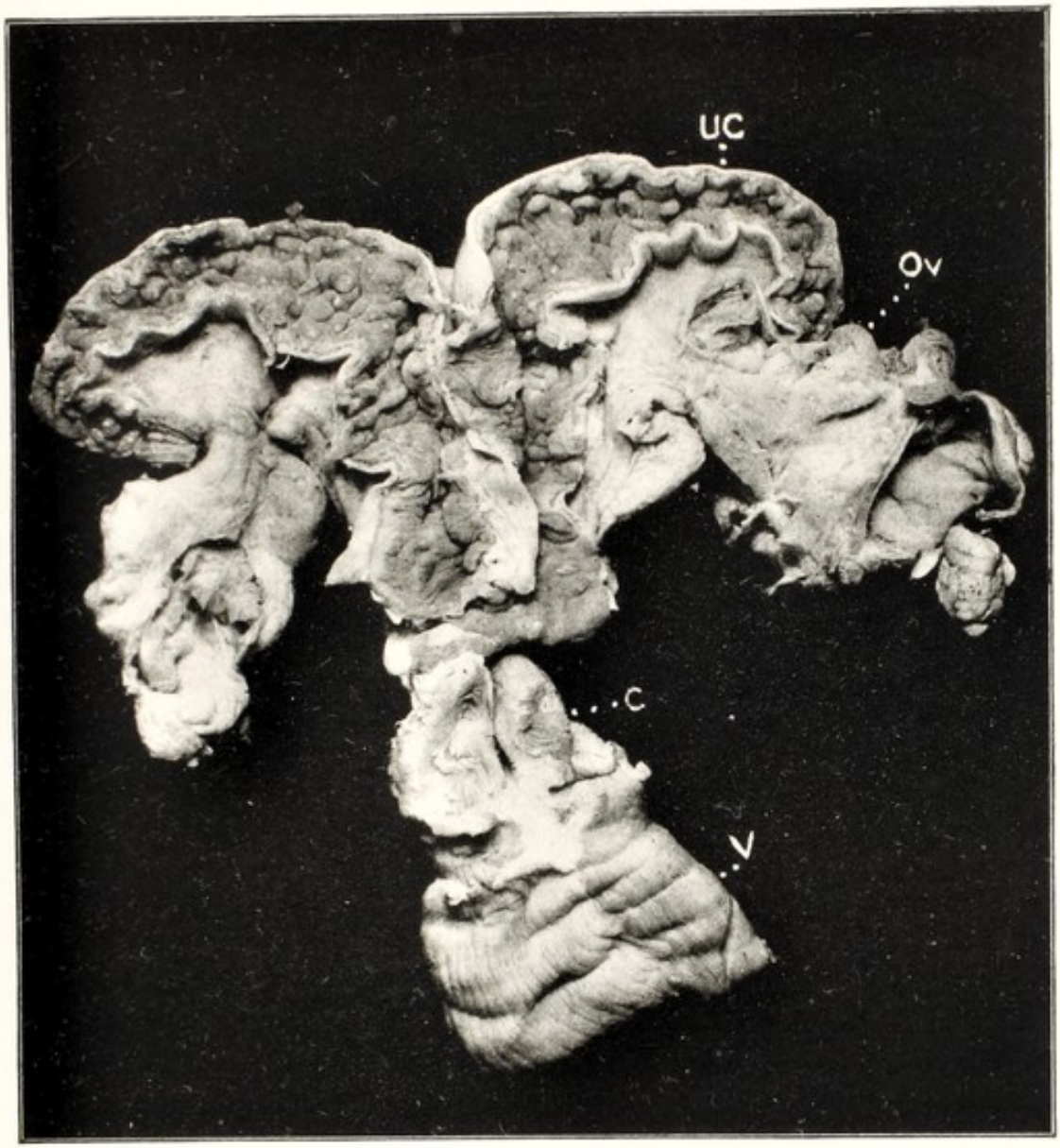


FIG. 88. Avian Tuberculosis of Bovine Genital System.

The uterine cornua have been laid open in order to expose numerous small tubercles in the endometrium. V, Vagina; *C,* cervix; *UC,* uterine cornua; *Ov,* oviduct.

the naked, protruding prepuce. The actinomycotic mass in that organ was about six inches in diameter. Secondarily the sheath became involved as well as the inguinal lymphatics. Actinomycosis of the glans penis also occurs in the bull.

A highly bred cow had long been infertile. I was called in consultation with a colleague. There was no significant history. The internal



FIG. 90. Genital Actinomycosis in Cow.

Above, dorsal view of genital system, irregularly enlarged and having pelvic adhesions: below, sagittal section of urino-genital tract. 1, 1, Vagina; 2, 2, os uteri externum; 3, base of left uterine horn; 4, apex of left horn; 5, actinomycotic base of uterine cornu; 6, actinomycotic abscess in cornu; 7, 8, cross sections of actinomycotic oviducts; 9, actinomycotic abscess in ovary; 10, corpus luteum (var. nigrum); 11, 11, actinomycosis of wall of urinary bladder.

reproductive organs constituted a great adhesive mass in which were numerous spheroidal swellings several inches in diameter. Autopsy revealed multiple sclerotic abscesses. The laboratory diagnosis was actinomycosis. A second case occurred in a cow which had suffered from retained afterbirth at preceding calving. Apparently doing well for a time, she failed to show estrum, and was therefore again examined. As in the preceding case, complete pelvic adhesions were present with large sclerotic tumors. I diagnosed tuberculosis. Autopsy revealed actinomycosis instead.

As indicated in Fig. 90, the genital organs constituted a great sclerotic mass with large indurated abscesses but without the nodular characters ordinarily observed in tuberculosis. Differentiation in the living animal is difficult. The subcutaneous tuberculin test is none too reliable in such cases because in tuberculosis the invasion may be so great that the test fails. Fortunately the differentiation is not important for the individual. The cow is fit only for slaughter. Autopsy reveals the true nature of the disease and permits timely precautions in case of tuberculosis.

The disease is so rare that the diagnosis of genital actinomycosis in either sex or in any species is improbable before the infection has extended beyond the point where the fertility of the animal may be restored or conserved.

Genital Bursatte

The disease of horses known as bursatte or "summer sores" seems to be common in most warm countries and is observed during the summer months in temperate climates. It is observed largely about the coronet and fetlock regions, and at the seat of harness or saddlery abrasions of the skin. It occurs now and then at the inner canthus of the eye. The cause has not been accurately determined. By some, it has been attributed to a form of fungus while others have claimed it is due to a small round worm. The skin becomes swollen and indurated at the seat of the infection, the margins of the diseased area are sharply elevated above the level of the adjacent skin, the surface discharges a thin pus, and embedded in the tissues are accretions called "kunkurs" which are yellowish or grayish in color, commonly about the size of an ordinary pea, which may be somewhat calcified. The disease becomes latent in winter and the ulcerating surface heals, but the induration largely persists and the disease resumes its activity with the advent of warm weather.

The literature fails to indicate clearly the incidence of genital bur-

satte but various writers state that it occurs in the external genital organs of both sexes. I have observed it only in stallions, all draft breeds. However, almost all stallions in my territory were of the draft breeds. With the exception of one case, in which the initial lesion of the genital organs occurred on the scrotum, all cases were of the penis and prepuce.

The first symptom usually observed is the presence about the urethral opening of an angry-looking fungoid growth, one-half to one inch in diameter, which bleeds upon touch. Hemorrhage usually follows copulation. If the diseased growth be critically examined there are usually discovered some yellowish "kunkurs" which can be enucleated with little difficulty.

A second fungoid patch commonly is present upon the ventral (urethral) side of the preputial ring. It occurs at the point of contact between the urethral opening and the free border of the prepuce when the penis is at rest within the prepuce and sheath. Accordingly it is a contact transmission from the suppurating urethra and the prepuce. The source of the urethral infection is not clear, but the fact that the disease is common about the coronet and pastern of the hind feet, suggests that the infection may occur while the stallion is lying down, with his prepuce touching the feet.

The disease is most active during the breeding season. It presents a quite annoying appearance although it does not seem highly dangerous to mares with which the diseased stallion copulates. I have not observed a case of coital transmission.

There is little, if any, cause for confusion in diagnosis. It bears no resemblance to dourine or to genital horse pox. It should not be mistaken for tuberculosis.

The diseased tissues should be generously excised or curetted away, and the parts cauterized with silver nitrate or electric cautery and watched closely, keeping them clean and applying the cautery to any parts which may suggest disease activity. Care should be taken to conserve the urethra in a manner to avoid stricture. This may not always be possible, in which case amputation of the glans may be indicated. The operation has been described under *benign tumors*. After apparent recovery has occurred, close attention should be given the parts during the warm season of the following year, and any recurrence energetically attacked.

SECTION V

INFECTIONS BELIEVED TO HAVE MARKED AFFINITY FOR THE REPRODUCTIVE SYSTEM. CONTAGIOUS ABORTION. CONTAGIOUS STERILITY

Chapter XX

THE SPECIAL INFECTIONS OF THE REPRODUCTIVE SYSTEM IN PERSPECTIVE

IT has already been stated in preceding chapters that numerous infectious diseases of domestic animals, which primarily disturb chiefly other organs or systems than those of reproduction, frequently attack the pregnant female with great virulence. The dual load of maintaining her physical life and the sustenance of the fetus within her uterus, deprives her of important reserves of vigor through which she might otherwise have overcome the invader and recovered. If she successfully resists the invader herself, the fetus within her uterus may succumb and be expelled.

Infections having special affinity for the reproductive system, while most conspicuously causing genital disturbances, such as sterility and abortion, frequently lead also to serious disturbances in other organs. *Bacillus abortus* (Bang), which is usually accorded first place among genital infections, not only invades the uterus of the cow in association with abortion, but encroaches upon diseased articulations of horses and cattle, participates as a pyogenic factor in fistulous withers and poll evil of horses and mules, and in various other maladies. Dourine provides an interesting example of a coital infection which causes marked pathognomonic lesions of the copulatory organs, without generally invading destructively the uterus of the mare; in the very severe type in either sex, the disease attacks destructively the cerebro-spinal system to induce lesions and symptoms suggstive of syphilitic ravages of the human nervous system.

The mammalian reproductive system stands conspicuously apart from all other organs of the body in one highly important respect. The other organs and systems have already assumed their principal functions during fetal life, or are prepared to do so at the moment of birth. All the reproductive organs are established in primitive form during early embryonic life, and some of them, especially the gonads, appear to perform functions vital to intrauterine existence and development. These functions are not clearly understood. It has been stated on page 63 that an ovum in which the essentials for the production of gonads is wanting, can not independently survive to become a fetus, but may do so—as is common in the cow—if it

acquires adequate vascular anastomosis with a gonad-equipped mate, upon which it may live, in a way, as a parasite, to be born a perfectly viable asexual or freemartin.

The genital system is not ready to assume its function of reproduction until the individual is essentially mature and has accumulated ample vigor and nutritive reserves to beget and nourish progeny without peril to its own health or life. From the time of its origin in the embryo until sexual maturity is reached, the genital system is constantly developing. If at any period during this era the development is suspended, because of disease or malnutrition, the defect caused by the omission may be irreparable, and the individual arrive at breeding age with hypoplasia of the testicles, ovaries, vulva or other organ.

Under domestication, ovulation and spermatogenesis, with sexual desire, may become abnormally advanced. Unless carefully segregated the immature female may become pregnant long before having reached the necessary maturity to produce young safely. A large ratio of highly bred dairy heifers begin ovulating at five or six months, and instances are recorded where conception has occured at ten or twelve weeks. Such conceptions largely end in abortion; if a calf is born, the heifer is ruined and the offspring worthless.

The ovum is unavoidably exposed to any infection resident within, or entering the uterus at any time during pregnancy. If the ovum is defective when it leaves the ovary, and later becomes fertilized, the imperfection persists permanently. The new individual is wanting in essentials of structure and function, and constitutes a highly attractive magnet for invaders, some of which are not necessarily dangerous for healthy ova.

Infection is not necessary for the death of the intrauterine young. When twin ova are discharged, as is well proven in the cow, one of the ova is frequently devoid of the basic elements required for the formation of the heart (acardia); of well nigh, or all distinct organs (amorphus), Figs. 23-25; or of gonads (asexuals or freemartins). These ova, so far as is known, universally perish early, except the defective attains ample vascular anastomosis with a structurally perfect mate. Such ova die from intrinsic structural and functional defects. No infection is required. No accident is called for. It is simply a fertilized ovum, non-viable except as a parasite upon a complete mate.

It is sometimes stated that Bang's bacillus is not usually present in early bovine abortion. Various inferences may be drawn therefrom, one of which is that early abortions are not usually due to

infection. The statements are not complete, however, and it is not always definitely declared that no bacteria or protozoa were present, and ordinarily no attempt is made to show that a virus capable of causing fetal death was not acting. If the inference be accepted that infection is not present in an early abortion, it at once becomes difficult or imposible to determine why other fetuses which survive for a time, although too defective in structure or vigor to develop into viable young, should not at a later date become invaded by pathogenic organisms which would intensify the intrinsic disturbances of function and structure.

Hart and Traum[84] report an extensive prevalence of abortion in a Bang-negative herd, in which neither *Bacillus abortus* nor other invader was found to which they could attribute the losses, although in these cases the fetuses were moderately mature. So in numerous other reports no invaders were recognized as responsible for the highly destructive prevalence of abortion in the herd.

The prevailing conception of retained afterbirth, especially in the dairy cow, tends to contribute to the confusion regarding the significance of bacterial invasion in abortion. It is commonly stated that retained afterbirth frequently *follows* dystocia and abortion. The date of origin of the disease is thus inferentially placed *after* the expulsion or extraction of the fetus. If instead it be stated that the placental disease, destined ultimately to become known as retained afterbirth, becomes established long before the termination of pregnancy and constitutes the basic cause of a large ratio of cases of abortion and dystocia, a clearer conception of all three phenomena becomes possible. The existence of the placental disease, long before abortion or dystocia, imperils the health and life of the fetus in at least three ways: it deprives the fetus of its physiological quality and quantity of nutritive materials; toxins may be generated within the diseased tissues and reach the fetus via the bloodstream; and bacteria, protozoa or viruses may multiply within the diseased tissues and be carried to the fetus through the umbilical vein. Either of the first two may apparently cause dystocia or fetal death in the absence of the third within the fetus. Hart and Traum, as mentioned above, apparently confined their observations to the aborted fetuses. An authentic bacteriological study of the afterbirth would usually call for the slaughter of the cow during parturition or abortion, or immediately after. The afterbirth, which constitutes the outer defense line of the ovum, may not be seriously injured without repercussions on the part of the fetus. It has been clearly shown by Williams, Udall, Frost *et al*[243] that this outer bulwark of the ovum is rarely

wholly without macroscopic proof of injury from some species of invader, just as the epithelial covering of the postnatal animal is constantly being exposed, and subjected to a great variety of injury, from trivial to grave. In the contribution cited, it is amply shown that the lesions of the chorion are parallel to those of the fetus: if the chorionic lesions be great, the fetus dies; if the lesions be moderate, dystocia may occur and a living fetus may be expelled or extracted. In either case the afterbirth is retained and the calf, if it lives, suffers from diarrhea, pneumonia or other disease, and if it survives, is of subnormal value. This view has not yet been extensively accepted by veterinarians and animal husbandmen. Some, such as Pomayer,[158] have conducted important studies upon the retained placenta after the occurrence of abortion or parturition, when postpartum or fetal postmortem changes may well have occurred, and have considered chiefly the possible effect of these changes upon the future sexual life of the mother. The basic disease of the placenta, at the date of its origin, and its behavior during pregnancy, with its threat to the health and life of the fetus, is ignored. Until adequate study in this field shall have been conducted, our conception of the abortion problem must remain regrettably incomplete.

After death the ovum may be invaded by phagocytes, and the fertilized cell thus disintegrated. So far as known, the above mentioned defective ova, prior to death, possess little or none of the power of resistance attributable to a healthy cell. There is no reason to assume that an ovum destined to develop into an amorphus, except it accomplish anastomosis with a mate, has any higher power of resistance to invaders than do dead ova. Phagocytes may invade and decompose them. Thus, through the entire scale of deficiency, the power of resistance runs parallel to the degree of perfection of function and structure of the fertilized ovum. Mall[133] found, in early abortions in woman, that of the fetuses aborted from the first month only a fifth were normal in structure, and of those from the second month, but a half. Such embryos required no infection to cause death, and they had no power to defend themselves against phagocytes or facultative invaders. When a dairy cow has recently suffered from nymphomania or other serious ovarian disease, has suffered severely from metritis, or has aborted, physiological ovulation is distinctly improbable, and any early pregnancies which occur are inevitably insecure.

The laws of infection are parallel for intrauterine and extrauterine animals: in both groups alike the subnormal in structure or

function largely become invaded and perish. There appears to have evolved the phantasy that intra- as compared with extrauterine animals are especially fortified against bacterial invasion, except for those invaders having a special affinity for the unborn. Seemingly it is forgotten that, at the moment when life begins, death shadows it. The mortality in domestic animals is by far greatest among the spermatozoa and unfertilized ova. Among the billions of spermatozoa ejaculated in a single coitus, only one survives to fertilize the single ovum of a uniparous female, while the many billions of living cells perish.

Schmaltz[178] cites Käppeli as having counted in heifers of three months an average of 75,000 primary ova per ovary, or 150,000 per calf. These largely perish from atresia until, in young cows, the average for each ovary is 21,000, a loss of 54,000, or 72%. In the herd studied by Wing,[271] 3.5 ova per cow were recorded as having been fertilized and developed to the stage where pregnancy was recognized, and of these 2.5 viable valves per cow were born. Thus one viable calf was produced for each 60,000 ova which had inferentially existed in the ovaries of the cows. The others died or were discarded with the cows.

The next greatest ratio of death occurs among the fertilized ova, within the uterus. So far as known, the ratio of intrauterine death decreases as pregnancy advances. The very early deaths, which pass unseen and are designated as sterility, take first rank; those which have attained such size that their death and expulsion are observed, known as abortion, come next, followed by those classed as "born dead," "prematurely born," "born in a non-viable state," and those perishing from diseases of the new-born.

The life of the fertilized ovum may be endangered by:

(1) being the progeny of the union of a spermatozoon and of an ovum, one of which is defective;

(2) infections or lesions existing in the uterus or oviduct at the date of conception, Figs. 173 and 175, which compromise the health and life of the ovum, rendering the production of healthy young virtually impossible;

(3) infection which may be introduced with the semen at coitus, as Bang[8] relates, citing Poulsen, who reported that seven cows from a herd were bred to a neighboring bull and all aborted, while the others cows, bred to the herd bull, all calved. Coital invasion is considered the general cause of the protozoan trichomonad disease. Abortion referable to an infected sire is common in horses and cattle;

(4) bacterial destruction of the uterine seal of the cow, Fig. 47

(which constitutes the most elaborate physiological occlusion of the cervical canal known in mammals during pregnancy) resulting in the invasion of the uterine cavity and the contained young;

(5) infecting organisms or their toxic products which may invade the uterine cavity and its contained young, via the blood stream, at any period of gestation. This is amply illustrated, among innumerable instances, by epizootic cellulitis (mare), foot-and-mouth disease (cow), sheep pox (ewe). The same is equally true of woman. Taussig[193] states: "Many acute infectious diseases have a deleterious effect upon pregnancy, and may indirectly be a factor in its interruption through the associated toxemia and hyperpyrexia. Typhoid fever, cholera, scarlet fever, smallpox, erysipelas, encephalitis lethargica, and malaria produce abortion in about one-half the pregnancies associated with these diseases."

The very early death of a fertilized ovum can not usually be distinguished from that of the unfertilized cell. The male may fail to create or ejaculate spermatozoa capable of fertilization and the females served by him remain non-pregnant. In such case the females served by him will ordinarily again be in estrum at the normal period. A study of the semen and stained spermatozoa generally leads to an authentic diagnosis. When a sire produces sperms like those in Fig. 28, probably a large ratio of the mares he serves conceive, but the ova almost all perish quickly.

Ova fertilized by such spermatozoa inferentially undergo bacterial decomposition, with injury to the endometrium, or other damages which interrupt the normal sexual cycle, the intervals between the cycles are prolonged, and the female becomes sterile. In cows such as those the uteri of which are illustrated in Figures 173 and 175, the evidence is conclusive that conception had repeatedly occurred, and the ova perished and decomposed, adding new injury to the endometrium, and rendering the physiological completion of pregnancy essentially impossible. The ova were starved, infection was certainly present in the uterine cavity, and death and decomposition of the ovum assured.

The destiny of twin pregnancy in the mare and cow serves to illustrate the great vulnerability of the abnormal ovum to bacterial invasion and destruction. Twin ovulation in both mare and cow is largely pathological, and the fertilized ovum subnormal in vigor or structure. More than 90% of equine twins are aborted. Often one of the twins undergoes putrid decomposition before expulsion. It is commonly termed "twin abortion" despite the putrid decomposition. Twin pregnancy in the cow commonly follows nymphomania or

other ovarian disease, metritis, hurried rebreeding after calving or aborting, or other form of genital disorder. The abortion ratio is far higher than in single pregnancy, the duration of pregnancy in those cases escaping abortion is much abbreviated, the calves are small and weak, many of them prove non-viable, they are highly subject to diarrhea, and the cows largely suffer from metritis and retained placenta.

The frequency of abortion indicates that the ovum, after it has attained such volume that its expulsion is readily observed, is highly vulnerable to pathogenic invaders. Not all diseased fetuses are aborted. Some newborn are congenitally diseased. Frequently they are non-viable and die within a few hours or days after birth. Others may linger for months, like the calf illustrated in Figures 115-117. Or they may prove viable and finally arrive at breeding age, as indicated in Table I, when they break down in their first breeding season, under such conditions as to justify the conclusion that the disaster may be in part attributed to congenital infection.

Although most diseased fetuses are aborted, "born dead," born prematurely, or born diseased at term, some are carried far beyond the physiological term in a living state. Thus, in the mare, the diseased fetus is not rarely carried to 365 days and beyond, to be born perilously diseased, usually unable to stand, and perishing within a few hours. In spite of the prolonged intrauterine life, the foal is usually smaller at birth than healthy young born at the normal period. In sharp contrast, the cow may continue pregnant to 365 or 400 days and the fetus continue to grow until tardy labor sets in, at which time it is so enormous that it can not be expelled, or extracted entire through the birth canal. The fetal cadaver sometimes shows the existence of diarrhea, or other disease, indicating the invasion by pathogenic organisms.

The artifically grown dairy calf, commonly suffering from malnutrition, as indicated by the adhesive fecal discharges which stick to the tail and buttocks, undergoes erratic sexual development. Its physical life demands a large ratio of its nutritive supply, the development of the reproductive system is repressed, and the animal arrives at breeding age in a state of genital hypoplasia (pages 99 and 391). Physiological reproduction by the individual demands the proper development of its genital system from the moment of the fertilization of the ovum until sexual maturity has been reached: during both its intra- and extrauterine life its environment needs be favorable and its nutritive supply adequate. When cattle, goats or sheep are intensely used for dairying, the young are regularly

removed from the mother and fed by hand. The evil effects of errors in feeding and handling of the newborn may be halted when the young have reached sufficient development to assimilate advantageously the usual foods of adults. This change is not retroactive and does not overcome the effects of the repression of the sexual evolution during the milk-feeding period. During the interval between the normal age at which the milk-feeding period ceases, and the arrival at breeding age, the dairy calf usually enjoys good health and grows well. Upon its arrival at the age when most young dairy animals are placed in breeding, other causes of genital diseases arise.

Wild animals largely breed in pairs. Among some species, powerful males assemble harems of females of considerable numbers, as in the fur seal. The vigorous, powerful adults drive the young, and the aged and decrepit males away, so that only the most vigorous among them act as sires. Apparently this process serves to promote the vigor of the species. Under domestication, the immature young male may be assigned a harem far beyond his powers to serve safely. Young bulls are not infrequently wrecked before having reached the degree of maturity for ideal reproduction. This is especially true of young dairy bulls in the process of making "proven sires." The economics of the production of proven sires invites the excessive use of the immature bull upon an extravagant number of cows, in order that he may have, at the earliest possible date, the required number of daughters to fulfill the rules for the production of milk and butterfat, thus enabling the breeder to sell the bull at a high price and leave the buyer to take the risk of securing a ruined sire. Thus Fincher, Olafson and Ferguson,[58] in their case No. 6, state that the immature bull, during his first breeding season, begat 18 or more heifers to magnify his reputation as a valuable sire. In order to accomplish this he must have been caused to serve at least 40, probably 50 or more cows, a heavy sexual load for the average mature bull. During his next three years he begat approximately 20 calves, or about one sixth the ratio per unit of time accredited to him during his first breeding season. He then broke down completely. Henderson[86] reports a similar instance, with the variation that the highly expensive bull, in carrying the excessive sexual load in order to become a "proven sire" had developed the significantly coital infection of trichomonad disease. This led to the slaughter of the high-priced "proven sire" along with another bull, and played havoc with a goodly number of highly bred cows. Both herds were officially "Bang-free." The herd producing the infective bull was one of the most famous in the United States for the creation of "proven

sires" and the distribution of the trichomonad disease of a virulent type in high-class purebred herds. Some would say that the sexual overload was not the cause of the malady, but so far as clinically determinable, those genital infections significantly transmitted by coitus are greatly intensified, and rendered more virulent thereby. The hybrid mule, and the castrated and spayed animals of other species, without sexual burden, are virtually free from diseases of the genital organs.

Bacteria, protozoa and viruses which invade animal systems, organs or tissues are roughly divisible into three groups: (1) pathogens, such as those of glanders and hog cholera, which usually induce characteristic symptoms and lesions designated as disease; (2) facultative invaders, like *Hemorrhagic septicaemia,* the bacterium of which is said by some to be quasi-ubiquitous in cattle but does not usually cause disease except the animals have been subjected to exhausting transportation (shipping fever) or other hardships which have depressed their normal powers of resistance. In such transported cattle the invader may acquire exalted virulence, enabling it to cause disease for a time in non-transported cattle of supposedly normal resistance; (3) saprophytic habitants, commonly present upon the mucous membranes of hollow organs, especially near their openings upon the exterior. They are said to subsist upon waste tissues and excretions and to be devoid of power to establish recognizable disease.

The lines of demarcation between these groups are not always perfectly clear to the clinician. Those invaders regarded as especially pathogenic may rarely assume a harmless behavior when the exposed animals are exceptionally vigorous due to favorable environment or other factor. Neither is it positively known that invaders classed as phagocytes may not become able to cause disease when the host is placed under extremely unfavorable surroundings, and the tissues with which the organisms are in contact have largely or wholly lost their powers of resistance.

Gould (Medical Dictionary) defines *disease* as "A disturbance of function or structure of any organ or part of the body." Thus in foot-and-mouth disease, the body functions are disturbed, as indicated by fever and other signs, and notable changes in structure occur in the form of vesicles and ulcers about the feet and mouth. Despite its high transmissibility, some exposed individuals resist the plague. The presence of the virus within the body of such resistant animals does not constitute disease, as defined above. But such an individual may be competent to transmit the disease to other ani-

mals. There are individuals which resist the infection of most, if not all, great animal plagues.

Environment sometimes significantly modifies the virulence of an invader for the animals occupying a given area. Prior to this century, glanders was one of the most destructive scourges of army horses; was extremely virulent for hard worked horses in great cities, such as those drawing street cars; and constituted an important economic problem among farm horses. Many of the animals in these environs, when infected, revealed disturbances of function and structure which permitted the clinician to make an authentic diagnosis, while other individuals resisted the infection sufficiently that they could not be condemned, and barred the pathway to effective control. During that era I had occasion to study glanders in the Rocky Mountains. Many horses were grown at very high altitudes, free upon the open range throughout the year. Some individuals brought down into the valleys, closely stabled and worked hard, not infrequently suffered from severe and mortal glanders. If the diseased animals were opportunely returned to the open range, the symptoms promptly abated, and aside from some very mild, though characteristic lesions, the animals enjoyed good health. Most significant was the fact that among hundreds of horses in intimate contact with these definitely glandered individuals, year in and year out, I could find no trace whatever of the disease. Had the mallein test been available and applied, some of these apparently ideally healthy horses would probably have reacted. In such case some would have said they were diseased; others would have called them healthy. There would have been no conflict of opinion regarding the facts: there would have been a mere difference of definition. Had the reactors to the mallein test remained on the open range, it would have been highly improbable that they would have become diseased, as signified by any disturbance of function or structure; had they been taken from the open range and used in the army during a trying campaign, they would have developed typical clinical glanders and have imperilled the lives of the horses and men with which they came in contact.

Some apparently hold that an individual is *diseased* if there are present, in its organs or tissues, bacteria, protozoa, or viruses of a species identified as responsible for important disturbances of function or structure in other animals of like species. On the other hand, the advocates of the BCG method for controlling bovine tuberculosis contend that the administration of the tubercle bacilli prevents the disease. In fact a large ratio of vaccinations against great plagues consists of the introduction, into the individual which it is desired

to protect, of cultures of the invader which naturally causes the scourge. Sometimes the invader has been artificially depressed or modified in virulence (rabies); sometimes an alleged variety of the invader has been discovered (strain 19 of Bang's bacillus) which prevents instead of causing disease.

Such examples serve to create the view that the presence within an organ of an invader admittedly pathogenic, under environment favorable to its multiplication, does not uniformly constitute disease. The infections having a special affinity for the reproductive system excel all other groups in their facultative capacity for behaving as phagocytes or pathogens according to environment.

Reference has already been made to the increase of virulence of genital infections caused by depressing factors in the environment, amongst which sexual overloading plays a conspicuous role. Innumerable examples of this might be cited. Thus a stallion, mildly affected with dourine, page 341, transmits the malady to only a small ratio of the mares which he serves, while the others may become pregnant and remain healthy. If he be continued in service, the disease advances in virulence under the strain of repeated coitus, and he transmits the malady to an increasing proportion of his mares, until he become unable to copulate. On the other hand, the mare usually copulates but once or twice during the breeding season, and usually suffers less seriously from the malady. The infected mare may grow worse for a time, and may in some cases succumb finally to the cerebro-spinal complications. If conception occurs in infected mares, the fertilized ovum apparently perishes early and disappears unseen. In the extensive outbreak in Illinois, I saw no case of pregnancy in an infected mare. The mares were on excellent pasture during the breeding season, and if at all worked were used only for gentle farm service. Most of the infected mares soon improved and continued in apparently good health, except that they carried in the vulva and surrounding parts authentic lesions of the disease for a year or more. Under breeding quarantine, they performed satisfactory agricultural work for the normal duration of life.

The recorded histories of outbreaks of bovine trichomonad disease are too inadequate for authentic analysis. The basic contribution by Abelein[1] indicates that the one bull of which he submits a considerable breeding history did not infect his cows so long as he was limited to one coitus in several days; when caused to serve two cows in one day, or one cow daily for several days, infection occurred; when the coital load was lightened, his infectiousness decreased.

Finally he became so intensely infected that he transmitted his malady to a large ratio of the cows with which he was mated.

The granular venereal disease (infectious vaginitis), page 365, is essentially ubiquitous in ruminants and swine, if the lesions shown in Figure 100 be accepted as proof. It has not been described as causing economically important disease except in dairy cattle. The dairy heifer, prior to her first coitus, usually shows only the mild type illustrated in Figure 100; within two or three days after her first coitus the infection explodes, as in Figure 101. Under the ordinary method of feeding and handling of newborn dairy calves, they clearly show, within a few days after birth, vulvar and preputial catarrh, with intense black staining of the vulvar and preputial tufts of hairs, as illustrated in Figures 31 and 33. As indicated in Figures 32 and 34, they may readily be so handled that the genital catarrh is avoided. The method of handling the newborn calf in order to guard against this genital catarrh and other defects, has been somewhat extensively discussed in the companion volume, *Veterinary Obstetrics*. This plan greatly ameliorates or eliminates the genital catarrh, which I consider the initial evidence of the granular venereal disease. If a heifer reaches breeding age, free from the disease, the first coitus with a bull having a penis like that shown in Figure 102 will establish a marked vulvo-vaginitis.

The vesicular venereal disease of horses and cattle, page 360, is one of the most spectacularly transmissible diseases known to veterinary medicine: its transmission, according to my experience, is virtually total. I have not observed the disease introduced by an affected animal from one herd or area into another: it just appears as if from nowhere. Although it may evidently be readily carried from one establishment to another, the symptoms are so manifest that intelligent animal husbandmen promptly suspend breeding, whereupon the clinical manifestations of the disease soon disappear. Inferentially the virus not infrequently exists as a phagocyte in the genital system until the sire is sexually overloaded, his powers of resistance depressed, the virulence of the invader increased, and disease follows.

The pathological phenomena of the genital system are far more frequently attributed to bacteria than to protozoa, viruses or other invaders. The picture may be somewhat misleading because some investigators in this field have concentrated their studies upon this group of invaders. For example, the trichomonad disease has probably been associated with economically important genital maladies of cattle for centuries, but the attention of microbiologists has been

too largely focussed upon bacteria for the protozoan to attract attention. It was not until thirty-six years after Bang's valuable discovery, that Abelein[1] authentically showed that a trichomonad was responsible for many abortions and other types of genital disease. Lothe[124] had well described what now appears to have been trichomonad disease, three years prior to Abelein's contribution, but his audience failed to recognize the significance of his work.

Similar difficulties in authentically identifying the invader responsible for abortion and associated phenomena are furnished by the literature upon equine abortion. In 1916 Good and Smith[70] announced that the one essential cause of abortion in mares was the paratyphoid bacillus. Their conclusions were quite generally accepted, and a vaccine recommended as a wholly reliable preventive. Sixteen years later, Dimock and Edwards,[42] studying the problem in the same area, could attribute only 4.8% of equine abortions to the paratyphoid organism, found 26.5% due to streptococci, and classed 64.0% as due to unknown cause. After another four years of study[43] they announced that a large ratio of the negative cases were due to a virus. This conclusion was supported by Miessner and Harms.[138] The disturbances of function and structure, so far as recorded, are identical whether the infecting agent be paratyphus, streptococcus, virus or other invader. If the disturbances of function and structure (symptoms and lesions) which we term abortion be regarded as the disease, we then have one *disease* which may be caused by any one of many invaders; if we regard the *invader* as the disease, we have many diseases with identical symptoms and lesions.

At the time of Bang's discovery it was generally believed by veterinarians and animal husbandmen that there was one supreme invader which was responsible for all abortions due to or associated with infection. Later it became evident that there occur many bovine abortions in which Bang's bacillus is absent, while vibrios, staphylococci and various other invaders are present. Such disasters are sometimes designated "sporadic bacterial abortion" regardless of the number of miscarriages in a given herd or area, whereas those abortions in which Bang's bacillus is recognized are classed as "specific contagious abortion." No explanation has yet been submitted for such classification. Evidently it served to concentrate attention upon Bang's bacillus. In all abortions where bacteria abound, so far as yet revealed, the invaders are unquestionably transmissible to other individuals of the same species and vulnerability.

Many abortions, perhaps an overwhelming majority of those associated with bacterial invasion, are preceded by metritis or placen-

titis as the initial lesion. Metritis is necessarily attributed to infection, but by no means to any one species of microorganism. When uterine tuberculosis exists in a pregnant cow, she probably aborts, and the disaster is regarded as the result of a specific infectious disease, but it is not ordinarily classified as specific contagious abortion. Similarly in wound infection, the invaders are legion in species and undeniably transmissible from animal to animal. The disturbances of function and structure are commonly alike for all. There are outstanding exceptions, such as tetanus, which is unavoidably classed as a specific wound infection, with clearly defined symptoms separating it from the scores of other species of invaders. It is less frequent than numerous other infections, and the sum total of losses from tetanus is far below that of the other forms. The designation of those bovine abortions in which Bang's bacillus is present as "specific contagious abortion" is based, so far as known, either upon an outworn tradition or upon the admittedly greater numerical frequency. The economic losses per unit are alike regardless of the species of invader.

The causes of disease are at times classified as *predisposing* and *exciting*. This applies, with special force, to transmissible maladies. It appears advantageous, especially in the field of genital disease, that the causes be designated as *primary* and *secondary*. In this treatise it is endeavored, with the one notable exception of dourine, to present the errors in animal husbandry as the primary, and by far the most important cause of interferences with reproduction, and to regard the bacterial or other invaders as chronologically and economically secondary. This permits the grouping of the bacterial invaders associated with abortion in cattle or other species and invites earnest cooperative study by veterinarian and animal husbandman in an effort to reveal the primary causes of sterility, abortion, retained placenta, calf scours and the rest, in the hope that more accurate knowledge may result in the effective amelioration or elimination of the primary causes, rendering unnecessary the development of separate remedies for each of the secondary infections. Many reject this philosophy and ignore the primary causes, through which destructive invasion of the genital system is rendered highly probable or inevitable. The above point of view does not detract from the importance of Bang's bacillus; no effort is made to compare its frequency or destructiveness with those of the paratyphus, streptococcus or other invader. It is sought to consider the entire group collectively. Presumably, if calfhood vaccination is a panacea for Bang's disease, regardless of the transgression of any or all laws of

physiological reproduction, then a similar vaccine, derived from the paratyphus or streptococcus, should be equally effective in the control of those invaders. Hence the emphasis in these pages is placed first upon a proper respect for the basic laws of reproduction, as evolved through eons of time, not upon remedies for the disasters resulting from the needless transgression of these laws. It is herein contended that the primary causes leading to the invasion of the reproductive system by microorganisms showing a special affinity for these organs are generally, if not always, identical for the entire group of invaders. Accordingly the *primary* causes may be approached as being alike whether the *secondary* invader be Bang's bacillus, paratyphus, streptococcus, vibrio or other factor. Should the measures invoked against the primary causes of abortion, sterility and associated disasters fail, the way remains freely open for the use of disinfection, quarantine, artificial immunization and other means directed especially against the individual species of invader.

Thus it is related that the 18 heifers shown in Table I: (a) were born of critically diseased mothers and were inevitably congenitally infected and non-resistant; (b) were erroneously fed and handled as young calves, and unavoidably suffered from diarrhea, malnutrition and serious repression of development (instead of having been so handled as to escape the usual genital catarrh as illustrated in Figures 32 and 34, they developed the signs shown in 31 and 33); (c) were bred when half grown, without opportunity for any possible degree of recovery from the injuries suffered under (a) and (b). Faced by this triple burden, each of which was avoidable, they were inevitably doomed to sterility, abortion and other allied disasters. The secondary invasion by bacteria was not eliminated or ameliorated by the use of the flamboyantly advocated bacterins; there is no ground for thinking that calfhood vaccination would have overcome such transgression of the laws of health; neither vitamins nor hormones could have prevented disaster; quarantine and slaughter would have eliminated any dangers of these heifers for others, but would not have protected other heifers in the least from the effects of improper transgression of hygienic laws. Prevention must be based upon the primary factors, and until this fails there is no attractive reason for attacking the secondary elements.

Apparently neither bacteria nor other invaders are usually identified in early abortions in animals. No clear explanation has been offered. It would appear that, if a healthy ovum were fertilized by such spermatozoa as those shown in Figure 28, the resulting embryo would be so defective that it would probably perish from inherent

weakness, without invasion by a pathogenic organism, as mentioned above. Mall[133] states that most human embryos aborted early are defective in their structure, and therefore would presumably not need to be invaded by pathogenic organisms that they might die and be expelled. But Mall does not deny the presence of invaders in the cadaver. Under general laws it would appear highly probable that, as the defective embryo approaches death, and almost certainly after death, bacteria or other destructive invaders would find their way to the cadaver. Similarly it is found that when a grossly defective ovum is fertilized, without the necessary elements for the development of brain or heart, it promptly perishes, except it attain adequate vascular anastomosis with a perfect embryo, in which case it may survive as a parasite until the mate is born, when it must immediately perish, like the monsters shown in Figures 23-25.

There are few exceptions in which the dead animal does not promptly undergo decomposition, whether extrauterine or intrauterine. When an inferentially healthy fetus passes from the uterus into the peritoneal cavity through a rupture, it may become encapsulated and continue static indefinitely, as indicated in Figure 166. So far as known, such cadavers contain no pyogenic bacteria or other destructive invaders. The highly distinctive bovine hematic mummy, page 523, has in its body and surrounding tissues no invaders causing decomposition, all fluids of the fetus and its envelopes are resorbed, and the cadaver remains year after year in a typical mummified state. There is some ground for suspecting the existence of an invader which reverses the usual action, preserving (mummifying) the dead tissues instead of causing decomposition. Instead of the hematic mummified fetus in the hermetically sealed uterus, there occurs somewhat infrequently the tesselated intrauterine hematoma of the cow like that depicted in Figure 188. I have observed clinically several cases of intrauterine hemorrhage following immediately after birth. There is enormous hemorrhage in the recently emptied uterus, without discoverable wound of the endometrium. Apparently the hemorrhage is from the placental capillaries. The blood is very bright red. It is discharged into an unquestionably diseased uterus, where its prompt decomposition would normally be expected, but instead it coagulates, remaining odorless, and the milk is colored a characteristic brilliant red, doubtless due to the absorption of hemoglobin from the coagulating mass, which is then excreted by the mammary gland. This would justify the suspicion that an invader prevents, instead of causing decomposition. Similarly in the phenomenon I have ventured to designate *static fetal cadaver*, the fetus at near

term dies, no hemorrhage from the placenta or elsewhere occurs, the amniotic and allantoic fluids are resorbed, while the fetal body retains its plumpness and becomes hard and unyielding as in rigor mortis and so remains from year to year without signs of decomposition or of mummification as described above.

An effort is made in Tables X and XII to show the variations in the rate of reproduction as affected by: (a) the degree of health enjoyed by young calves; (b) the age at which heifers were first bred; and (c) the breeding interval between calving and re-breeding. The 55 foundation heifers, none of which had previously calved, came from eight different purebred Holstein herds, scattered from Massachusetts to Minnesota, and were shipped by railway and boat 5000 miles to a tropical island of volcanic origin. They were maintained as a single herd, and lived day and night throughout the year in the open field, excepting the brief interval twice daily during which the lactating cows were confined in the stable for milking. As indicated in Table X, page 99, Group I produced 44.8% more viable calves per cow than did Group II. The only explanation available seemed to be that Group I had been more healthy as young calves and were held out of breeding as heifers until far more mature than the other group.

The far higher power of resistance possessed by the animals in Group I rendered them almost invulnerable to the invaders which were causing much destruction in Group II. This permitted Group I to achieve the highest average calf production I have observed or found recorded for an equally numerous unit of purebred dairy cows.

A study of the surrounding herds belonging to the same establishment, and in intimate contact with the purebred Holstein dairy animals, provides a highly interesting, and unusually instructive illustration of the relative significance of the primary and secondary causes in the interferences with reproduction.

The purebred Holsteins were centrally located, in an establishment of about 500,000 acres, with good grazing reasonably assured and a reliable artificial water supply which served the entire ranch. Sterility, abortion, retained afterbirth, calf diseases and other genital affections abounded in the purebred Holstein herd, gradually growing in severity, until within a decade the losses became unendurable. Changes were then instituted in the plan of rearing the calves, the age at which heifers should first be bred, and the rest interval between pregnancies. These changes markedly ameliorated the prevalence of disease. Hand breeding was practiced. The lactating cows had hay and grain in moderate amount.

Adjoining the terrain occupied by the purebred Holsteins was a herd of several hundred purebred Herefords for the production of high-class bulls for use as sires in the large herd of grade Herefords mentioned below. Hand breeding was followed exclusively. The cows were constantly in the open field, and received neither hay nor grain. The bulls were kept together in a commodious enclosure with facility for grazing and were allowed some grain. For mating, the desired bull was quietly driven by mounted men to the herd of cows, and following a single service was returned to the bull paddock. As indicated in Table IV, different sires were caused to serve various numbers of cows during the year. My records do not indicate the number of services per day by a given sire. My impression is that a bull rarely served two cows in one day. The number of services per annum was not excessive as measured by the usual standards. In the tropics, the breeding season could well be prolonged. Sterility, abortion, calf diarrhea and other allied diseases prevailed to an undesirable extent, especially during a year of limited rainfall, as indicated in Chart I on page 98. During extremely dry seasons the grass became scant and woody, the better species of grasses becoming inactive, and drought-resistant species prevailing.

Nearby the purebred Holstein herd was a long-established grade Holstein dairy herd of approximately 100 cows. These were pasture bred and the calves were permitted to suck their mothers for a brief period twice daily. Except for the pasture breeding and near-natural growing of the calves, there were no recognizable differences of importance in environment or handling from that of the purebred Holsteins. No important interferences with reproduction were reported.

Surrounding the terrain occupied by the three groups mentioned above, was a grade Hereford herd of 25,000, in which it was planned to produce 5,000 to 6,000 calves per annum. One paddock, through which I passed daily, in which more than 1,000 breeding cows were kept, was separated from the purebred Holsteins by an ordinary wire fence. The fence was sometimes crashed by bulls, and sometimes a gate would be left unfastened. There was abundant opportunity for the passage of any infectious disease from one group to another. The bulls for this herd, in a ratio of one to each ten cows, were supplied from the purebred Hereford herd described above. Neither bulls nor cows knew grain, hay, silage or other cured or prepared food. I rode over an ear of corn, which had accidentally dropped from a wagon and was lying in plain sight in the roadway, in the paddock occupied by over 1,000 cows and bulls. It lay for

several days until finally crushed under the wheels. The cattle, unacquainted with maize, accordingly let it alone. During one and one-half years I saw among these cows one case of dystocia, and one of uterine prolapse, both in heifers. I did not observe a retained afterbirth, or a calf suffering from diarrhea. There were no fecal masses adhering to the tails of the calves. The preputial and vulvar tufts of hairs were clean, like those shown in Figures 32 and 35. Although some abortions must have occurred, they regularly passed unseen, and these and allied losses were too restricted to arouse serious inquiry.

The herd had existed for over half a century without, so far as learned, having suffered seriously from infertility. A fairly reliable index of the genital health in a large herd is the sex ratio of calves. The records show that during the 17 years from 1913 to 1929 inclusive, 89,570 calves of branding age were produced, showing a male ratio of 102.45:100.00 This yielded over 2,000 heifer calves per annum. Large numbers of these were held in sex isolation and fattened for market.

In one area along the outer rim of the establishment, upon the slopes of a long-extinct volcano, at an altitude of about 7,000 feet, was maintained a herd of 20,000 sheep, primarily for wool production. They were constantly out of doors, were pasture bred, and were wholly dependent upon grazing for food. No abortion, pregnancy disease of ewes, diseases of lambs or other important interferences with reproduction were reported during the several decades of the existence of the herd.

In another area, bordering upon the outer fringe of the grazing lands of the grade Herefords, high up on the slopes of an active volcano, were countless hundreds of wild sheep and goats. These were the escaped progeny of domestic ancestors, introduced about a century before. They were dependent for sustenance upon herbage growing within limited oases, surrounded by vast flows of undecomposed, sharp lava. The lava beds were essentially impassable for horses, cattle or men. No predatory animals existed on the island. The character of the lava fields rendered them impassable for soft-footed wolves, dogs, or other such animals. There were no springs, lakes, streams, or other source of water supply than dew upon herbage. There was no salt or iodine.

The wild sheep and goats multiplied all too luxuriantly for the economic prosperity of neighboring animal husbandmen. When the bands became too large for the food supply in the oases, they invaded the grazing areas of the cattle. When opportunity offered,

mounted horsemen surrounded a few hundred and provided for their slaughter. They obstinately refused to 'catch' contagious abortion, in marked contrast to the sheep and goats in those lands where they are used in intense dairying. In the center of the scene, the purebred Holsteins demanded serious attention in order to combat sterility, abortion, calf scours and other phenomena interfering with economic reproduction; upon the periphery all readily available means were devoted to curbing the undesirable overproduction of the wild sheep and goats. The slaughter advocated for the control of abortion was equally effective in curbing the undesired fertility in the wild sheep and goats: the slaughtered dairy cow ceases to abort, and the slaughtered sheep or goat ceases to breed.

There had been created an extensive assembly of domestic and wild animals, in an unsurpassed panorama for the observation of the effects of environment and handling upon the transmissibility and virulence of bacterial and other invaders having special affinity for the reproductive system. The situation was unclouded by any technical experimentation, but developed naturally, from the period when the newly introduced animals escaped from the natives into the lava beds and reverted to the wild state, until the advent of the purebred Holstein dairy herd more than a century later. While the observations are admittedly incomplete, like all observations in the field of the pathology of reproduction, a summary of the above supplies some interesting facts, which justify some conclusions:

1. The ravages of genital disease were by far the most destructive in the purebred Holstein herd.

(a) The bulls and the lactating cows had a grain ration added to grazing.

(b) Hand breeding was followed.

(c) The calves were artificially grown and suffered from diarrhea and chronic indigestion, with fecal soiling of the tail and perianal region. There was distinct malnutrition.

(d) Heifers were bred at 15 months, when about half grown, and calved at 2 years, while yet decidedly immature.

(e) They were hurriedly rebred after having calved or aborted, sometimes as early as 30 days postpartum.

(f) Heavy lactation was sought.

2. The adjacent purebred Hereford herd came next in the order of losses from genital diseases.

(a) The bulls were fed grain. The cows were wholly dependent upon grazing.

(b) Hand breeding was followed.

(c) The calves were naturally raised by their mothers.

(d) The heifers were bred at about 24 months.

(e) After calving, it was aimed to rebreed at 70 to 80 days, with intent to have them calve each 12 months.

The most important losses in this herd occurred prior to the introduction of the Holstein herd and veterinary measures taken for their control.

3. The grade Holstein dairy herd experienced no important losses from breeding troubles.

(a) A very scant allowance of grain was given the lactating cows; the bulls had no grain.

(b) Pasture breeding exclusively.

(c) The calves were reared by the near-natural plan of permitting them to suck their dams briefly twice daily.

(d) The heifers were not bred until fairly mature, about 20 to 24 months.

(e) It was aimed to rebreed the cows at 70 to 80 days postpartum.

(f) Heavy lactation was not demanded.

4. The grade Hereford herd of 25,000 animals was without significant losses from genital disease throughout its history of more than half a century.

(a) Neither bulls nor cows had grain, hay or other processed foods.

(b) Pasture breeding, with a ratio of 1 bull for each 10 cows, was practised.

(c) The calves were naturally grown with their dams.

(d) The heifers were bred at 24 to 27 months.

(e) The rebreeding after calving was not under artificial control: it presumably occurred at about 85 days postpartum, to produce annual calving.

(f) Lactation was scant, the calf removed at about 6 months, and the cow dried off spontaneously.

5. The herd of 20,000 wool-producing sheep were without reported losses from genital disease.

(a) They were wholly dependent upon grazing.

(b) They were pasture bred.

(c) The lambs were naturally grown.

(d) The ewe lambs were held out of breeding until well matured.

(e) It was aimed to breed for one lamb annually.

6. The wild sheep and goats reproduced so luxuriantly that they constituted an economic burden. They were without water or salt, and by overproduction consumed all grass and other herbage in their

oases and then raided the grazing grounds of group 3. They bred according to nature's laws.

Each of the six groups constituted an authentic control upon each of the other five. They served to illustrate, with exceptional clarity, the effects of the various methods of feeding, breeding and handling, and upon the transmissibility and virulence of bacterial and other infections of the genital organs.

The country of origin and the date of the introduction into the island varied. The oldest group, represented entirely by the offspring of the originally introduced ancestors, are the wild sheep and goats. They were introduced by Vancouver, but from what European country they were derived is unknown. The next oldest herd is the beef Herefords, group 3. This was established more than half a century ago, by an immigrant from Massachusetts. The initial breed was not learned. It was later graded up by the establishment of group 2 of purebred Herefords, which supplied the bulls for the large herd. Group 2 has existed for several decades, the foundation animals coming from the United States, from areas where contagious abortion is ample.

Group 1, the newest herd, was established in 1920 by importations from Massachusetts. Seventeen heifers constituting Group I in Table X came from an exceptionally healthy herd, which had not been pressed for milk records. Three other heifers were obtained from a more fashionable herd, with considerable genital disease, and a year later 35 more heifers were purchased from various states in the Mississippi Valley. They doubtless carried with them ample numbers of Bang's and other bacilli associated with genital diseases, although all were apparently healthy according to the prevailing standards. While the older groups retained their established fertility, the Holstein dairy herd developed genital diseases of highly destructive character.

For economical reasons, the animal husbandman needs to modify the established laws of reproduction, in order that he may obtain a greater amount of meat, milk or other valuable product. The primary necessity of animal husbandry is the birth of an adequate number of healthy young to supply the needs of the community and of the state. The dairyman strives to add to this production of young an exalted activity of the mammary gland, by which he may obtain a vastly increased quantity of milk and butterfat. Since the mammary glands are part of the genital system, this places a dual load upon the dairy female. Stockfleth[188] stated, sixty-five years ago, that in the dairy cow there was a war between the uterus and the udder,

with the uterus commonly losing the battle, resulting in abortion.

In the above panorama providing observations and records upon large groups of animals upon one ranch, where conditions were unusually favorable for accurate study, some of the facts which affect the fertility of domestic animals seem to stand out clearly. Among the observations recorded above, there were mentioned:

(a) The food supply. In Groups 1, 2 and 3 there was some artificial feeding. There was no high feeding. If the feeding caused injury, it was possibly indirect, especially with the bulls, by causing them to refrain from ample physical exercise. On the other hand, as may be seen by a careful study of Chart I (page 98) and Table X (page 99) a want of adequate food apparently depressed fertility. When the rainfall was inadequate, the grass was less in quantity and nutritive value. So far as could be seen, the low rainfall led to diminished vigor, resulting in absence of estrum and ovulation. I saw no significant evidences of greatly increased abortion. I recall vividly two aged purebred Hereford cows, which during the severe drought of 1920 lost all their incisors pulling at the tough grass. They were in advanced pregnancy, and finally went down, unable to rise. They were knocked on the head, and the fetuses removed by hasty laparotomy. They were apparently healthy, were soon on their feet, and did well. If starvation would cause abortion, they should have aborted. Yet it seems clear that, if infection abounds in the uterine cavity, starvation would increase the danger of abortion.

(b) Pasture and hand breeding. These have been briefly described on pages 167-175. The hand breeding in the purebred cattle described above did not involve the usual rigid control of either sex. When a cow was discovered in estrum, the bull with which it was desired to mate her was gently driven by mounted men from the bull paddock into the enclosure occupied by the cows, guided to the desired animal, permitted to copulate once, and returned to his paddock.

In pasture breeding the bull regularly serves the cow as soon as she is in heat; in hand breeding the mating awaits the discovery of the estrum by the attendant. Ordinarily the observations were limited to twice daily and comprised a period of 12 hours or less. When the ideally healthy cow comes in estrum after the final observation of one day, she may well be out of it before the next day's inspection. At any rate, the heat period would be approaching its end. There is no authentic proof of the probability of conception as affected by the period of heat at which coitus occurs. Some advise that service is best just prior to ovulation, and contend that this occurs near the

end of estrum. No authentic study has been made regarding the influence of coitus upon ovulation. It is known that the doe rabbit does not ovulate until after copulation. If coitus does not occur, the ripe ova degenerate and are resorbed. Schmaltz[178] places the domestic cat in the same category, while other authors believe that coitus greatly hastens ovulation in the feline species. These observations suggest that in other domestic females copulation hastens ovulation. The belief seems justified that it is advantageous to cause early coitus, thereby hastening ovulation and abbreviating the duration of estrum. There is no evidence to indicate that the ovum, after having ripened to the extent to cause heat, improves by prolonged retention within the ovary. The view held by some, that it is advisable to delay breeding until late in the estrual period, is perhaps largely based upon prolonged heat periods. These, in my observation, are largely pathological, and the breeding of such females undesirable. When the heat period is definitely prolonged, conception is uncertain and pregnancy insecure.

Sexual overloading of sires occurs in both hand and pasture breeding. Table IV indicates that a sire may readily be overburdened by hand breeding. Numerous instances might be cited. The inclination is to regard some exceptionally vigorous sires as typical for the species. Schmaltz[178] mentions the thoroughbred stallion, The Sand, as having regularly served 200, and in one year 271 mares. The noted American trotting stallion, Hambletonian 10, had a similar record. But Schmaltz and all other eminent authorities recommend but one-fourth to one-third of this number of females in hand breeding. The danger is especially acute in dairy cattle during recent years in the creation of *proven sires*. In this industry the young bull is severely overloaded, as already mentioned above, in order to magnify the selling value of the sire.

Bulls used for pasture breeding are quite frequently overloaded because of the low ratio of males employed. This was controlled in group 4, the ratio being fixed at 1:10, all of which were well matured. In the absence of a safe ratio of males, danger of excessive coitus inevitably arises, immature, senile and weak sires especially suffer, and their inefficiency depresses the ratio of conceptions, the pregnancies are rendered unsafe, and the viable young are lowered in value. Had immature, or senile bulls been included among the sires in group 4, they would have been driven into isolation and the cows would have been almost wholly served by mature bulls of high vigor. With an abundant ratio of sires, pasture breeding closely approaches the natural method of reproduction among the wild.

Bulls used in hand breeding, as described above, had more physical exercise than many of the stabled sires, but it was not highly invigorating. Supplied with some grain and hay, they were not compelled to travel much for forage, and their water supply was close at hand. The pasture-breeding bulls in group 4 necessarily travelled over a considerable area for forage and were frequently obliged to walk one to several miles for water. The pasture bulls were compelled at times to maintain their harems by combat, while in hand breeding battle was waged only against new arrivals in the paddock; once installed, fighting was not interesting, in the absence of cows.

The influence of sexual desire and its satisfaction by coitus upon physiological reproduction has not been determined. In natural breeding the sexual desire of both male and female is developed with the advent of estrum, promptly attains its maximum, and is gratified by coitus. When hand breeding is practiced, the arousal of sexual desire is abrupt and coitus is at times attempted prematurely, and ejaculation of semen by the male fails, requiring a second effort. There seems to be ground for the belief that pasture, or natural breeding, evolved through eons of time, is superior in its final results, to hand breeding and to artificial insemination, as measured by conception and the birth of adequate numbers of healthy young. Hand breeding may be so employed that it retains almost all the values of pasture breeding. It has certain outstanding advantages. Repeated coitus in rapid succession with one female may be eliminated, and the sexual powers of the sire thereby conserved. The quality of the breed or family may be advanced through the intelligent selection of the two animals to be mated. The time of conception being known, the approximate date of parturition may be estimated. The duration of pregnancy is known, enabling the breeder to determine the future value of the young for breeding or other uses. The problems involved can not be solved in a single breeding season, but demand for authentic decision observations through several generations. In the meantime it seems prudent so to practice hand breeding that it approaches as nearly as possible the natural method. Whatever the conclusion may be, it is certain that in the two groups of cattle described above, where hand breeding was practiced, fertility was definitely lowered and genital disease increased. But the marked amelioration of the pathology of reproduction in group I, following radical changes in the growing of calves, the advancement of the age of heifers at first breeding by 60%, and the interval between pregnancies increased by about 200%, indicate most emphatically that these factors played the major role.

Chapter XXI

BACTERIAL INVADERS

WHEN abortion occurs in any species of domestic animal, the aborted fetus, its fetal membranes, and the uterine cavity often contain recognizable bacteria. The bacteria found are generally regarded as the cause of the abortion, premature birth, retained afterbirth or other disease phenomena present. If the invader be cultivated and a sufficient amount of it be introduced directly or indirectly into the blood stream of another equally susceptible animal, it may abort and the bacterium be found in the aborted fetus.

When a microbiologist searches an adult cadaver, the identification of a given pathogenic bacterium or other invader can not in itself authentically fix the cause of death. When glanders is diagnosed, the law generally dictates that the horse be promptly destroyed, and the discovery of the glanders bacillus in the cadaver can not show whether death was due to bacteria or bullets. Should an aborted fetal cadaver yield a microorganism which the examiner believes pathogenic, the conclusion is commonly drawn, that the recognized invader is wholly responsible for the death of the fetus.

This view largely, or wholly ignores those factors which have been classed as the primary causes of abortion in the preceding chapter. If no pathogenic invader is identified, to which the investigator chooses to assign the death, he by-passes the many obvious primary causes, and, like Traum and Hart,[198] classifies the disaster as "Manifestations of abortion disease without demonstrable etiological factor," or, like Dimock and Edwards,[43] ascribes the abortions to an unidentifiable virus. Evidently if one outbreak of abortion may be due to an undemonstrable factor, then any number of such unrecognizable causes may exist: if one unidentifiable virus may cause a destructive enzootic of abortion, there may be countless species of viruses which may limit their destructiveness to the death of intrauterine young, without causing other significant evidences of disease.

Owing to such failure to give any trace of consideration to the primary causes of abortion, the clinician is left in hopeless confusion. For example, a practitioner writes that he had recently tested a valuable herd of beef cattle for Bang's disease, nearly one half of which reacted. The reactors were promptly quarantined. Abortion was plentiful, and almost equal in both groups. The client became querulous. It was impossible for the practitioner to show that the force which

caused the abortions in the Bang-negative cows was not equally active and abundantly able to cause the abortions in the positive group, without the invasion by Bang's bacillus. Presumably this primary cause was definitely established in the entire herd, before the veterinarian had induced his client to have the test applied. So far as advised, the practitioner had not inquired into the sexual health of the herd bull; he may have had seminal vesicles, like those shown in Fig. 138, and the breeding inefficiency of which is shown in Table II, or he may have ejaculated spermatozoa like those shown in Fig. 130. In such, and countless other defects and errors in breeding, invasion by pathogenic organisms is not known to be essential to embryonic, or fetal death. In fact, as stated in Chapter III, those defective ova destined to become amorphous, acardiac or asexual monsters, reverse the hypothesis that the death of a fertilized ovum is necessarily dependent upon invasion by a pathogenic microorganism. Instead the fertilized defective ovum itself must attain parasitic relations with a normal twin, or perish.

The list of bacteria found in aborted fetuses, fetal membranes and the uterine cavity of the aborting female is extensive, and constantly increasing as researches advance. Stoss[189] attributes three-fourths of all equine abortions to infection and believes almost all of these due to *Bacterium paratyphi abortus equi*. Lütje[126] studied 1472 aborted equine fetuses and found:

Paratyphus infection.	41.7 per cent.
Diplococcus infection.	7.00 per cent.
Colon bacillus.	3.6 per cent.
Pyosepticum bacillus.	0.95 per cent.
Streptococci.	0.24 per cent.
Negative	48.3 per cent.

Dimock and Edwards[42] in contrast found among 325 fetuses:

Paratyphus infection.	5.8 per cent.
Streptococcus.	26.5 per cent.
Colon bacillus.	1.2 per cent.
Staphylococci.	1.8 per cent.
Pyosepticum.	0.6 per cent.
Negative (including twins)	64.00 per cent.

Later, Dimock and Edwards[43] announced that amongst the thoroughbred mares of Kentucky there existed a highly destructive type of epizootic abortion due to a filterable virus, page 363. This served to decrease to an unstated extent the 64 per cent of negative abortions in the prior report, took a large ratio of equine abortions out of the bacterial group, and placed them among the filterable viruses.

Later Miessner and Harms[138] investigated an outbreak of abortion in a harem of 23 mares, 17 of which aborted. Rigid search failed to

find bacteria, and Miessner and Harms felt compelled to ascribe the disaster to a virus.

Numerous influential writers claim that mares abort from dourine. Thus equine abortions are ascribed to bacteria of many species, to protozoa and to viruses.

Abortion and allied phenomena in dairy cattle have occupied the leading position in the researches upon animal diseases for four decades and naturally the list of bacteria invading the pregnant uterus is greater than that known for any other animal. Lütje[126] lists especially six species of bacteria, in addition to tuberculosis, which has already been discussed—*Bacillus abortus* (Bang), paratyphus, vibrio, diplo-streptococci, colon bacilli and bacillus pyogenes. The relationship, if any, between these and similar forms occurring in the mare is undetermined. Hutyra and Marek[98] present a similar list of bacterial species. Recently Abelein[1] drew attention to the prevalence of trichomonad disease, which is chiefly characterized by death and maceration of the fetus with obstinate pyometra. His observations have been duplicated in rapid order in many countries. There remain, after those abortions attributed to bacteria and trichomonads, almost 50 per cent of negatives yet to be acceptably explained. If the views of Dimock and Edwards and those of Miessner and Harms are accepted, the interesting possibility of virus abortion in cattle arises.

The prevalence of a given variety of bacterium apparently fluctuates widely according to location and date. Udall[205] reports that in the ambulatory clinic of the New York State Veterinary College, among 253 abortions studied 38.7 per cent were negative for *Bacillus abortus*, while more than 80 per cent of the cases of retained afterbirth, metritis and dystocia were negative.

Lienhardt, Kitselman and Sawyer,[120] reporting their findings upon materials from 119 cows which had recently aborted, identified *B. abortus* in 34.5 per cent. Among the 41 cases yielding *Bacillus abortus*, twenty other pathogenic bacteria were also present. Among the other 65.5 per cent negative to *B. abortus*, 59 cases yielded pathogenic forms and 19 were sterile. Udall's observations were in a highly concentrated dairying region; those of Lienhardt, Kitselman and Sawyer were among beef breeds and dairy cows not under high pressure. The ratio of infected to negatives was reversed in the two observations.

Lütje[126] recognizes, as causes of abortion in swine, *B. abortus*, paratyphus, and diplococci. Most writers mention especially the *B. abortus* infection.

The first infection to be prominently mentioned in association with abortion of sheep was a vibrio, closely resembling that found in aborting cows, and it still retains much of its prestige. Lütje assigns first place to paratyphus and recognizes diplococci as causing some abortions. In association with me, Carpenter[26] studied a highly virulent outbreak of genital disease in a flock of 235 ewes. The flock produced 57 viable lambs per 100 ewes; the others aborted or dropped non-viable lambs. Ten per cent of the ewes perished from puerperal sepsis. Studies were made upon twenty-four aborted fetuses or lambs which died young or were killed soon after birth. Ten of them yielded spirilla, and nine of the ten also revealed other bacteria including streptococci, staphylococci and colon-aerogenes. Twelve yielded other bacteria without vibrios.

McFadyean and Stockman[128] report studies upon outbreaks of bovine abortion in numerous herds, with an abortion rate of 25-50 per cent. They observed chiefly the vibrios and also considered them the cause of infectious abortion in sheep. Other bacteria present in some cases were not deemed significant. Howarth[93] studied a virulent outbreak of genital disease in a herd of 820 ewes. Two hundred and nineteen aborted, of which 101 died. The symptoms were like those described by Carpenter and myself. Vibrios could not be found. A colon-like organism was found, and the injection of cultures of this into ewes was followed by abortion. Hindmarsh and Blumer[92] observed 120 abortions in ewes. One-fourth of the diseased ewes died. They recovered cocci and bacilli, but no vibrios. The symptoms were as described by Howarth. Harms[80] mentions, as the cause of infectious abortion in ewes, an organism closely resembling *Bacillus abortus* and designated *Bacillus paratyphi abortus ovis*. Cesari,[29] studying the problem of the prevention of undulant fever in man, cites numerous investigations by high authorities, in which the disease in man was apparently caused by the contact with, and consumption of the milk, of ewes positive to the agglutination test for *Micrococcus melitensis* (Bruce), which if not identical with, is unquestionably closely related to *Bacillus abortus* and pathogenically interchangeable with it. As in cows, so in ewes, according to available literature, the infection is most common and virulent among those used for dairying.

Among goats the *Micrococcus melitensis* type of organism prevails. The goat, not only through contaminated milk, but by contagion to the milkers as an inherent peril of their work, originally occupied the first place in the consideration of undulant fever in man. Probably other organisms are present in abortions in goats, as well as in

other species of domestic animals, but the prominence of the goat in the undulant fever problem has served to rivet the almost exclusive attention upon the melitensis organism.

The incidence of abortion in goats harboring *Micrococcus melitensis* has been both asserted and denied. Apparently the observations have not been made in similar environments. The researches of Meyer and Eddie[136] present incontrovertible evidence that the abortion ratio is very high in herds where a large proportion of the animals are positive to the agglutination test. As in swine, so in the goat, the infection appears to be of exalted virulence.

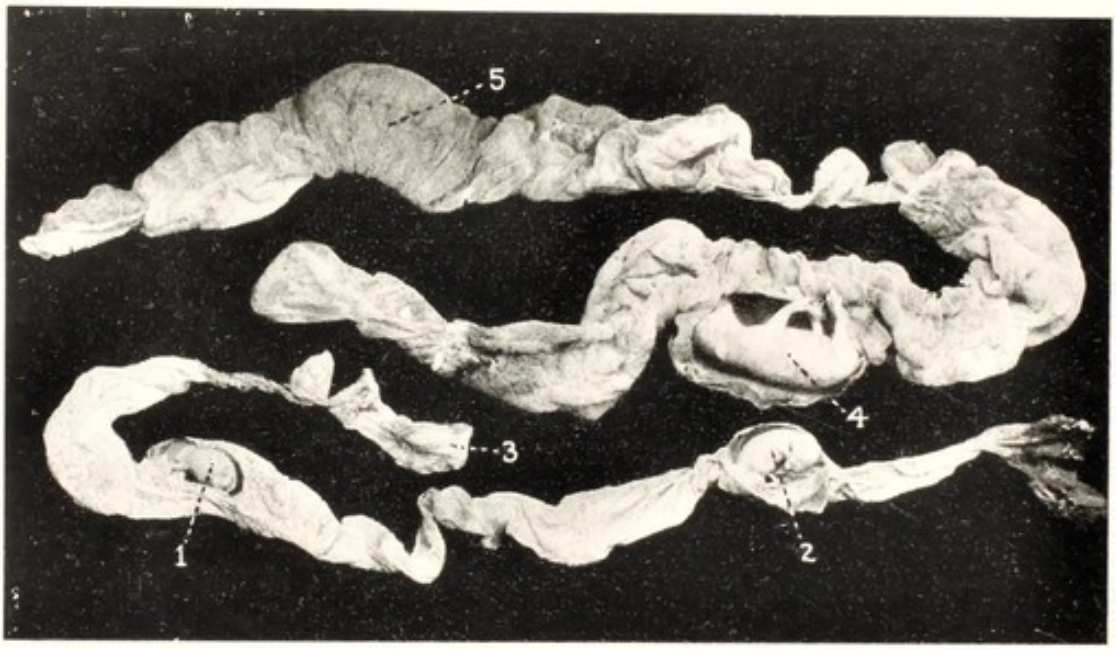


FIG. 91. Necrotic Swine Embryos.

1, 2, Necrotic embryos, the membranes of which have been incised to bring the embryos into view; 3, a necrotic embryonic sac in which the embryo can not be identified; 4, 5, healthy fetuses from the same uterus.

If abortion be defined as the observed expulsion of a fetal cadaver or of fetal cadavers, it is preeminently a phenomenon of unipara. When a uniparous fetus perishes, the earlier it is expelled, the less danger to the uterus from fetal decomposition. Once the uterus has been emptied of the cadaver, the maximum facility is provided for any available recovery of the inevitably diseased maternal placenta, and the greatest opportunity is offered for the preservation of fertility. Among multipara, the expulsion of a fetal cadaver, while there are numerous apparently healthy fetuses present, almost inevitably involves also the expulsion of all these and thereby causes serious interference with reproduction. The pregnant uteri of sows commonly contain one to several dead ova, which as a rule are not expelled before term. Instead they are resorbed if they die in early

pregnancy, or if larger, as in Figs. 18, 91 and 92, they become desiccated as papyraceous mummies. They apparently carry no great peril for the other fetuses, which may develop approximately normally. When birth occurs, the papyraceous remnants are expelled along with the normal young, and are immediately eaten along with the fetal placenta. They are not mentioned as abortion. When a multiparous fetus dies at or near term it tends to undergo putrefaction with marked emphysema. Again this is not classed as abortion by most observers. Abortion in multipara is generally taken to mean the simultaneous emptying of the uterus of all fetuses either dead or non-viable. In such cases, as already stated, *Bacillus abortus* is often present in swine. The bacteriology of the papyraceous mum-

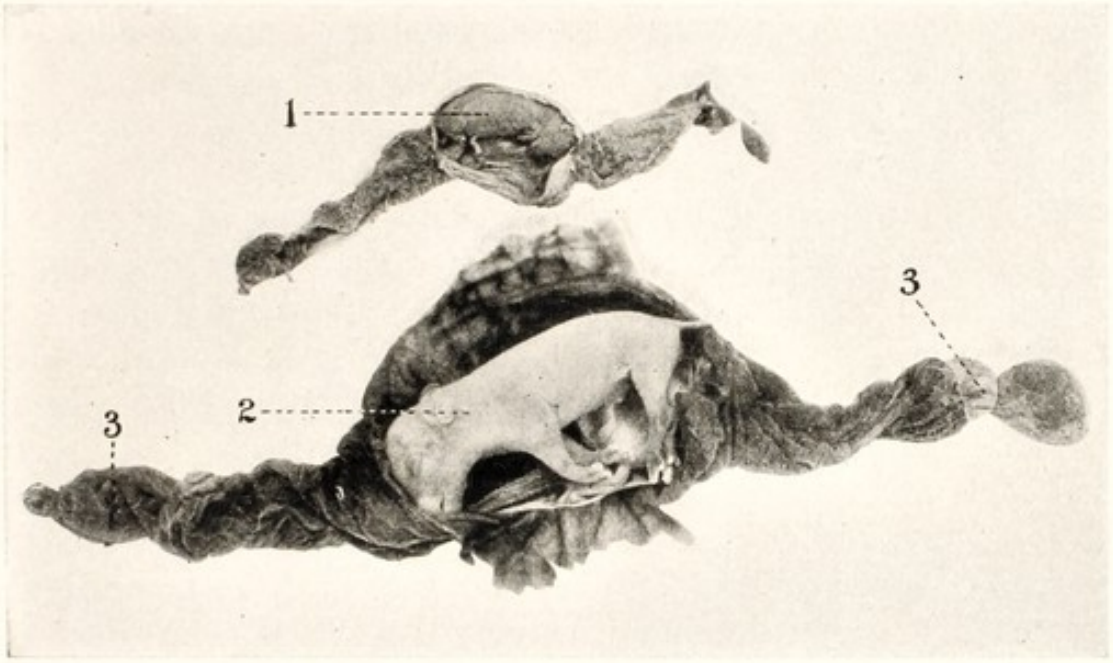


FIG. 92. Necrotic and Healthy Swine Embryos from the same Uterus.
1. Necrotic embryo; 2, healthy embryo; 3, necrotic tips of fetal sac.

mies, and of the putrid, emphysematous single fetuses in the presence of viable ones, is unknown.

By far the most search has been made for *Bacillus abortus* and it has been recorded in man, in all domestic animals, the domestic fowl, rabbit and guinea pig. Tunnicliff and Marsh[200] report the prevalence of the bacillus in the bison (*Bison americanus*) and in the elk (*Cervus canadensis* or Wapiti). Approximately one-half of the tested animals were positive by the agglutination test and several bison bulls yielded pure cultures from the testicles. Tunnicliff and Marsh failed to establish the occurrence of abortion in association with invasion. There are no records of extensive search for the bacillus in any species of animal, domestic or wild, in which it has not been found.

The available records justify the assumption that it may be present generally in wild ruminants.

The Symptoms of Bacterial Abortion

When a fertilized uniparous ovum dies very early, before any firm tissues have been formed, it promptly disappears without leaving definite trace. If the ovum of the cow, for example, dies within a few days after fertilization, its inhibitory action upon the corpus luteum is destroyed, that body disappears much as if fertilization had failed, a new follicle matures, and the cow may again be in heat at 21 days. It is impossible to differentiate such early death from nonfertilization. The ovaries of the multiparous sow, when examined shortly after conception, reveal, by the number of corpora lutea, the number of ova discharged but do not authentically indicate that all were fertilized. If an ovum has lived for several weeks, the uterus frequently shows in certain areas, embryonic debris.

Somewhat later, if the bovine ovum dies, it may in many cases be resorbed, or it may be expelled. Infrequently one sees hanging from the vulva an elongated embryonic sac, looking like a string of mucus, in which may be seen, upon careful examination, an embryo 5 to 10 mm. long. Most such ova pass unseen, and the phenomenon is designated *sterility*. Such embryonic death disturbs the estrual cycle and the cow is again in heat after an irregular interval of 30 to 60 or more days. The dead embryo is retained in the sow, if live embryos are present, and the cadaver slowly disintegrates or desiccates. The corpus luteum occupying the follicle from which the ovum came, so far as known, always remains intact.

As pregnancy advances, the death and expulsion of the uniparous ovum becomes increasingly obvious. Among stanchioned dairy cows, when a fetus 10 to 20 cm. long dies and is expelled, it is not infrequently found in the gutter, the ovum being complete, with all its membranes. As a rule early abortions in cows are not associated with recognizable bacterial invasion. The bacterial search in the dairy cow has been preeminently directed toward *Bacillus abortus* infection, which may have sometimes diverted attention from other invaders. There is much reason to suspect, in a large ratio of cases of early abortion, where bacteria and other invaders have not been identified, that the fertilized ovum is so malformed or perilously weak, owing to fundamental defects of the unfertilized ovum itself or of the fertilizing spermatozoon, that it is incapable of embryological survival.

When pregnancy has become more advanced, and the bovine ovum perishes at the 4th or 5th month or later, the abortion may be recognized by the discovery of the fetus in a large ratio of cases. A second symptom then emerges: the afterbirth is retained for several hours. Bacteria of various types, and other invaders, appear in an increasing ratio of cases.

After mid-term, impending abortion is often revealed by untimely engorgement of the mammae, but this is not universal. Enlargement of the udder with lactation may occur in a non-pregnant female when in heat. When one of equine or bovine twins dies, there is frequently an associated engorgement of the mammae, although abortion may not occur; instead the dead fetus may undergo papyraceous mummification and the pregnancy may continue to term. The death of one or several swine fetuses, while others remain healthy, does not, so far as known, produce any visible symptoms: the mammae are not enlarged or otherwise changed; the sow exhibits no signs of ill health.

When an equine or bovine fetus dies at or near term, and the lesions are such that its expulsion follows, the abortion frequently occurs unheralded until labor sets in. In other cases there appear the usual symptoms of impending parturition. The expulsion of the fetus, when large, is regularly less vigorous and more prolonged than in physiological birth. The atonic state of the uterus, together with the inanimate fetus, leads frequently to dystocia.

The lines of demarcation between abortion, still-birth (born dead) and premature birth are technicalities which vary in their meaning according to the interpretation of the speaker. Thus Lothe[124] speaks of a cow which aborted and the calf lived. The designation was based upon the duration of gestation. Lubbehusen, Fitch and Boyd[125] report that certain cows calved normally in which the calf (fetus) was dead. When the important facts are stated the definition of the phenomenon is only of academic interest. Unless otherwise indicated, it is intended in this treatise that abortion, in unipara, shall signify the known expulsion of a fetal cadaver. In multipara, it shall signify the expulsion of fetal cadavers, unassociated with the birth of viable young. It will be aimed to restrict birth to the expulsion of viable young—that is, birth shall mean that living young has passed from the intra-uterine to the extra-uterine life.

The Lesions of Bacterial Abortion

Writers frequently report the examination of aborted fetuses in which they identified no macroscopic lesions. Some of them appar-

ently fail to clearly distinguish between the fetus and the ovum, with the latter term including the fetus and its envelopes and fluids. Some pass by the fetal membranes, which constitute the outer line of defense of the young, and are logically the first tissues, along with the endometrium, to suffer injury from bacterial invaders.

The inference is possibly drawn that the abortion was not caused by lesions. The conclusion is probably erroneous. I recently had occasion to examine, after slaughter as a non-breeder, a cow which had been repeatedly bred at intervals of 30 to 40 days. The uterus contained an ovum of perhaps 20 days. The small embryonic sac bore no macroscopically visible cotyledons, as was to be expected at that time. Upon the ovarian side of the ovum there existed a comparatively large volume of thick pus. The volume of pus exceeded the size of the ovum. The ovum would logically have been expelled at an early date, and had it, by chance, been found, it would not have shown macroscopic lesions.

The pus might readily have been expelled unseen. But lesions existed in the endometrium which were not clinically visible. The abortion would not have been lesionless.

The fetal membranes of the adeciduate domestic animals—solipeds, ruminants and swine—provide in each case, after their expulsion, an authentic replica of the maternal placenta. If the membranes are retained, the retention itself is proof of lesions.

My colleagues and I[243] in extended studies encountered no case of abortion in which disease was not authentically registered upon the fetal membranes, and it was indisputably shown that parallel lesions existed in the uterine placentae. The fetus itself sometimes fails to reveal lesions which are recognized. However, the fetal membranes and fetus jointly constitute the ovum, and the fetal placenta is by far the most vital organ of the intra-uterine individual. *As is the fetal placenta, so is the fetus.* The oft seen statement that the new-born young was physiological and that its afterbirth was retained is in direct conflict with the fundamental principles of reproduction and with clinical observation.

The lesions accompanying abortion vary according to genus and species. The placentae of ruminants are caruncular. The bovine uterus, as indicated in Fig. 15, has four parallel rows of caruncles, with a total of about 140. Corresponding with these is an equal number of cotyledons upon the allantochorion. The caruncles, as pregnancy advances, grow out from the general level of the uterine mucosa for a distance of approximately two inches. They constitute a vast colonnade, contacting the cotyledons of the chorion. The

resultant utero-chorionic space constitutes a vast sac. When near term the allantochorion of the cow, with a single fetus, is eight to ten feet long, and about three feet in circumference, so that the chorionic walls on one side and the uterine mucosa on the other provide fifty to eighty square feet of sac wall. The walls are indifferently vascular, compared with the caruncles and cotyledons. In this vast cavity, bacteria of favorable type may grow and spread widely without important mechanical barrier, within a comparatively neutral sac. This cavity is well illustrated in Colored Plate I.

The cavity serves as a commodious repository for any exudates and tissue debris which may result from the activity of invaders. In the presence of chronic infection the exudates and debris, mixed with the invader, may attain a volume of several liters. Early in the researches this exudate attained the distinction of "the exudate of contagious abortion." It is a yellowish, or brown-yellow, putty-like mass, extremely adhesive. It is largely massed about the stalks or necks of the caruncles, apparently because the caruncular columns tend to create a vacuum at this point, into which the exudate is pressed. The foci from which it usually extends are the cervix and the ovarian poles of the cornua.

The exudate and tissue detritus apparently emanate chiefly from the periphery of the placental areas, in which the notable macroscopic lesions occur. (Vide Fig. 39.) These consist chiefly of necrosis of the placental tufts of the fetal cotyledons and a corresponding destruction of the tissues of the caruncles. Both the uterine and chorionic tissues may undergo complete necrosis and the necrotic non-gravid chorion, in a state of papyraceous desiccation or of purulent decomposition, lie immersed in the exudate as shown in Colored Plate II. Inevitably the inter-cotyledonal area of the chorion, in contact with the exudate, undergoes important pathological changes. The chorion becomes edematous, thickened, and sometimes definitely indurated, of a leathery consistency. Naturally, in such environment, superficial calcification of both endometrium and chorion frequently occurs.

This "exudate of contagious abortion" is not readily observed after the female has aborted or given birth to young. The flood of mucus or other uterine secretions tends to dissolve the exudate and cause its disappearance. It is best observed in slaughtered animals while the fetus remains in utero and the membranes intact. It is distinguishable by careful inspection of the promptly expelled fetal membranes. It is not confined to aborting females, but may be present in females calving at term, with viable young.

Similar exudates occur in genital disease of ewes associated with abortion, non-viable young and metritis, but in my observations they are of a darker brown color. In the abortion of goats the attention has been focused upon the presence of *Micrococcus melitensis* and the associated undulant fever of man. I find no description of the lesions of the fetal membranes and endometrium; presumably they are quite similar to those in the cow and ewe.

The injuries to the uterine walls are without limit. Often the caruncles become necrotic and slough away. The necrosis may rarely include the entire endometrium and leave the cornual cavities as suppurating fistulae. Septic metritis is not rare in bovine abortion. In Table I, Heifer 1 died suddenly from acute sepsis without having shown signs of abortion. After having aborted, it is not at all rare for the cow to perish from sepsis so soon after aborting or calving that it is impossible to avoid the conclusion that the infection had existed within the utero-chorionic space for weeks or months. The incidence of mortal sepsis in abortion in ewes is appalling, reaching as high as 50 per cent.

The lesions accompanying bacterial abortion in the mare and other solipeds unavoidably differ from those in ruminants, owing to the absence of a utero-chorionic cavity. Research workers upon abortion in mares have generally focused their attention upon bacteriological tests and paid scant attention to the lesions in the fetal membranes and endometrium. Mares in the act of aborting or immediately following the phenomenon have not been destroyed and post mortem studies made. It is common to state that the mares show no abnormalities in temperature, appetite or general appearance, and that no genital discharge commonly follows. The diffuse placenta of solipids, contacting the entire endometrium uniformly from the two ovarian poles to the cervix, offers scant opportunity for pus or other product of disease to accumulate between the maternal and fetal placental tissues. In one mare I observed first a discharge of two or three liters of pus from the vagina. Upon vaginal palpation it was readily determined that it had come from an abscess cavity at the os uteri internum, the walls of which consisted of the chorion and the uterine wall. After the fetal cadaver was expelled and the fetal membranes came away, the walls of the chorion over the abscessed area presented the ordinary macroscopic appearances of an abscess wall of other parts; there was induration and very marked thickening. Such inter-placental abscessation is probably rare.

According to my observations[245] the lesions are most frequently general over the entire placental surface. Thus in Fig. 30, the lesions


5
4
3
2
1

Plate II

Gravid Uterus of Cow at About Four Months, Showing Abortion Exudate in the Non-Gravid Horn

1. Right (gravid) horn. 2, 2. Cut margins of non-gravid horn. 3. Cavity of non-gravid horn, the mucosa covered with the "exudate of contagious abortion." 4. Necrotic chorion of non-gravid horn covered over with brownish-yellow "exudate of contagious abortion." 5. Healthy base of the fetal sac of the non-gravid horn, continuous with the healthy fetal sac of gravid horn.



FIG. 93. Grossly Diseased Equine Allantois, Viewed from the Amniotic Side.

1, A Y-shaped fibrinous clot 12x2x1 inches. Upon placental side, opposite the center of the clot was an old brown necrotic patch which extended through the chorion to the clot upon the amniotic side. About the necrotic area the diseased chorion, 3, was greatly thickened, as compared with 4, a less diseased area. A smaller necrotic area is shown at 2. *The non-viable foal, born at term, died from sepsis after a few hours.*

which caused prolonged gestation (the mare carried the critically diseased foal 387 days—or one year, 22 days) included universal thickening of the chorion, which was deeply injected and, as shown in the figure, thickly studded over with small calcified areas. The afterbirth weighed 24 pounds, approximately two or three times the normal. Good and Smith[70] state that the incubation period of equine infectious abortion is ten to twelve days. Their conclusion seems to have been based upon the experimental inoculation in the jugular vein of pregnant mares with the organism they indicted for the abor-

tion. How widely that may differ from natural invasion is not known, but we do know that as a rule bacteria or other injurious substances injected directly into the blood stream act far more rapidly and surely. Most of the lesions I have observed in equine abortion were not produced in ten days. The afterbirth of the mortally ill foal, born at term, illustrated in Fig. 93, weighed eighteen pounds and,



FIG. 94. Vascular Allantois of Mare, Prepared Same as Fig. 162 in order to show vascular anastomoses near the mesian line opposite the mesometrial line of attachment.

1, 2, Arterial trunks; 3, 3, ordinary anastomoses; 4, multiple anastomosis. *(Cornell Veterinarian)*

besides the general induration and thickening, showed a necrotic area, Y-shaped, twelve inches long by two inches wide and one inch thick, involving the entire thickness of the allantochorion. A smaller necrotic area, also extending completely through the chorion, was so far developed that it fell out of the wall of its own weight during gentle manipulation.



FIG. 95. The Equine Vascular Allantois from a Nine Months' Abort, Showing Gross Enlargement and Obscure Outlines of the Sinusoidal Vessels. *(Cornell Veterinarian)*

The more common type which I have observed, consists of a diffuse placentitis, which some might term arterio-phlebitis. The vascular allantois of the mare consists of a highly spectacular vascular tissue in which the capillaries constitute a vast mass of vessels. The death of the vascular system and the fetus dependent upon it may be very swift. In a short time the death of the tissues, with serous infiltration beclouds the organ, the capillaries are enlarged, nodular in appearance, and constitute an indistinct mass of vessels as indicated in Fig. 95. While the spread of the infection was apparently very rapid, it is not known how long it may have existed in the cervical canal or in other convenient hiding place pending favorable opportunity for expansion.

The lesions of abortion in swine have not been notably recorded.

The Diagnosis of Bacterial Abortion

Following the discovery of *Bacillus abortus* and its virtually universal acceptance as the cause of all abortions resulting from contagion in cattle, the lesions found in the utero-chorionic space, with the presence of the peculiar exudate designated as "the exudate of contagious abortion," were generally accepted as authentic proof of the identity of the invader. When it became known that many species of bacteria invaded the pregnant bovine uterus and were associated with abortion, the confidence in the existence of lesions by which the species of the invader could be positively determined began to lose force. As early as 1909, in the course of abattoir investigations, I[234] had occasion to study more than twenty diseased uteri of pregnant cows, in the slaughter houses at Chicago, Kansas City, Omaha, Denver and Fort Worth. The uteri contained the characteristic "exudate of contagious abortion." Material from practically all these was forwarded to the Bureau of Animal Industry and to the bacteriological department of the N.Y. State Veterinary College, and from a majority of the cases to Dr. L. Enos Day in charge of the B.A.I. Chicago laboratory, for bacteriological search. *Bacillus abortus* was not recognized in any case by any one of the three laboratories. Smith[182] reporting studies upon a series of bovine abortions associated with spirilla, in which *Bacillus abortus* was absent, states that a macroscopic examination of the fetus fails to reveal lesions by which the bacterium to be found can be foretold. Later he states that the lesions in the fetal membranes associated with the two invaders are unlike, but fails to state in what respect. Plum[157] states that in bovine abortion due to tuberculosis the lesions are not

distinguishable from those of *Bacillus abortus*. Udall[205] states: "While the symptoms and lesions of Bang's disease are characteristic, they are not pathognomic, since identical phenomena may be caused by other viruses or causes." There are few today who consider that the lesions occurring in the uterus, fetus and fetal membranes, in association with bacterial abortion, afford secure ground for foretelling the species of bacterium which is present.

The differential diagnosis is based upon bacteriological search or serological or other tests based upon bacteriology. If *Bacillus abortus* is identified in the aborted fetus or its membranes, or if the blood of the dam reacts to the agglutination test, the abortion is ascribed to that bacillus.

Early abortions in the cow largely pass unseen. The abortions occurring during the first three months which have been studied have generally failed to reveal the presence of any species of bacterium or other invader. No adequate cause for these early abortions has yet been discovered. No logical reason has yet been offered to indicate that the factor which causes the very early abortions ceases to be effective with the advent of bacteria. If the pathogenic force which causes early abortion persists after the invasion of bacteria, some question may arise regarding the relative importance of the two forces. This is rendered more important by the fact that many abortions occur throughout gestation in which no bacteria or other accepted causes are recognizable. So there is room for the suspicion that forces not yet indentifiable act in numerous abortions, even though pathogenic invaders are recognized. Further studies of those abortions caused by as yet unrecognizable agencies would vastly strengthen the authenticity of diagnosis and might aid materially in any efforts at control.

The chief diagnostic methods are: microscopical search for the invader in the diseased tissues or excretions; the inoculation of vulnerable laboratory animals with the diseased tissues; the growing of the bacteria on suitable laboratory media; and the agglutination and complement-fixation tests. Positive recognition of given bacteria by any of the three first tests is factual and enables the observer to draw conclusions. The agglutination test, which is by far the most frequently used for diagnostic purposes, especially in the detection of the presence in the system of *Bacillus abortus*, is generally accepted as being highly reliable. Like all diagnostic means, it has its weaknesses. In 1912 I[232] inoculated three heifers in first pregnancy, intravenously, with living abortion bacilli. The heifers had been reared in isolation, had conceived at first service and were

negative to the agglutination test at 1:25 when inoculated. All became positive at 1:1000 in from eleven to twenty days; the titre dropped to 1:200 after 65-95 days and to negative at 1:50 in 137 to 164 days. One heifer aborted at 164 days. Her blood at that date was negative and her uterus yielded *Bacillus abortus* in abundance. Hart and Traum[84] report that in 29 milk samples positive for *Bacillus abortus*, the blood of the cows was negative to the agglutination test in six cases among fifteen cows. Doyle and Beckett[44] find that cows negative to the agglutination test not rarely show *Bacillus abortus* in their milk. Lubbehusen, Fitch, and Boyd[125] show that among fifty experiment animals they found three (six per cent) in which the blood of the cow was negative to the agglutination test while the milk, and in one case also the placenta, yielded *Bacillus abortus*. Comparatively little study has been devoted to the question, but the available records make it clear that an important ratio of animals carry abortion bacilli in the milk or in the placenta when the blood is negative to the agglutination test.

The Avenue and Date of Invasion

There has been great conflict of opinion during the past forty years concerning the date and avenue of invasion by *Bacillus abortus*, the principles of which are applicable generally to bacillary invasion of the reproductive system. The divergence of opinion still persists. When an ovum and a spermatozoon unite to constitute a new individual and one or both cells are defective, the fertilized ovum is inevitably weak and may readily fall prey to invading bacteria not highly dangerous for ideally healthy ova. When a fertilized ovum enters a uterine cavity containing bacteria not sufficiently virulent to bar fertilization by one of the billions of spermatozoa, the invaders may have the power to destroy the life of the young ovum.

Here arise conflicts in observation. In 1909, Dr. L. Enos Day in charge of the Bureau of Animal Industry laboratory at Chicago, at my solicitation made cultures from the uterus or the fetal fluids from forty-three bovine females in the abattoir. The results were published in 1918.[237] Eighteen non-pregnant uteri yielded bacterial cultures (streptococci, staphylococci, bacilli), while twenty-five gravid uteri yielded cultures in eight animals. The fetuses ranged from one-fourth to fourteen inches in length. They were largely discarded dairy animals. Hagan[76] reported the results of bacterial search upon twenty uteri. Among these were ten non-pregnant animals, eight of which yielded cultures, while among the sixteen gravid uteri, twelve

yielded bacterial growths and two were negative. The material studied by Hagan was all from dairy discards. Two of the non-gravid uteri were macroscopically diseased, and exudate was present in two of the pregnant uteri. The bacterial types included micrococci, streptococci, and colon organisms. *Bacillus abortus* was not identified. The duration of pregnancy was estimated at from two to eight and one-half months. Growths were obtained from the utero-chorionic space, umbilical vein, allantoic, and amniotic fluids. The materials of Hagan emanated from a concentrated dairying region, while those of Day came from a mixed beef-producing and conservative dairying area. Day made as a rule but one or two cultures per uterus, while Hagan made several. In the same publication also Carpenter reports upon the bacteriological search of the uteri of fifteen sterile or tubercular, non-pregnant animals, with bacterial growths from eleven.

Beller[10] reports extended bacteriological studies in cows and ewes at the termination of pregnancy. In one group of twenty-three cows, with two abortions, one case of septic metritis, one non-viable calf and nineteen healthy calves, Beller obtained cultures from the navel sheath or placenta or both in twenty-two animals. Among fifteen ewes he obtained cultures from the amniotic fluid in seven, from the allantoic fluid in seven and from the placental vessels in eight. Cultures from the amniotic fluid of eight cows were positive in all. The allantoic sac of the cow regularly ruptures too early in labor to facilitate reliable cultural tests of the allantoic fluid: one such culture was made and was positive. The placental vessels of all eight yielded bacterial cultures. Hagan[76] studied the meconium of fourteen calves from two herds and recovered bacteria from five (35.7 per cent) under conditions which indicated intra-uterine infection.

Numerous investigators claim that the uteri of healthy domestic animals are bacteria-free. Fitch and Bishop[60], after studying 126 uteri, secured bacterial growths in 28, or 22 per cent. They attribute the occurrence of bacterial grows upon their media to contaminations due to their errors in technic. They conclude: "It is our judgment, based upon our experiences and the results we have obtained in this study, that many of the bacteria mentioned by other investigators as occurring in the bovine uterus were external contaminants and were not present within the cavity of the uterus,"—"the healthy bovine uterus is, in general, free from bacteria."

The conflict of opinion between bacteriologists regarding the frequent presence of bacteria not pathogenically active in the uteri of clinically healthy females, places the clinician between Scylla and

Charybdis. It is a question of vital concern. If the clinically healthy uterus is uniformly free from bacteria or other invaders capable of producing disease, then the clinical veterinarian and the animal husbandman may neglect with impunity, in so far as eventual reproductive power is concerned, all regard for the health of the ovum,



FIG. 96. Necrotic Tips of Bovine Fetal Sacs.

1, 1, 1, Living chorion; 2, necrotic tips; 3, chorionic placenta. *(Cornell Veterinarian)*

the new-born and the young up to breeding time. It will only be necessary to erect a barrier against invasion during pregnancy. That has been the attitude in general in the past, and those who have watched the effects know only too well that the losses from reproductive diseases are advancing rapidly.

Fortunately it is not essential that in arriving at conclusions the

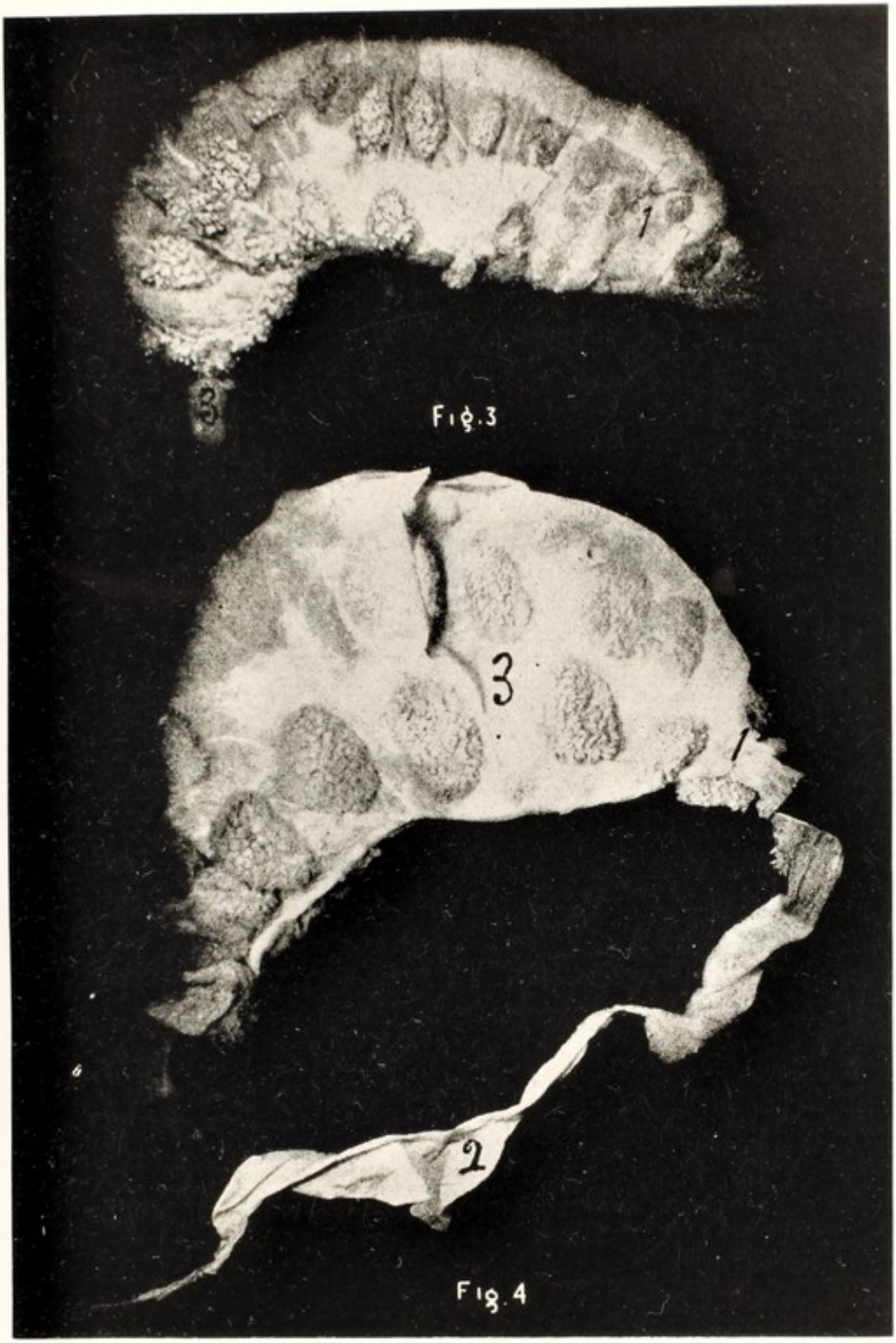


FIG. 97. Bovine Fetal Sacs from Uteri in Which the Endometrium Has Been Largely Destroyed.

Upper figure: entire endometrium of one cornu destroyed. 1. Cervical pole of functioning cornu, with imperfect cotyledons; 3, vestige of non-pregnant horn. Lower figure: 1, the cervical area of gravid horn; 2, the necrotic non-gravid horn; 3, the functioning horn of chorion.

clinician or animal husbandman shall decide which group of bacteriologists is in the right. In the contribution by my colleagues in the Veterinary College and myself[243], devoted chiefly to studies of the fetal membranes associated with abortion and sterility, it is clearly and abundantly shown that macroscopic lesions are present in the fetal membranes and the endometrium of the pregnant animals which serve to explain the pathological phenomena. Bacteriologists at least generally attribute these macroscopic lesions to invasion by bacteria. (The term "bacteria" is used here to include bacteria, molds, protozoa, and viruses.) If the enquirer will follow my directions for studying the bovine fetal membranes[244] and will study each and every fetal cotyledon in detail under a gentle stream of water, it will be rare indeed to find a single case in which necrotic chorionic tufts, plainly recognizable to the naked eye, are not present. When these are abundant, and *Bacillus abortus* is present, all bacteriologists declare that the necrosis is caused by that invader. If vibrios are found, the lesions are ascribed to them. If neither of the two are present, and the bacillus of tuberculosis is identified, that organism is indicted as the cause.

Traum and Hart[198] record the prevalence of abortion in the University of California "abortion-free" herd, along with associated phenomena. No bacteria were found and Traum and Hart ascribed the abortions to an undemonstrable cause. They apparently confined their search to bacteria, ignoring molds, protozoa, and viruses. So important losses from reproductive diseases may occur without bacteriologists being able to find bacteria. The clinician cannot evade the conclusion that abortion and associated phenomena are the natural outcome of certain lesions present and that these are brought about by pathogenic activities of living forces. These lesions are not confined to abortion and its associated phenomena. Many writers vehemently declare that the necrotic tips of the allantochorion of ruminants and swine are physiological and are in no way associated with the invasion of bacteria or other pathogens. The necrotic tip is universal. In the healthiest uteri it is a hard, desiccated cord, one or two centimeters long and one or two millimeters in diameter, invaginated and buried in the chorionic walls: there is no ground for designating it as disease, in the sense that it visibly imperils the well-being of the pregnant female or intra-uterine young. As a rule the necrotic tip is more extensive, as indicated in Fig. 96, and again there is no clinical evidence that it carries any threat for physiological reproduction. When the necrosis reaches the magnitude shown in Fig. 97, most clinicians of experience would regard it as distinctly

pathological and armed with definite peril to the life of the fetus and to the future fertility of the mother. When one meets necrosis of the tip of the allantois to the extent of that depicted in Colored Plate II, with the non-gravid cornu filled with pus, in which the necrotic tissues lie, few would deny that it is pathologic and that it is due to a living invader. Between such extensive lesions and the normal necrotic tip there is no line of demarcation and no possible means as yet available to ascertain which are due to bacteria and which are not. If the normal necrotic tip is non-bacterial, then logically the lesions in Fig. 96 and in Colored Plate II are non-bacterial.

Perhaps much of the confusion lies in the vagueness of the definition of *disease*. Prior to the advent of bacteriology, disease commonly indicated a visible departure from the ideal structure or functions of organs or of the body as a whole. When the science of bacteriology became established, a tendency arose to modify fundamentally the original definition of disease, and if bacteria known, or believed to be potent to disturb functions were found in an organ or system, the individual was thereupon designated as *diseased*. For example, Wing[271] reported that at the date of the issuance of his bulletin Glista Ernestine was the fourth cow of history in the lifetime production of milk and butter fat. A clinician or a dairyman would regard her as a preeminently healthy dairy cow; but at the zenith of her career her blood reacted at 1:500 according to the agglutination test for *Bacillus abortus*. This causes the bacteriologist to say that she had *Bang's disease*. There is no conflict regarding the facts.

The available evidence indicates, from the clinical viewpoint, that various bacteria may persist in the reproductive tract over an indefinite period, as saprophytes, ready to assume pathological activity at any time when the external environment, or the period in the generative cycle (pregnancy, puerperium) lowers the powers of resistance. When so viewed it becomes of fundamental importance to animal husbandmen and veterinary practitioners that all available measures be applied to reduce to the minimum the numbers and virulence of the invaders present and to advance to the maximum the powers of resistance in the breeding animal.

Bacillus abortus, which has commanded far more study than all other invaders of the reproductive system combined, attacks first the fetal membranes and then the fetus. As stated above, there regularly exists, whether Bang's bacillus be present or absent, necrosis of some chorionic tufts and corresponding lesions within the crypts of

the uterine caruncles, and through these lesions it is believed that bacteria may invade the blood stream of either fetus or mother. Beller[10] and other investigators have identified various bacteria within the placental blood vessels and in the umbilical cord of clinically healthy animals. From these tissues the bacteria logically enter the fetal fluids. These fluids are commonly passed over as inactive, static liquids, which from the clinical standpoint is erroneous, especially with the amniotic fluid. This, clinically, is a rapidly circulating liquid, performing an important role in fetal physiology. If one inserts his finger into the mouth of an intrauterine bovine fetus, it at once sucks vigorously. It constantly swallows the amniotic fluid and with it any shed hairs, epithelium or other tissue debris; the solids are filtered out in the alimentary tract, and the filtered liquid returned into the fetal circulation. With the hair and debris there is ample reason for the belief that bacterial invaders also pass into the alimentary tube and are stored as a part of the meconium. The bacteriologist in his search for *Bacillus abortus* in an aborted fetus turns regularly to the stomach as one of the most reliable locations in the body, and provides thereby an interesting analogy between the fetus and the adult, that the most common avenue of invasion is via the alimentary system. There is further clinical evidence of such invasion of the fetal digestive tract in the very frequent and pronounced functional disturbances of the gastro-intestinal system. A large ratio of aborted fetuses show conclusive evidence of such invasion by being smeared over with meconium. This is especially marked in cattle and sheep, and probably is common in goats. Bacteriologists report that in the aborted bovine fetus there is commonly present in the fetal stomach fragments or pellets of meconium: the fetus has had diarrhea, has evacuated the meconium into its amniotic fluid, swallowed it, and started the meconium upon an unending circulation. Here again is a reminder of the pathology of the new-born, unhealthy calf, which evacuates its bowels upon the bedding and then swallows its own excrement. Apparently healthy cows not rarely give birth to calves, at approximately full term, which have suffered prior to birth from profuse diarrhea, so that the calf is covered over with meconium, and the amniotic fluid loaded with meconium. So in sheep abortion, as observed by Carpenter[238] and myself, born lambs were thickly and repulsively covered with semi-fluid meconium. Many ewes refused to suckle such lambs, apparently owing to the repulsive odor, which was foreign to the species in health.

In equine abortion the conditions are reversed and the alimentary

tract is commonly impacted with very hard meconium, calling for special attention in new-born foals to see that the impacted masses are expelled. Exceptionally the bovine fetus is born with impaction of the colon and rectum, instead of with diarrhea. The desiccation occurs in the small intestine, the meconium being molded into spherical pellets, united into a nodular strand by thread-like connections. The strand, when passed into the colon and rectum, is folded and

TABLE XIII

Studies of *Bacillus abortus* infection of heifer calves in a genitally diseased herd. A = Abortion

Number	Age in days when tested	Agglutination	Age in days when bred	Days after last breeding when tested	Agglutination	Duration of pregnancy in days	Result of pregnancy	Remarks
1	130 357	10 —	406	2 25	300 50	218	A	
2	108 335	600 —	401	7 33	1000 600	201	A	
3	137 364	10 10	411 432	4 30	300 300	146	A	
4	199 426	— 25	472	5 31	1000 50	251	A	
5	110 337 390	10 10 —	393	21	50	227	A	
6	118 345 398	— 10 50	397 416	25	50	219	A	
7	211 438	25 —	487 508	3 29	1000 —	217	A	
8	102 329 382 408	— — 50 50	410			209	A	
9	204 431 484	— — 600	495	13	50	216	A	
10	201 428	100 —	461 679	9 35	50 10			Killed Tb. reactor
11	205 432	10 —	458 510 554 593	11 37	— 600			Pregnant 194 days

TABLE XIII (*Continued*)

Number	Age in days when tested	Agglutination	Age in days when bred	Days after last breeding when tested	Agglutination	Duration of pregnancy in days	Result of pregnancy	Remarks
12	98	—						
	325	—						
	378	25	458					
	404	10	501					Pregnant 183 days
13	117	—						
	344	10						
	397	50						
	423	50						
14	80	10						
	307	—						
	360	300						
	386	25						
15	211	25						
	438	—						
	491	—						
	517	10						
16	8	—						
17	62	—						
18	36	—						
19	28	10						
20	62	—						
21	57	—						
	284	—						
22	120	10						
23	13	600						
24	71	10						

compressed into a firm mass. Hence the alimentary canal, to all clinical appearances, serves as a vast reservoir in which the bacterial invaders of the fetus are stored. In most cases these are effectively controlled, possibly through the action of the bile; when such control fails, diarrhea or meconial impaction follows and, if the fetus is born, serious disease may be manifest at birth or may develop immediately afterward.

Research workers are practically unanimous in the belief that

new-born calves taking milk from cows which carry *Bacillus abortus* are promptly invaded via the alimentary tract because the milk contains the organism. Presumably this occurs as well with other bacteria which may have existed in the pregnant uterus. Certainly the young of sheep, goats and other ruminants carrying *Bacillus abortus* likewise ingest the organism with the milk. It is difficult at present to determine by the agglutination test the status of new-born calves and of heifers regarding the presence of *Bacillus abortus* in their organs. The recorded data are not adequate. In the report of the N.Y. State Veterinary College for 1913-1914 I have recorded irregular and incomplete data upon the agglutination of the blood of twenty-four calves born late in 1912 or early in 1913 and followed up to late in 1914. The tests were made in the bacteriological laboratory of the college. From these data I have constructed Table XIII. It shows that the agglutination is highly erratic; that a large ratio of calves in a diseased herd reacted at one time or another; that coitus caused a sudden and spectacular advance in the reaction; that the high reaction continued for about a month in most heifers, after which there is a hiatus until they aborted. Presumably, had the test been frequently applied, the reaction would have been found fairly constant throughout gestation.

The date of invasion in this group is uncertain. Since the cows which produced them were genitally unsound, ample opportunity was present for intra-uterine infection. After birth they had raw herd milk which certainly carried *Bacillus abortus* and other invaders. They suffered much from diarrhea and pneumonia. Table XIII shows quite well that as a group they carried the infection at least from when a few days old until abortion occurred. At the epoch involved most heifers in the herd aborted their first pregnancies, had retained afterbirth or presented other serious pathological phenomena.

As in other features of the invasion of the reproductive system, so in the problem of the persistence of the invaders from fetal, or early post-natal life to breeding age, conflict of opinion arises. The conflict has a bearing upon the question of the immunization of animals against abortion. Clinically the young born of diseased parents, as in Table XIII, and unfavorably grown as in that herd, largely abort, calve prematurely or have retained afterbirth at the close of the first pregnancy. There are, however, herds in which genital disease in the adults is rife while the health of the young may be advanced, as indicated in Table X, by better control of diseases of the new-born. Perhaps the conflict of opinion in this

field is largely due to the failure of the one group to appraise adequately the value of the hygiene of the young.

The date and avenues of invasion of the reproductive system of the adult have not yet been determined to the satisfaction of all. One group has enthusiastically claimed that the artificial introduction of virulent cultures of *Bacillus abortus* into the system of the female, thirty days before breeding, is overcome by the animal and a highly valuable immunity acquired; others stoutly deny this. After several decades of effort, the first group has greatly decreased in numbers and the second has correspondingly increased. It was originally believed by Professor Bang that *Bacillus abortus* was transmitted by the bull at coitus; for some years that belief was largely repudiated, only to be renewed recently, so that it is generally considered inadvisble to use as a sire a carrier of bacteria associated with abortion. The belief generally prevails that the bacteria associated with abortion largely or chiefly invade the system during gestation. Opposed to this is the general view that *Bacillus abortus,* once in the animal body, persists indefinitely. In the dairy cow, ewe or goat, it is believed to acquire a permanent habitat in the mammae, where, through the bacilli-containing milk, it acts as a permanent focus for the spread of the bacteria. The alimentary tract is considered the chief portal of entry in the fetus, the new-born and the adult.

Studies of the fetal membranes of aborted equine and ruminant fetuses indicate with much emphasis that in a large ratio of cases the infection begins at the ovarian poles of the cornua or at the os uteri internum. The "exudate of contagious abortion" is generally focused about the cervical or ovarian poles and spreads thence to involve other parts. Accordingly in the fetal placenta of ruminants, as indicated in Fig. 37 and in Colored Plate II, the evidence strongly suggests that the apices of the cornua were the primary seat of trouble. In other cases it is quite as evident that the focus is at the cervical pole. This does not authentically settle the question. If the bacteria enter the pregnant uterus from the blood stream, the invasion should apparently be diffused throughout the uterus, or occur as frequently at one location as another, unless the cervical and ovarian poles supply the areas of least resistance. In studies of the fetal placentae it is found that, as a rule, lesions referable to prior gestations are most common and extensive in these areas. In multipara, when a fetus dies at near term and becomes emphysematous, it is most often either at the cervical or at the ovarian pole.

The Pathogenicity of Bacteria Associated with Abortion and Allied Phenomena

It has already been definitely stated that in many animal plagues, such as hog cholera, foot-and-mouth disease and tuberculosis, the infection clearly causes abortion. When Harms (against his judgment) was caused to vaccinate one thousand ewes against sheep pox, and four hundred of them promptly aborted, there appeared no reason to deny that the vaccination was the direct cause of the disaster. When bacteria or other invaders are recognized in the uterine cavity of an aborting female, in the placenta, or other fetal organs, it is fair to assume that the identified infection is the chief, or an important contributory cause of the abortion. If the invader found in an aborting female is transferred to another female of the same species, by the same avenue, in equal volume and virulence, and the animal later aborts, the invader in question will in all probability be found in the uterus and in the fetal organs. It need not be the fundamental cause of the abortion. Thus, had Traum and Hart[197] inoculated virulent cultures of *Bacillus abortus* or of vibrios into the series of heifers and cows which they studied and reported as having aborted "without demonstrable etiological factor," they would probably have recovered the bacteria after the abortion. In the ordinary course the introduced bacteria would have been regarded as the direct and sole cause, although it is quite evident that the animals were already on the road to abort. The fact would remain that the artificially introduced bacilli might well have contributed materially to the disaster.

In the realm of infectious diseases, the injury caused by the invader is commonly dependent upon the numbers and virulence of the organisms as compared with the power of resistance of the host. Nowhere is this more spectacular, or fundamentally important in the control of disease than in the infections of the reproductive system. This variation in abortifacient power of a bacillus is perhaps nowhere more emphatically illustrated than as recorded by Moore and Fitch.[146] Two groups of pregnant cattle, of five each, were inoculated with living cultures of *Bacillus abortus*. The cultures for both groups were prepared by Fitch from the same stock and both groups were inoculated by me. One group was regarded as my experiment; the other as that of Moore and Fitch. The inoculations were made two months apart except for one animal in my group. The dose of the culture was 10 cc. in each case. Since my five animals were heifers and those of Moore and Fitch were adult cows, approxi-

mately twice the size of the heifers, my group had about double the volume of culture per unit of body weight. All were in the same pasture and cared for by the same personnel. All five of the Moore-Fitch cows aborted in seven to eighty-five days after inoculation. My five heifers were slaughtered, one at forty days after inoculation, the other four at 120 to 180 days. No traces of *Bacillus abortus* were found in any animal. The utero-chorionic space of one contained "abortion exudate" but no abortion bacilli were identified. It is unusual to find in veterinary literature records of the inoculation of pregnant adult cows as a test for the abortifacient power of *Bacillus abortus*. The cows used by Moore and Fitch came from a herd which was being dispersed owing to the bankruptcy of the owner. I was delegated to examine the purchases for pregnancy. The manager related that abortion had run riot in the herd the previous year and that but one of the five animals selected had a live calf. According to the views of many, they should have been immune, but according to clinical observation they should have largely aborted without additional experimental exposure. My five heifers were purchased from a dealer, looked vigorous and had been on the premises for a sufficient time to be at ease, while the cows had been on the premises for a few days only, when subjected to artificial infection. There is no ground for assuming that the inoculation of the Moore and Fitch cows did not hasten and render more certain the universal abortion; there is no ground for believing that a larger dose of the culture may not have caused the five heifers to abort. If the inoculation of ewes with sheep pox virus by Harms, as already cited above, caused forty per cent of them to abort, then logically, had enough abortion bacilli been injected into the jugulars of the five heifers, abortion or other injury would have followed.

Most researches upon bovine abortion are conducted in regions of concentrated dairying, and efforts to demonstrate the power of *Bacillus abortus* or other bacteria associated with abortion are conducted upon dairy heifers of poor quality and bred when decidedly immature. They are already highly prone to abort, if not from *Bacillus abortus* then from the undemonstrable etiological factor described by Traum and Hart.[197] It is extremely easy to apparently cause abortion in such groups of heifers as those shown in Table I. They were already doomed to abort, and any additional insult, such as changing environment, food and attendant personnel, and adding pathogenic bacteria or bacterial products, logically added to the danger.

No accounts are available of the experimental production of abortion in heifers born of healthy dams; grown naturally without diar-

rhea, adhesive feces, pot belly or rough coat; held out of breeding until well matured; mated with healthy bulls, as indicated by authentic study of stained spermatozoa; and kept under thoroughly hygienic conditions during pregnancy. In all probability if massive doses were repeatedly given the animals would largely abort, but clinical observation indicates that the dose would need be many times as great, far beyond that which they ordinarily encounter in the course of natural exposure. The experimental exposures with living abortion bacilli are usually far beyond any reasonable estimate of the probable natural exposure, and the pathogenic force of the invader thereby advanced.

The evidences regarding the pathogenic power of bacteria or other etiological factors present in the uterine contents of aborting mares are similarly variable. Superficial study tends to compel the conclusion that the results of experimental inoculation are erratic. As a private practitioner from 1879-1891 at Bloomington, Illinois, I had unusual opportunity for the clinical observation of abortion and sterility in mares. The foremost industry of the region was the breeding of heavy draft horses, mostly Percherons. Clients annually imported approximately one thousand heavy draft stallions and mares. Coming chiefly from France, but also from Belgium, Germany, and the British Islands, they brought with them essentially all diseases available in Europe. They were intensely bred and fed. About 1886 abortion appeared in definitely discouraging intensity, and the losses increased year by year until they reached their climax in 1889 and 1890.

Toward the close of the latter year I undertook some investigations,[228] apparently the first in the United States, to test the transmissibility from mare to mare. Doubting the value of mares of the affected locality, I procured from Texas a group of pregnant plains mares, grown wholly out of doors in a semi-wild state. These were shipped nearly one thousand miles by railway, at that time a tedious and extremely rough journey. Had pregnant mares from my area been shipped to Texas instead, it would have been expected that a large portion would have aborted from the journey alone without artificial infection. Ten pregnant mares were exposed, of which two aborted. One of the abortions was attributed to violence in handling. They were essentially wild horses and the handling of them was enlivened by many falls and accidental injuries. The experiments were crude as measured by present methods, but there remain striking evidences that the Texas mares I had chosen were highly refractory to the infection, or supposed infection, I had applied.

In contrast Good and Smith[70] experimentally infected two preg-

nant mares. One died from acute enteritis attributed to the ingestion of the abortion bacilli; the other had a four-degree rise in temperature and aborted in ten days. Good and Smith used mares from the location in which abortion prevailed and hence might well have aborted without experimental infection. Already facing a distinct possibility of aborting, they were moved into new environment, were in all probability far more closely confined than under their former handling, the food was probably changed, and they were handled by a new personnel. The intravenous injection of the cultures, which elevated the temperature four degrees, would logically facilitate abortion enormously, and reasonably assure the invasion of the uterus by the injected bacilli, transferred indirectly from the uterus of a similar mare when aborting. The data available for study are scant, but it appears that as in bovine abortion, so in mares, positive results from experimental infection are reasonably certain to follow when animals are drawn from an area where abortion prevails and are given excessive doses of living cultures. So far as known, the rule applies to all species of bacteria known to invade the pregnant uterus of any species of animal, and to be associated with abortion, still-birth, premature birth, retained afterbirth, or other allied phenomena. If a given bacterium be found in the uterine contents of a sexually diseased female, and it be directly or through cultures transferred to another uterus of an animal of like vulnerability, it will multiply in the organ of the second host essentially as in the first. Those invaders which may be carried to the uterus by the blood stream may enter the vascular system through the digestive, or the genital tract, or may be introduced artificially by subcutaneous or intravenous injection. It is not known that any form of bacterial intra-uterine infection exists which may not be introduced by way of the blood stream.

The Control of Sterility, Abortion and Related Phenomena Attributed to Bacteria or Other Invaders Alleged to Have Specific Pathogenic Powers upon the Reproductive System

The firmly establishel tradition, which still persists, that contagious abortion is a specific infectious disease, or a group of specific, transmissible maladies which override all, or essentially all laws of sexual hygiene, has caused animal husbandmen and veterinarians to almost totally anchor their efforts at control upon artificial bactericides, immunization, segregation and slaughter. Those factors which have been mentioned in the preceding chapter as the primary causes of these phenomena have been ignored or only casually men-

tioned as of minor importance. This perspective has logically led to the greatest riot of alleged remedies, cures and panaceas which have yet appeared for any one disease or group of maladies in veterinary history. In the meantime the ravages of genital diseases have flourished luxuriantly. Along with error there has inevitably evolved much quackery. In no other veterinary field has it proven so difficult to draw an authentic boundary line between excusable error and deliberate charlatanism. Both quackery and error largely have as their basis inadequate facts, and no other disease or group of diseases provides the volume of available data which may so readily mislead. It has been stated above that if a pregnant female be inoculated with bacteria, virus or toxin from an aborting animal, and the experiment animal is vulnerable, owing to faulty environment or handling, she will quite probably abort and it is thereon concluded that the invader employed possesses distinct abortifacient power; if the experiment animal is in ideal health, and has been mated with a perfectly healthy male, it is highly probable that she will give birth to healthy young. Either termination, taken alone, may be very misleading. The chief obstacle barring the way to a safe conclusion is that it is usually difficult, or impossible, to determine from the published report what the result may have been without the experiment. Parallel to such perplexities, an alleged preventive of abortion is administered: if the pregnant female is ideally healthy and destined to produce young physiologically, the abortion repellant is apparently effective; if the pregnant female is foreordained to abort, owing to one or more of the factors discussed in the preceding chapter or elsewhere, she aborts. In the first instance, the conclusion is invited that the alleged remedy is highly valuable; in the second instance it may be concluded that some other invader than that it was sought to destroy wes present and caused the abortion. Thus if the highly commended vaccine of Good against *Bacillus abortivo-equinus* was administered to a mare and abortion followed, the disaster could be attributed to the virus of Dimock and the efficiency of the vaccine go untarnished—no one could disprove the conclusion. It thus becomes easy to submit indisputable facts as evidence of the virtue of an abortion panacea; but careful study reveals the fact that the evidence is incomplete and any conclusion based thereon may prove very misleading. Hence innumerable alleged abortion remedies have been luxuriantly advertised by vendors and have been eagerly purchased and administered until time and experience shall have shown them to be worthless. So the bacterins administered to the heifers in Table I were followed by virtually total collapse of reproductive power, while in other herds their use was followed by reported

excellent results, supplying vendors of these products with excellent advertising material. It would be quite unjustifiable to attempt a discussion of all the alleged panaceas for sterility, abortion and retained afterbirth. A few examples will serve to illustrate.

Tradition has largely dictated the plans for the prevention, cure, and elimination of abortion and sterility. Prior to the advent of bacteriology, abortions in animals were usually divided into two classes—accidental and contagious. Sometimes a third class—enzoötic abortion—was mentioned.

The accidents believed capable of causing abortion were legion, and each pregnant female was unavoidably exposed to many of these, so that if she aborted it was only necessary to recall one of the vast number in order to explain the debacle. Neither cure nor prevention of accidental abortion was possible. When the first symptoms of accidental abortion appeared, the fetus was dead or mortally injured and there could be no reason for delaying its expulsion. Yet some writers advise that the animal showing signs of impending abortion be kept quiet. Apparently this concept of preventing impending abortion has been borrowed from the medical profession. When women exhibit definite clinical signs that all is not well with the pregnant uterus, the obstetrist may sometimes avert disaster. The veterinarian is far more familiar with the clinical evidence that an abortion has occurred than that one is impending. In principle, if the human obstetrist may prevent threatened abortion, the veterinarian should be able to imitate him. This principle seems to be largely set aside because of the radical differences between the placentae of women and those of domestic unipara and of swine. Abortion in domestic carnivora, with deciduous placentae, is of scant interest, and little is known of it. When impending abortion is observed in domestic unipara it is commonly desirable economically that it be completed as early as practicable, in the hope that the uterus may be so repaired that the female may retain some breeding value. Available knowledge suggests that if some fabulous means should be discovered by which threatened abortion might be prevented, it would be undesirable, because the young animal in the case would more probably be a liability than an asset. The human practitioner strives to save a life; the veterinarian is interested only in life worth saving. If the young is to be weakly, of slow growth and of low fertility or infertile—except in such cases as the mule—the veterinarian and animal husbandman are not interested in the preservation of life.

Accident may open a favorable avenue for the invasion of the

uterus by bacteria. Severe injury to the pregnant female certainly lowers the resistance and facilitates the multiplication of bacteria already within the uterus. Thus it is generally held that the transportation of pregnant females over long distances is frequently followed by abortion. The belief in the power of accident to directly cause abortion has declined during the past few decades. The term accidental abortion is now and then used by writers to veil those abortions in which no invader is found which, according to their judgment, can be satisfactorily charged with the injury. The lesions in the ovum and endometrium do not differ from those associated with bacteria or moulds. As the belief in accidental abortion disappeared, the efforts to prevent or control abortion became increasingly focused upon contagion or infection. Since tradition dictated one infectious abortion for the mare and one for the cow, search was begun for the discovery of the offenders. When Bang and Stribolt discovered *Bacillus abortus,* it was almost universally accepted as the cause of all abortions in cows due to contagion. Bang believed, as many others did, that since the invader had been identified, the control of abortion was assured, and that either immunization or quarantine would solve the problem. Others believed that it might be controlled by the use of bactericides.

ENDOCRINES OR HORMONES

Endocrines have been advocated by some technical laboratory workers and have been advertised quite enthusiastically by some vendors. No one denies that they are essential, but it has not yet been made clear why it should become necessary to administer these artificially to an animal properly fed and handled. They may be of value in some forms of genital disease as adjuncts to other remedies, but they cannot permanently cure deficiencies due to errors in hygiene.

VITAMINS

Moussu[147] contends that *Bacillus abortus* can only invade the uterus virulently in the absence of adequate quantities of vitamin E. It is difficult to comprehend why an animal inured to a given food for ages should now require new elements, or increased amounts of old materials in order to be capable of producing young.

MINERALS

Some champion an artificial supply of mineral elements such as phosphate of lime, iodine, and others. Some desert countries produce

vegetation definitely wanting in mineral constituents. This is by no means always due to a deficiency of minerals in the soil, but may be due to deficient rainfall for the proper solution of these elements so that they may be taken up from the soil by the vegetation. In some desert, or semi-desert areas, heavy dews enable vegetation to grow sparingly without the moisture having entered the soil. Du Toit and Bisschop[46] and their co-workers in South Africa have well shown that the grasses of some velds are grossly deficient in phosphates during long-continued drought and that the defect may be overcome experimentally by feeding large quantities of bone meal. It has not been clearly shown that the needed amount of phosphorus can be artificially supplied within economic limits.

In the United States during recent years, large areas have been rendered unfit for animal husbandry because of severe drought. Grass has ceased to grow in adequate quantity, and the quality is inevitably defective. A movement is discussed by which it is hoped to restore fertility to the soil by forestation with increased rainfall.

Other food elements, and methods of feeding have been held responsible for low resistance of the animal economy to injurious invaders, especially to those having distinct peril for the reproductive system. The invasion of the reproductive system, however, is at least as frequent and serious in those animals having an abundance of those foods richest in vitamins and minerals. At the two extremes of food supply occur the highest incidence of destructive genital diseases.

DISINFECTION

Prior to the discovery of *Bacillus abortus,* Brauer[19] presented highly optimistic data indicating that abortion could be effectively controlled by the internal administration of carbolic acid. His studies made a profound impression and he had many followers for some years.

In 1913 Rich[164] reported with great enthusiasm the value of the internal administration of methylene blue. Ten years later he presented a second report in which he admitted that he had quite overestimated the value of the drug.

Horse Dung. Reindl[162] added variety to abortion panacea by submitting data in 1909 apparently showing that piling a liberal quantity of horse manure in a dairy stable, where bovine abortion raged, thus changing the atmosphere from acid to alkaline, eliminated the plague. The evidence submitted was apparently as convincing as that of Brauer or Rich. Much was written at this time concerning the great

value of stable disinfection, regardless of the fact that the process could not disinfect the intestinal or genital tract of the cows.

Other workers in the abortion-sterility field have advocated and continue to advocate the topical disinfection of the reproductive tract. The value of this is generally, if not universally admitted, but its efficiency has been overestimated. It is a general principle that a mucous membrane cannot be disinfected without serious injury which may invite a more virulent invasion. The mechanical washing of the genital tract is certainly of distinct value when properly applied in prudently selected cases. Aside from the occasional injection into the problem of vitamins and minerals, previously discussed, the use of hormones, concerning which speculation is greater than practical knowledge, and disinfection, just mentioned, the efforts to control, prevent or cure infectious abortion, sterility and associated phenomena in domestic animals have revolved almost wholly around the original prophecy of Bang—that the problem would be solved by artificial immunization or by quarantine.

IMMUNIZATION

The discussion of immunization has been thus far limited to those cases of genital disease associated with *Bacillus abortus* in cattle and with *Bacillus abortivo-equinus* of horses. Immunization has not been advocated in the battle against other bacterial invaders of the reproductive system. Although *Bacillus abortus* invades all domestic and some wild animals, immunization has been advocated for the cow only, and chiefly for the dairy cow. The plans for immunization vary infinitely, and new methods are being continually brought forward.

(1) *Immunization with Killed Cultures of Bang's Bacillus, Administered to Pregnant Cows*

Early in the fight against "contagious abortion" bacterins or killed bacteria were highly lauded. Thus Reichel,[161] representing a commercial house engaged in the manufacture and sale of bacterins, presented before the U. S. Live Stock Sanitary Association a highly optimistic series of data following the administration of bacterins. Immediately following Reichel, it fell to my lot to present the data constituting the basis for Table I. The two reports were as diametrically and irreconcilably opposed as is possible. Similar conflicts in observation were innumerable, and bacterins lost favor, so that few clinicians now have faith in them and the federal government has forbidden their inter-state shipment. I do not understand that the inter-state shipment is forbidden because the use of bacterins is

directly dangerous, but rather because of the belief that they are useless and consequently misleading to the public. The debacle in the group of heifers in Table I was not caused, in my opinion, by the administration of bacterins. The heifers were of the Cornell University dairy herd described by Wing.[271] For a number of years the first pregnancy heifers had regularly aborted in a highly destructive ratio. Added to this there occurred, as related by Wing, a virulent outbreak of septic metritis during 1910 and 1911. While Wing does not go into details, I, as veterinarian to the herd at the time, computed the losses at twenty per cent mortality and twenty per cent discarded because of hopeless sterility. In this virulent storm the eighteen heifers of Table I were born. I knew, as well as any one not a prophet could know, that except for the intervention of a miracle, those eighteen heifers would mostly abort. Bacterins were not that miracle.

Abundant data are recorded in veterinary literature showing abortion bacterins as highly efficient against abortion. Most data showing the sinister side of their efficiency do not appear in print.

(2) *The Vaccination of Non-pregnant Cows and Heifers with Virulent Strains of Bang's Bacillus*

The vaccination of *pregnant* cattle with virulent cultures of abortion bacilli was advocated by but few. The philosophy of it was directly antagonistic to the generally accepted theory that the administration of virulent cultures would quite certainly cause, not prevent abortion. There were interesting exceptions. Thus Bland[15] in his extensive and famous experiments, administered the virulent cultures to as many as eight cows or heifers which he assumed to be vacant but proved pregnant, and much to his astonishment all *calved correctly,* which was a far better record than that of the non-pregnant vaccinated animals.

Living vaccines prepared from virulent strains of Bang's bacillus and administered to cows and non-pregnant heifers of breeding age were extensively advocated by some research workers. These optimistic reports were luxuriantly advertised by the vendors of abortion cures and were much used over a period of several decades. Highly favorable reports were submitted, consisting largely of conclusions rather than facts. Dairymen and non-critical practitioners were largely misled. I purchased samples of these on the open market from most of the advertisers in the United States, and a colleague in the bacteriological department reported to me that almost all the

"living" samples were "dead." Few alleged remedies for any animal disease have been so enthusiastically advertised and exploited. Vaccines have now been advocated for about forty years, approximately as long as the Israelites were in the wilderness, and many veterinarians still believe that the promised land is near. The plan of vaccination has varied greatly.

The diseases attributed to *Bacillus abortus* and other bacteria associated with abortion, sterility and retained afterbirth are of the distinctly chronic type, and in this group no outstanding victories have yet been made known from artificial immunization. Before vaccination against abortion and allied phenomena can well be finally accepted, clinical proof of irrefutable character needs be submitted. One of the most difficult barriers to overcome is the absence of a well marked immunity after an attack of the disease. There is a fable, less frequently recited now than some decades ago, that once a cow has aborted she is thereafter immune. It would be bad indeed for the dairy industry if no cow which has once aborted or had retained afterbirth might not thereafter produce a live calf. Clinical observation shows clearly that when cows are studied in groups, those which have aborted, have been temporarily sterile, have calved prematurely, have dropped non-viable, weak calves or have had retained afterbirth are far more likely to abort their next pregnancy than are those cows which have not previously aborted and, after a pregnancy of about 279-288 days, have calved physiologically without retained afterbirth.

The literature in the vaccination field is ponderous and vague. Amongst the earlier reports which carried great influence were those of Bland[15] who submits data upon six hundred vaccinated cows and 416 controls. On the surface his data are highly favorable, showing an abortion incidence of 4.8 per cent in the vaccinates and 19.7 per cent among the controls. He fails to indicate the basis of the selections of vaccinates and controls. He does show that when the numbers of aborted, barren, died, or sold are massed together in each group, the failures to produce calves in the vaccinated group totalled 21.3 per cent and, in the controls, 24.7 per cent. Among the 1021 cows listed by Bland none were reported as having calved prematurely, expelled a dead fetus at term, dropped a non-viable calf or had retained afterbirth. It is now freely admitted that vaccination with virulent living cultures causes in numerous cases permanent infection. If this be true it may well be inferred that the injection of virulent bacteria sixty days prior to breeding might well destroy the

life of the fertilized ovum soon after conception, thereby establishing what we call sterility.

Lubbehusen, Fitch, and Boyd[125] report studies upon the use of virulent living vaccines, during a period of between three and four years, in a dairy herd of sixty females. While the data are incomplete in some important respects, they afford exceptional opportunity for critical analysis. The herd consisted of four dairy breeds, which indicates that it is the property of an agricultural college or experiment station and that expert advice is available in dairy husbandry, genetics, animal nutrition, and clinical and bacteriological veterinary service. The herd does not suffer from scarcity of funds for first-class quality of animals, ideal housing conditions, and abundant food of highest quality. In 1918, 72 per cent of the herd was positive to the agglutination test for *Bacillus abortus*. For five years prior to the researches, the incidence of abortion had been 11.3 to 21 per cent. Twenty-seven females, comprising members of all four breeds, were vaccinated, and thirty-three kept as controls. The females of each breed in both groups were served by one bull of the corresponding breed, and all were handled in common except for the vaccination. The ages of the animals, and their history prior to the experiment are not recorded.

By their computation there had been forty-two terminated pregnancies in the vaccinated group, of which thirty-four were reported as normal calvings and eight were designated abortions. A careful study of their chart shows that four of these pregnancies had already terminated before the vaccination of the cows, three of which calved and one aborted. One cow "calved normally" and the "calf" was dead. Two of the cows which had terminated one pregnancy prior to vaccination failed to become pregnant after treatment. Excluding the four pregnancies terminated prior to vaccination, there remain thirty-eight terminated pregnancies following vaccination, thirty of which resulted in living calves and eight in abortion or in calves dead at birth. This is 23.7 per cent of abortions and stillbirths—2.7 per cent greater than the highest abortion rate prior to vaccination. Among the thirty-three controls, sixty-seven pregnancy terminations are charted with an abortion ratio of 28.4 per cent.

The authors state that the vaccinates required 2.55 services per pregnancy, compared with 2.56 for the controls. Their method of computation is not clear. Their chart shows 165 services, of which seven were for pregnancies which had terminated prior to vaccination. This leaves 158 services for 51 pregnancies, or 3.1 services per

pregnancy. As nearly as may be learned, the services to cows which were discarded subsequent to the termination of the last pregnancy were not counted. Thus in one case a cow was discarded after ten fruitless copulations, and the ten services were apparently not counted; another cow had twelve services, the last of which was successful, and the twelve were counted. Had she been discarded after the eleventh service, the eleven would apparently have been excluded from the computation.

Excluding the four pregnancies which had terminated prior to vaccination, the twenty-seven vaccinates produced thirty live calves, or 1.1 per cow during a period of approximately 2.5 years. Since the recorded duration of gestation bespeaks weak calves and a consequent high mortality, amongst the thirty living calves one could not rationally expect as many as twelve fertile heifers to replace the twelve cows which died or were discarded.

The authors fail to record the ages of the animals, the age at which heifers were first bred, or rest interval after the termination of a pregnancy until the cow was rebred. They do say that heifers were vaccinated sixty days before breeding and that the cows were vaccinated as soon as uterine involution had occurred. The inference is justified that vaccination occurred at about ten days post partum and that service was permitted sixty days later, thus allowing a minimum rest of seventy days. The vaccination thus served as a barrier against very early post partum breeding. This barrier was absent in the control group, in which the chart reveals no less than five cows became pregnant at less than sixty days post partum, of which 40 per cent aborted and 60 per cent had retained afterbirth, as was perfectly natural. Sterility logically followed. Numerous other controls were repeatedly bred before conception in a manner to suggest that they too had been bred at less than sixty days post partum, the fertilized ova had perished unseen, and the phenomenon had been classed as temporary sterility. This early rebreeding of the controls placed them at a distinct disadvantage compared with the vaccinates.

Two of the thirty-three controls were transferred to the vaccinates. Of the thirty-one remaining animals, five—or 16.1 per cent—died, were killed or sold, compared with 44.4 per cent of the vaccinates. Two vaccinates did not become pregnant after vaccination. Three were in twin pregnancy (the only twin pregnancies in either group) in the first and only gestation after vaccination. These five animals cost the vaccinated group 18.5 per cent of its total; the corresponding loss for the controls was 3.33 per cent.

The authors do not comment upon the three twin gestations, which gave a ratio of 7:100 for the vaccinates and none for the controls. The three pregnancies cost thirteen services, all by the bull of the breed represented by the 300s. It is not stated that any of the twins were viable or whether they were of the female, male or no sex.

It has been stated in Chapter III that the duration of gestation constitutes a highly valuable basis for computing breeding efficiency. If the ideal duration is placed at 279-288 days inclusive, then it is found that among the vaccinates 54.8 per cent fall within the optimum, as compared with 66.7 per cent among the controls. In the two groups together, those pregnancies falling within the optimum were accompanied by 10.2 per cent of retained afterbirth; those above 288 days 20 per cent; and those below 279 days 29.2 per cent.

In their bacteriological search of the abortions, the authors were uncertain as to the cause in two of the vaccinates and three of the controls. Satisfactory diagnoses were made in six vaccinates, of which 83.3 per cent were attributed to *B. abortus* and 16.7 per cent to other causes which were unknown. Among the sixteen abortions in the controls in which they were positive that *B. abortus* was or was not the cause, 68.7 per cent were declared due to that organism and the cause of 31.3 per cent undetermined. The ratio of the abortions definitely due to the abortion organism was much higher in the vaccinated group than among the controls—the abortion organism was present in a higher ratio of the abortions in those animals in which additional bacilli had been added to those already carried by the animals.

The vaccinates produced during the researches 1.1 live calves per cow, while the controls produced 1.4—a superiority of 36.4 per cent. The authors attribute this inequality to the fact that more of the vaccinates were sold, but fail to indicate why they were sold or why the authors selected for vaccination the inferior animals doomed to be sold. They were not sold because of overcrowding, since it is clear that new animals were being bought in order to maintain the herd. The reproductive efficiency of the entire herd was very low. They state that the Jersey cow, 101, apparently the best breeding cow in the herd, produced twelve healthy calves although positive to the agglutination test for abortion disease. Assuming that the Jerseys are indicated by the 100s in the charts, the next Jersey is 132, leaving a gap of 31 missing Jerseys. The authors leave this group an unsolved mystery. While they state that cow 101 produced twelve calves, they fail to indicate their destiny. Her three charted calvings

show that less than one year elapsed between each two. After she had displayed a distress signal by having calved at 274 days, she was rebred thirty-seven days later. She was wrecked, as was logically to be expected, had retained afterbirth and became hopelessly sterile. The authors were greatly disturbed over the debacle, bred her ten times without success, and were industriously doctoring her in search of a retroactive panacea which would go back and cure the wreck caused by sexual overloading.

Upon the above evidence the authors conclude: "Although some degree of immunity toward *Bac. abortus* (Bang) is induced by the use of the living vaccine, it does not reduce the abortion rate to a desirable minimum and should never be used in other than badly infected herds. . . . We have been unable to demonstrate that vaccination retards conception nor that increased sterility is directly associated with the use of living vaccine"!

Kitselman[108] studied the use of living vaccines in a herd of fifty beef cows and heifers. Ten heifers negative to the agglutination test before vaccination were all positive at the close of the researches one year after vaccination: two proved sterile, two aborted, five calved and one remained pregnant. Ten cows with young calves were 60 per cent negative before vaccination and 20 per cent after one year. Sixty per cent of these were sterile. Thirty cows had aborted or failed to calve during the year preceding vaccination, six of which tested negative. At the end of the year one was negative. Twenty per cent aborted and a like number were sterile.

Hart and Carpenter[82] pursued studies in vaccination with living vaccines in a group of dairy heifers, with less favorable results than those of Lubbehusen, Fitch, and Boyd, and in a form which did not permit so complete an analysis. Their report was criticized by me at some length.[242] The examples cited are believed fairly representative of the literature upon the use of living abortion vaccines of the virulent type in cattle.

(3) *Calfhood Vaccination with Avirulent Bacillus abortus Cultures*

The vaccination of calves with avirulent strains of abortion bacteria has recently stimulated the hope that a method has been discovered by which a highly valuable immunity may be attained against abortion in cattle. The reports measure the results by the incidence of abortion, not by the reproductive efficiency. The plan is as yet too young to determine the longevity of the vaccinated animals. The basic health of the vaccinated calves is not clearly stated and the

method of selecting controls is not generally clear. Numerous research workers have published data which upon the surface read well, amongst which the report by Cotton[35] is considered representative. In general the reports are optimistic, but not notably more so than the carbolic acid cure of Brauer, the methylene blue remedy of Rich, or the horse dung panacea of Rindl, noted above. Its greatest merit, if it has value, should be in such thoroughly saturated herds as those illustrated in Table I, or in the herd studied by Lubbehusen, Fitch and Boyd, above mentioned. The calves in these herds acquired the bacilli in utero, obtained them abundantly in their milk, and were in association with diseased animals constantly. The question then arises regarding the value of administering avirulent bacteria to animals already carrying virulent forms—has an avirulent strain the power to destroy a firmly established virulent infection? It appears logical to believe that if avirulent abortion vaccines administered to calves at four to eight months would effectively prevent abortion in them as adults, the invasion of the fetus and of the new-born calf by virulent abortion bacteria should give it valuable powers of resistance, especially in those cases, said to be in the majority, in which the blood test for contagious abortion becomes negative at six to twelve months of age. Not all calves positive to the agglutination test become negative. Thus the calves included in Table XIII were infected early, as indicated by the agglutination test, and remained infected until they aborted their first calves. Hilton[91] says, upon this plan of vaccination: "Approximately 15 per cent of infected calves remain permanently infected." Udall[205] used avirulent cultures on forty-eight heifers and heifer calves, followed by excellent breeding efficiency. The herd was kept essentially free from abortion infection according to the agglutination test, the abortion rate in the herd was low before the vaccination. Udall is one of the foremost advocates of the growing of healthy calves. It might well be suspected that calves born of healthy cows and grown under healthful conditions would largely remain sexually sound after vaccination with avirulent organisms. It is therefore difficult to measure the effects of the vaccination. If the cultures are actually avirulent, they can admittedly do no harm.

The reports upon calfhood vaccination are inadequate in the number of animals, the period of time covered, the basic state of health and vigor of the calves used, the age at which they were bred, the health of the sire with which they were mated and many other vital factors influencing reproduction.

This is well illustrated by the report of Stevens[186] who submits his

findings from the calvings of the first year after vaccination. Upon hasty reading, a highly optimistic view may be attained, while upon closer study it is revealed that the report consists essentially of conclusions without facts. The title indicates that the animals vaccinated were calves, but he fails to state what he means by calves. Elsewhere in his contribution he says that some of the animals vaccinated were pregnant as much as six months. Like Bland[15] he was somewhat surprised that these pregnant animals should have calved properly after vaccination during pregnancy, which some say *causes* abortion instead of preventing it. Stevens states "There have been over 200 of these vaccinated heifers calve in the last six months and from the reports of the dairymen and my own observation they have with the exception of three head all produced healthy, full time calves." He does not state how many females vaccinated during the same unstated period failed to become recognizably pregnant, fails to say what he means by "full time calves" or whether the "healthy" calves lived beyond 24 hours. While he says that over 200 pregnancies had terminated, he apparently did not know how many more. There may have been 230 or 280. He fails to advise his readers of what occurred to the three females which did not produce healthy calves at full term.

In some of the herds he asserts that prior to vaccination 50 to 100 per cent of the first pregnancy heifers regularly aborted; he does not say that he vaccinated any heifers from such herds. He fails to state whether the vaccinated heifers had been sired by, or bred to healthy or diseased bulls. Throughout his article he fails to indicate that any attention whatever was paid to the health of any bull.

Stevens' report seems fairly representative of the vagueness and incompleteness of the researches and observations in this field. Caution should be exercised by the reader and all the reports critically studied. Before arriving at important conclusions and definitely committing himself by extensive application of calfhood vaccination, the practitioner would do well to first apply it experimentally in a herd where all significant factors are known, and adequate controls are maintained.

Calfhood vaccination need not be summarily condemned. In studying the problem it should be remembered that so far as yet known, a heifer calf sired by a healthy bull, born of a healthy cow after 285 days of pregnancy, without dystocia, retained afterbirth, calf scours or other allied pathological phenomena, properly fed and handled, and bred when not less than 24 months of age to a perfectly

healthy bull, will calve physiologically at about 285 days whether vaccinated or not vaccinated at any period of her life.

For those abortions in cattle in which *Bacillus abortus* can not be held responsible, no immunization or other prophylactic has been seriously proposed. Thus Lubbehusen, Fitch, and Boyd, as stated above, decided that 16.7 per cent of their abortions were *not* due to *Bacillus abortus*, and, having failed to discover the cause, suggest no remedy. The occurrence of these abortions from unknown cause leaves undetermined the influence which this cause may have exerted in those abortions in which *B. abortus* was identified. Hart and Traum[84] after the elimination of contagious abortion from the Univ. of California herd report the persistence of a 10 per cent ratio of abortion, and the prevalence of sterility and retained afterbirth in the absence of *Bacillus abortus* or other, to them, recognizable cause. In another Bang-negative herd studied, 33.3 per cent of primipara aborted from an unrecognized cause. In one highly prized purebred herd devoted to the breeding of valuable sires, and kept abortion-free according to the test, the abortion rate runs as high as 50 per cent from unidentified causes, and obviously this can be neither controlled nor ameliorated by vaccination with *Bacillus abortus*. In those cattle abortions associated with vibrios, streptococci and other known infections, no immunization has been urged. When *Bacillus abortus* is established in the mammary gland, and, while the cow is breeding well, the bacillus is being excreted in the milk, artificial immunization has not yet been definitely advocated. Artificial immunization has not been prominently advised against *Bacillus abortus* infection in sheep, goats, or swine in which it is common.

(4) *Vaccination in Equine Abortion*

Vaccination has been highly lauded for the control of equine abortion associated with *Bacillus abortivo-equinus* or paratyphoid bacillus. As among cattle, so in the mare, it was at first believed that *all* infectious abortions were due to one invader only, and it was fondly hoped and firmly believed that infectious abortion could be eliminated through immunization. Good[71] announced in 1902 that he had discovered a highly successful vaccine which had proven reliable after four years of trial in the famed Blue Grass region of Kentucky, the greatest center of thoroughbred horse breeding and of equine genital disease in the United States. Four years later the colleagues of Good in the University of Kentucky, Dimock and Caslick,[41] state: "In more recent years, the number of mares which have failed to breed, the number of stallions that have proved to be impotent or shy

breeders and the diseases of the new-born, *not to exclude outbreaks of contagious abortion,* have all been very notably on the increase."

In 1932 Dimock and Edwards[42] re-assert the efficiency of the vaccination against equine abortion associated with the paratyphus bacillus. The bacillus, however, was no longer regarded as the cause of all abortions linked with infection. Instead 64 per cent of abortions were negative to *Bacillus abortivo-equinus;* 26.5 per cent were due to streptococci; 5.8 per cent to *Bacillus abortivo-equinus* and 3.6 per cent scattering. In one stud of 32 mares negative to *Bacillus abortivo-equinus,* 24 aborted and no micro-organism could be identified, leading them to suspect a virus abortion. Thus while they had found a highly efficient immunizing agent against the original contagious abortion, a new, and more disastrous streptococcic abortion had shown itself and a third yet more virulent contagious abortion due to a virus was suspected. In 1936 Dimock and Edwards[43] definitely declared the existence in Kentucky of a highly destructive virus abortion of mares. The belief of Dimock and Edwards in a virus abortion has recently been supported by Miessner and Harms who observed seventeen abortions in a stud of twenty-three mares, in which all efforts at the discovery of bacteria which might be incriminated, failed. The subject will be further considered under virus infections of the reproductive system.

Dimock and Edwards reasserted their faith in the efficacy of artificial immunization against the original equine infectious abortion and advised its annual use upon brood mares in foal. The sole preventive measure they could offer against the streptococcic abortion was the mating only of sexually healthy animals—in an area where about fifty out of each one hundred mares bred one year, drop a viable foal the next. Just how easy it is in such a situation to mate sexually healthy animals they do not state. They offer no means for the prevention, cure or amelioration of the virus abortion. They thus claim that one of the three chief types of equine infectious abortion may be controlled by vaccination, while the other two, each of which is apparently quite as destructive as the paratyphus bacillus, are left uncontrolled and uncontrollable. Several factors have served to make the famed Blue Grass region of Kentucky a great center of equine reproductive diseases. It is the most concentrated area of thoroughbred horse breeding in North America. Sentiment regarding a famous stallion or mare largely takes precedence over hygiene, and disease runs riot. Judging from the available literature and from two seasons of personal study in the area, the control of sterility, abortion, and diseases of foals in the Blue Grass region demands other means for

their effective control than vaccination. On page 129 I have related that in 1926 I had reported studies indicating that abortion in mares was almost three times as frequent in those bred and conceiving on the 9th day postpartum as in those conceiving at a later date. The statement elicited no immediate comment. Fifteen years later, in 1941, Jennings[101] presented far more voluminous data substantiating my findings, and placing the abortion rate even higher than I had done. He did not state whether these abortions following 9th day conceptions were due to paratyphoid bacillus, streptococcus or virus. Rather he attributed it to the 9th day conception, which calls for prudence instead of vaccination. Neither will vaccination, vitamins nor hormones prevent disaster when a mare is bred to a stallion with spermatozoa like those shown in Fig. 28.

QUARANTINE AND SLAUGHTER

Quarantine in veterinary medicine usually ends in slaughter. Prolonged or permanent quarantine within or between herds is well nigh impossible to maintain effectively, and the cost is generally prohibitive. The basis for successful quarantine is the isolation of the diseased individual in a manner to eliminate all danger of the transfer of the contagion to healthy animals. It is usually, if not always, safer and more economical to slaughter the affected animal. The difficulties of permanent quarantine are well illustrated in the case of human leprosy, which has been so handled for some forty centuries without demonstrated success. At the other extreme, Canada and the United States, acting as a single people for the North American continent, have succeeded conspicuously in barring such animal plagues as foot-and-mouth disease, bovine contagious pleuropneumonia and some other disastrous maladies of domestic animals. Foot-and-mouth disease has rarely surmounted the barriers against it and penetrated the United States, but in such cases local quarantine has been established for a brief period until the diseased and exposed animals have been slaughtered. The markedly chronic type of essentially all distinctively genital infections of domestic animals, offers virtually insuperable barriers against successful control by quarantine within or between herds for the control of bacterial genital diseases. Quarantine has not been advocated prominently except in the *Bacillus abortus* infection of cattle, although that infection invades all species of domestic, and an unknown number of wild animals. Various types of quarantine have been tried.

In the United States, intra-herd quarantine was for a time frequently attempted, and continues to be applied in a lesser degree.

The effective isolation of diseased animals within the herd is very difficult of application. Ordinarily the animals cannot well be placed far enough apart for safety, and separate personnel is very costly and, in the main, ineffective. Since all domestic animals carry the bacillus, it becomes extremely difficult to insure against contamination of the free animals indirectly through other species. In many areas wild ruminants exist. As already noted the American bison and the wapiti or "elk" of the West are known to be carriers of *Bacillus abortus*. It is quite probable that the various species of deer, friendly free-booters who ignore quarantine restrictions, also carry the infection. Intra-herd quarantine is at best of questionable efficiency and as in vaccination may only be claimed to exert a favorable influence in those cases of sterility, abortion and retained afterbirth in which *Bacillus abortus* may be incriminated. Thus in the researches of Lubbehusen, Fitch, and Boyd, already discussed, less than 60 per cent of the abortions could be definitely ascribed to *Bacillus abortus*, while in 22.2 per cent no bacteria were identified. Quarantine could only affect those cases authentically attributed to the abortion bacillus.

I have described elsewhere[256] a group of twins and free martins, emanating from three Bang-free, purebred herds in which genital diseases were severe. One unusually highly bred herd had an abortion rate of fifty per cent. There was no basis for segregation of diseased from healthy because the conclusion was inevitable that any apparently healthy individual carried the same factor as those which aborted. When a cow aborted, she could be segregated and the fetus incinerated, after associated animals had already been exposed. Then isolation could merely diminish, not eliminate, the exposure of the uninfected animals, if such existed.

There is one outstanding example in which intra-herd segregation or quarantine becomes highly important but is frequently overlooked. Especially in dairy herds where hand breeding is applied, and two or more sires are kept, as well as under parallel conditions in the breeding of horses and beef cattle, the strict maintenance of the breeding unit is of great importance. Thus in the large dairy herd owned by an agricultural college, it is shown in Table V that the pregnancies of Bull 7, in which living calves were produced, had an average duration of 8.5 days less than those of Bull 3. All the progeny of Bull 7 regularly became positive to the agglutination test for Bang's disease; all the get of Bull 3 remained negative. The progeny of the two sires ran together as a common herd. The natural inference is that in some way the sires were responsible for the differences

in the abortion test. It seems unjustified to suspect that Bang's bacillus was transmitted as a permanent infection from sire to offspring. The explanation apparently lies in the gross variation in the duration of the pregnancies of the two sires.

All data I have thus far been able to assemble emphatically indicate that the optimum duration of pregnancy in the cow is approximately 285 days, and that when the duration drops below 279, or exceeds 288 days, there is a marked decline in the fertility of the progeny upon arrival at breeding age, parallel to the degree of departure from the optimum duration of their life in the uterus. Further evidence upon this inference appears in Table II, in which Bull 2 suffered from abscess of the seminal vesicle, as shown in Fig. 138. The genital disease of the bull was vividly reflected in the abbreviated gestation period of those of his pregnancies which survived to birth.

There is no occasion to assume that Bang's bacillus or other given species of invader was directly transmitted by the sire to his offspring; it is shown clearly enough that the sire was defective and all available data indicate clearly that young animals born outside the optimum duration of intrauterine life are feeble and possess inadequate resistance to bacterial invaders. Since Bang's bacillus is the most abundant invader of the genital system of the dairy cow, it would logically be the most frequent invader in the heifers born after an abbreviated pregnancy. It accordingly becomes highly important in animal husbandry to vigorously maintain the breeding unit to the end that the efficiency of the sire may be more exactly determined and measures taken to discard the defective or dangerous.

It is an all too common practice to break the boundary lines of breeding units owing to the failure of one or many females to become pregnant by the assigned sire, and breed them to another male. The importance of the intact breeding unit may well be illustrated by the contribution of Cameron, Fincher, and Gilman.[23] An agricultural college had imported from a distant state a fashionably bred bull and assigned to him a breeding unit of cows. Some of these, failing to become recognizably pregnant after several services, were transferred to another sire of the same breed in the herd. The imported bull was assumed to be healthy and had met the requirements of the State of New York that he should be free from tuberculosis and contagious abortion. After the second sire had served sterile cows from the breeding unit of the imported bull, his cows too began to fail to conceive and it was discovered that the new bull had

brought in the trichomonad disease. The breaking down of the breeding unit had multiplied the disaster.

Such perils constantly confront the custom of community, or association sires unless adequately guarded. If the females of one member of the association have a communicable genital disease, the other females of the association are at once in peril; if a member of the association errs in the purchase of a new female, all females within the unit become exposed.

The maximum of peril is attained in concentrated centers of the breeding of thoroughbred horses where usually no breeding units are accurately fixed. Famous sires are not usually limited in their services to the stud of the owner, but the use of the stallion is offered, in a chivalrous gesture, at spectacular fees to other owners of mares. If the stallion has won great races, or his get have proven great winners, his services are in corresponding demand. He is then overbred, becomes less fertile and his mares return more and more. With advancing age and fame, and decreasing capacity, his fertility drops lower and lower. Mares conceive, the fertilized ovum dies early and vanishes unseen, leaving the uterus more or less seriously damaged. The next year the owner of the mare may feel discouraged and transfer her to another famous sire. The owner of the new sire may demand a bacteriological examination of the mare before acceptance, which she may pass. Thus sterile mares, often made sterile by the stallions, are shifted to other stallions, of perhaps low fertility, and each may expose the other to a new infection. Numerous stallion owners quarantine against barren or aborting mares and require a bacteriological examination, but admit to service without question, those mares having a foal at side. Under this ruling the mare whose afterbirth is shown in Fig. 30, and whose foal was born at 387 days and was unable to stand for several days, would be accepted for service by the stallion, although profoundly diseased. Though a large ratio of the thoroughbred mares in the Blue Grass area of Kentucky according to my observations, are clinically diseased on the ninth day post partum, yet they are bred, and the stallion owner who would refuse a mare which had failed and had a year of sexual rest, accepts a diseased mare with a nine-day foal at her side by some other sire.

On the other side, the owner of the mare has no authentic information usually regarding the fertility of the sire, and hence has no certain method of exclusion in case of low fertility and perilous disease. Some stallion owners have examinations made by a veterinarian of their choosing, but the condemnation of a stallion of low fertility

whose owner can command service fees of $1,000 to $2,000 is not often publicly announced.

The establishment of a permanent harem of females for each sire is one of the most vital hygienic laws in reproduction. A sire may die or break down and some disposition needs be made of his females. Unless the field is perfectly clear they should on no account be dispersed to carry peril widely. Instead in many cases, such as related of the trichomonad disaster, the females should be held intact in an isolated group until safety is assured.

Inter-herd, inter-state and exhibition quarantine is now common in cattle regarding *Bacillus abortus* infection. It is well known that some cattle are negative to the agglutination test and yet harbor *Bacillus abortus,* while others are positive and the diagnosis cannot be confirmed by bacteriological search. The incidence of these is unknown. It is generally held that such defects are negligible in number. Sometimes cattle to be shipped long distances are negative at point of shipment, and positive at destination; the variation, at least in some cases, is apparently due to intensification of low infection. Most fairs in the United States now require a negative agglutination test for exhibition of cattle. Sometimes the owner finds his cattle positive upon his return and various hypotheses are advanced to explain.

The inter-state quarantine of cattle against contagious abortion is at present generally imposed. It is not without peril. Reference has already been made to the imported bull mentioned by Cameron, Fincher, and Gilman in connection with the trichomonad disaster. The buyer accepted the bull as genitally healthy because he was negative to the agglutination test. No one warranted the bull as healthy in such a manner as to effectively protect the buyer, and his assumption that being negative to the agglutination test made the sire safe sexually was largely his own interpretation. Or in the highly pedigreed herd referred to above, negative to the agglutination test with fifty per cent abortions, the breeder would logically become a buyer of heifers and seller of bulls. His bulls, being negative, were salable into the best of herds and incapable of being good sires. Hence the buyer needs beware and constantly bear in mind that the agglutination test provides uncertain security only against that one infection.

Slaughter of the infected animals is the logical termination of the usual types of quarantine in the field of genital diseases in domestic animals. As early as 1921 the Bureau of Animal Industry of the Pennsylvania Department of Agriculture had promoted officially

a plan, which came to be known as the *Pennsylvania plan,* involving repeated agglutination tests of all animals in a herd, with the segregation or discard of all cattle positive to the test. The plan permitted the retention under strict isolation of valuable dairy cows positive to the agglutination test. Quarantined cows are usually neglected cows, are assigned the poorer stables, caretakers and food. Except in unusually valuable animals with convenient environment for effective isolation, the owner prefers to discard the positive animals. After a herd had passed several tests satisfactorily to the state, they were officially declared free from contagious abortion. The Pennsylvania plan was modified and revised from time to time and more recently adopted in its chief features by the United States Bureau of Animal Industry in a nation-wide campaign, in cooperation with the states, for the eradication of abortion.

The Pennsylvania Bureau of Animal Industry has published statistics following the application of the plan, which in themselves appear favorable. The herds from which statistics were published showed that abortion had decreased. The decrease in abortion is quite natural when all cows positive to the agglutination test are discarded, unless new cases develop. After seventeen years of application in the state of Pennsylvania, no authentic evidence is available that there is less abortion, sterility, and retained afterbirth among dairy cattle in that, than in the adjoining states. Neither is there available a clear accounting of the expense involved.

The plan of test-and-slaughter is stated to be voluntary upon the part of the owner. Technically this is quite true, but practically it is highly misleading. Associated with this alleged voluntary character of the plan is the fact that the breeder of purebred cattle of high quality, for sale as breeders, is compelled to have the test applied for interstate shipment, for entering for show at most fairs, or for entrance into public sales. So far as he is concerned, the plan is sharply dictatorial.

Certain difficulties stand out clearly. Turning again to the Cornell University dairy herd described by Wing[271] covering forty years, from 1889-1928 inclusive, it is to be recalled that one of the herd, Glista Ernestine, attained fourth highest place of history in the lifetime production of milk and butter fat. At the zenith of her career in 1915 her blood was positive at 1:500. My records show that in all thirty-five of the cows in the herd at that time there were but two negative reactions. Those two were not heavy milk producers, the fat content was very low and they had only bull calves or heifers which never appeared in the breeding lists. Slaughter of the herd,

except the two negatives, would clearly have blotted out contagious abortions and the herd.

White, Rettger, Johnson, and McAlpine[225] after discussing the economics of abortion disease and finding differences between reactors and non-reactors, state significantly regarding one cow: "This cow, although positive, milked through the eleven years, the entire period. . . . Her influence was great enough to make the reacting group of Ayrshires more profitable than the non-reacting group. This cow did not abort at any time." Elsewhere it is made clear that this cow bore nine calves, the greatest number produced by any one animal among the eighty-three cows, of four breeds, in the herd under observation.

In the herd studied by Lubbehusen, Fitch, and Boyd, already used as a basis of illustration, the one Jersey cow, 101 in their chart, positive to the agglutination test, was an outstanding individual, having borne twelve calves. There is a significant hiatus between her and the next of her breed; the next number is 132. Apparently thirty-one cows or heifers younger than she had died or been discarded because of inefficiency.

The actual ratio of outstanding cows which are positive to the agglutination test for *Bacillus abortus* is unknown. It is not known that the individual instances constitute exceptions. Cows of low vigor are logically more vulnerable to invasion by abortion bacteria, tubercle bacilli, vibrios, streptococci or trichomonads. The problem is not always confined to individuals but may affect an entire herd. An instructive example is furnished by the 1927 report of the Dominion of Canada Experimental Farm at Aggassiz, B.C. There is listed a group of eleven cows which had been reactors for years, throughout the application of the test, and had averaged 5.5 viable calves each; a group of nineteen younger cows constantly negative in which one pregnancy had terminated, with an average of 2.5 calves; and a third group of nine heifers of breeding age, all negative, of which four were pregnant and five apparently hopelessly sterile. All animals were kept in a common stable and grazed as a common herd. The elimination of the eleven reactors would have bankrupted the herd. Being a government herd it could have been wiped out without recognizable loss to any single individual. The taxpayers of Canada, however, would have been losers. So, in the present cooperative plan between the federal and state governments in the United States, while the dairyman may not lose overmuch in the elimination of reactors, the citizens pay for the cattle. The cost of the plan to the public, and its ultimate effect upon breeding diseases, remain to be determined.

Cows which are of low fertility, or are sterile, cannot be profitable in the dairy, nor can their milk be generally desirable or safe. No agglutination test need be applied to them; they should be discarded by the dairyman as one of the losses pertaining to the industry. The slaughter of such cows at public expense is only justifiable when ample evidence is presented to show that the public is responsible for the disease, or at least that the dairyman is the victim of forces beyond his control and is entitled to government relief. In the latter case the dairyman is equally entitled to relief when the losses are due to vibrionic, streptococcic, or trichomonad infection. Again referring to the researches of Lubbehusen, Fitch, and Boyd[125] in connection with their cow 101, when they rebred her thirty-seven days post partum, after she had raised a signal of distress by calving at 274 days, the question arises whether the government should reimburse a dairyman for ruining an outstanding individual by placing a load upon a twelve-year-old cow which a six-year-old could not safely bear. There is the other side. Eminent professors of genetics and dairy husbandry recommend such breeding; they are in the employ of the state for the express purpose of teaching dairymen how to breed and feed dairy cattle. Viewing the problem from such angle the conclusion may be reached by some that the state is responsible to its dairymen for such disasters.

Private cattle owners do not publish authentic or comprehensive data upon the breeding efficiency of their herds. Neither do the forty-eight agricultural colleges and experiment stations in the United States, maintained jointly by the federal and state governments. One of their most important functions is that of researches in the reproduction of domestic animals. These institutions are admirably equipped and have the services of the foremost animal husbandmen and veterinarians of the nation. No complete report upon the reproduction of any species of domestic animals is available. There are numerous reports featuring a single phenomenon, like the milk yield, or the ratio of abortion over a brief period. The most instructive report, in many respects, is that of Wing[271] covering the Cornell University herd of Holstein-Friesians for a period of forty years. There remained in the herd in 1928, fifty-four females, all of which disappeared without a trace shortly thereafter. The public, which might well claim ownership of the cattle, is not advised what became of them.

Recently the agricultural college of the University of Illinois issued a flaming circular "$5,000,000 reward to Illinois cattle breeders for stamping out the costly Bang's disease." When details were requested

it became apparent that the reward was a figure of speech. The agricultural college herd is presumably free of the infection but no accurate statement is available of its breeding efficiency.

Elsewhere Thorp and Graham[196] of the veterinary staff of the agricultural college, and presumably the authors of the five million dollar circular, relate that a highly valuable herd of cattle under their professional guidance, had been officially accredited as Bang's-disease free for some years, when *Bacillus abortus* infection broke out and wiped the herd out completely within two years. All rules for maintaining the herd free from the infection had been rigidly enforced and the most intensive search possible failed to so much as faintly suggest the source of the infection. Thus Thorp and Graham in one publication, spectacularly urge cattle owners to free their herds from the disease; in another confess their total inability to keep the herd free after the diseased animals have been destroyed. There are numerous herds like that reported by Thorp and Graham; and there are many herds which are officially accredited as Bang's-disease free in which sterility, abortion and retained afterbirth cause appalling losses, but these are not usually reported fully and frankly.

A news release by the United States Department of Agriculture dated September 15, 1936 states that in 2801 herds tested, containing 72,488 cattle, 26.7 per cent reacted at the first test. In the fourth test 2.7 per cent were positive. Assuming a gradual decline during the four tests, approximately 45 per cent of the cattle reacted and were destroyed. The fourth test showed that one-fourth of the herds remained affected. Various explanations were suggested for the persistence of the infections in these herds, such as the negligence of the owner or the virulence of the infection. The latter reason is perhaps the more vital, and warrants the inference that the eradication of the disease is most difficult, or impossible in those herds where its elimination is most urgent.

The advocates of the test-and-slaughter plan for the control of Bang's disease sometimes claim results for which they submit no evidence and no citation of data supporting the claims. Elder[49] as already cited on page 141, states that Bang-free herds produce upon the average one calf for each 11.5 months, whereas Bang-positive herds commonly produce one calf for each 20 months—a gain of 42.5%. The same statement was made in 1940 by Mohler, Wight and Lash[145] without submitting any supporting data, although there were at that time, according to their report, some 32,000,000 cattle in the United States said to be free from Bang's disease. From this vast number they cite no herd in which each female was producing one healthy calf every 11.5 months.

Mohler, Wight and Lash submit no facts based upon their personal knowledge of the results of the test-and-slaughter plan. Instead they present the conclusions of numerous dairymen and cattle breeders much in the form commonly used in testimonials by vendors of panaceas for disease. None of the reporters is identified and no material facts are submitted by any one of them. For example one of the breeders cited is made to say that prior to having adopted the test-and-slaughter plan he lost 90 per cent of his calves from Bang's disease, whereas during the year immediately prior to his communication he "had not lost a single calf through Bang's disease." He fails to divulge whether his original herd consisted of 10 cows, 9 of which aborted and were destroyed at public expense, and fails to state the behavior of the tenth cow except that she did not lose her calf from Bang's disease. Others state that by test-and-slaughter they freed their herds from abortion, sterility, dystocia, udder troubles, etc.

The political benefits are sharply emphasized, stressing the obvious facts that in many areas Bang-positive cattle can not be sold for breeding or dairying, can not be shown at fairs to win prizes which would be valuable advertisements, and in some communities they could not be used for dairying for the sale of raw milk.

Mohler, Wight and Lash state in one place that "experience has proved that infection usually occurs as a result of the cattle taking the causative organism into their systems on feed or in drinking water." Later with their inferential endorsement they cite a testimonial reading "All cows served and settled by this bull aborted premature calves."

Mohler, Wight and Lash admit that "in a small percentage of cases much trouble is encountered in freeing herds of Bang's disease." They are silent regarding the ratio of such herds and the causes of the failures. Neither do they comment regarding the frequence of such herds as that described by Thorp and Graham[196] in which a valuable Bang-negative herd was completely wiped out in a brief period while under the most rigid expert care against the introduction of the infection from without. Neither do they explain how test-and-slaughter for Bang's disease may prevent abortion from vibrios, streptococci, trichomonads, etc., etc. They do not attempt to explain how cows bred to bulls ejaculating such spermatozoa as those illustrated in Figures 126, 127, and 130, or having such seminal vesicles as those shown in Figures 137 and 138, might be prevented from aborting by the test-and-slaughter method.

Their colleagues in the United States Department of Agriculture, Miller and Graves submit incomplete data in which they state that while the Bang-positives and negatives were kept as a common

herd, the breeding efficiency was 86.06 and 86.56% respectively. Later the two groups were segregated and the conceptions were 86.57 and 86.14% respectively. After the positives and negatives were separated it is indicated that 16.40% of the negatives aborted or "gave birth to dead calves," as compared with 30.08% in the positive herd. Data are given regarding 59 of the abortions and born dead in the postive group, 33 of which had been transferred from the negatives. These shifted animals more than accounted for the higher ratio of abortions in the positive group. It is not shown whether these 33 shifted animals aborted because they were Bang-positive, or became Bang-positive because they were en route to abort. In contrast, among 16 Bang-negative abortions, 3 of the fetuses yielded bacteria other than Bang's bacillus, and 13 were bacteria-negative. No cause for these abortions was discovered and no evidence was submitted to indicate that the 59 abortions above mentioned in the infected group were not also caused by the undiscoverable factor, despite the presence of the abortion bacillus.

Miller and Graves classify the failures to produce living calves either as *abortions* or as *calves born dead*. They speak of the aborted objects as *fetuses* but quite fail to indicate at what date a fetus becomes a calf. They state that they classify "mummified fetuses" regardless of age or size, as *calves born dead*, without suggesting how desiccation may convert a fetus into a calf. They further state "The increase in percentage of calves born dead can be partly explained by the fact that a number of heifers in the *abortion-negative* group had genital tracts which were too small to deliver live calves." They do not suggest that the dwarfed birth canal was due to the absence of abortion infection or venture other explanation.

Miller and Graves authentically show that the Bang-negative group required an average of 2.83 services per conception. Estimating the heat cycle at 21 days, and that the cycles would be regular, there would follow a delay of 38.43 days in conception. In order to produce a calf each 11.5 months, after a pregnancy of 285 days, it is essential that the cow conceive at 70 days postpartum, which after the delay of 38.43 days in conception would require that she be bred 31.57 days postpartum. Most cows are not in estrum so early, unless the corpus luteum of the prior pregnancy be surgically dislodged.

The evidence submitted by Miller and Graves, fellow-workers in the same department with Mohler, Wight and Lash, does not show that abortion-negative cows produce one healthy calf each 11.5 months. The slaughter of cows which have aborted, had metritis or retained afterbirth, calved at less than 275 days, or dropped twins

eliminates all possibility of their aborting later, but before some of the very luxuriant claims that Bang-free cows breed ideally, or better, simply because negative to the abortion test, are accepted, it is prudent to study the actual facts over an adequate period of time.

Interferences with reproduction are very virulent in large dairy herds closely confined and subjected to high pressure for the production of market milk. These constitute a valuable test for the efficiency of any method of control. In such herds it is openly confessed that in some of them at least the test-and-slaughter method is not financially practicable. Thus Mills[141] in the employ of one of the leading milk-producing corporations in America, deplores the great financial burden which he feels is not permanently bearable and can see no material relief ahead from the persistence of the infection while being handled by present methods.

It needs be frankly admitted that the test-and-slaughter method not infrequently involves the total annihilation of the herd; in numerous herds where a considerable ratio of the animals react, the infection persists and heavy discard losses follow each test; once the herd is supposedly free, there is no assurance at present that the freedom may be continued; after *Bacillus abortus* or other specified infection has been eliminated, other invaders of unknown numbers may continue to cause unendurable losses.

It has not been aimed in the preceding pages to either deny or to minimize the importance of *Bacillus abortus* (Bang), of *Bacillus abortivo-equinus* (Good) or of any of the innumerable bacteria known to invade the reproductive system of either sex or any species. An effort is made to submit evidence that the group of invaders discussed are secondary to needless transgressions of the established laws of physiological reproduction, and to arouse the interest of the reader in the value of adjusting breeding and handling methods in harmony with these age-old laws.

Efforts to control contagious abortion and associated phenomena in domestic animals have consisted largely of the identification of bacterial invaders and devising means by which it is hoped to eliminate or prevent their injuries through disinfection, artificial immunization, quarantine and slaughter. The artificial immunization plan demands for its success a separate vaccine or other agent for each species of the innumerable invaders. This leaves the breeder utterly helpless in all those cases wherein no bacterium, protozoon or virus is recognizable. These are significantly abundant, and up

to the present those who believe infection to be the primary, if not sole cause of all those abortions in which microorganisms are identified, have failed to offer any tangible explanation for any of those cases in which invaders are not present. So long as it is held that destructively numerous abortions occur in which no tangible cause is present, the conclusion must inevitably follow that the fetus which is so defective in function and structure that it may succumb without bacterial invasion, will probably follow the general laws of nature and be invaded in most instances by bacteria or other destructive forces. In such case the defective ovum becomes undeniably the primary, and the invading bacteria the secondary cause of the disaster. Hence the chief aim in this treatise is the identification of the transgressions of hygienic laws and the discussion of any promising measures for their avoidance, thereby enabling the parental reproductive system and the intrauterine young to successfully repel the invader. The measures employed need be such that they shall not defeat the justifiable aims of the breeder, and especially the dairyman, who suffers the greatest losses from this group of diseases. The field for study is interminable and only a part of the important facts have been fairly well determined. Progress calls for continual study by the animal husbandman in cooperation with his attending veterinarian in an effort to determine the causes of interferences with physiological reproduction in the herd, stable or individual. Only when this is determined may the causes be economically eliminated. Some of the apparently most important of these methods elsewhere discussed, may be assembled here as examples.

1. Mark for future use as breeders only those newborn animals which are the progeny of healthy parents, have been carried in utero for the optimum duration of time, and have been born without dystocia, retained afterbirth, metritis, etc. The optimum duration of pregnancy in the cow should be placed at 285 days and the selection of future breeding stock limited quite closely to a duration between 279 and 288 days. For horses the optimum is at about 330 days and there is reasonable safety from 320 to 340 days.

2. The newborn should be so handled that its health shall be assured, in order that it may enjoy uninterrupted development. Otherwise there is serious danger of hypoplasia of the reproductive system. It is especially important that the artificially fed dairy calf be intelligently handled, protecting it against calf scours, pneumonia, navel ill, and chronic malnutrition as revealed by sticky feces, pot belly and rough, dry coat. It should also be wholly, or virtually

protected against preputial and vulvar catarrh, as illustrated in Figures 31-34.

3. The young animal should be held out of breeding until amply matured and ample nutritive reserves assembled, beyond its individual needs, for the begetting and sustenance of physiological young.

4. Mate only individuals of ideal general and sexual health. Retain no female for breeding after she is known to have suffered from serious genital disease. Especially do not attempt to breed females known to have suffered such uterine injuries as those shown in Figures 93, 97, 171 and 182. Employ only vigorous, mature sires which ejaculate abundant, active spermatozoa like those shown in *a* of Figure 128—avoid emphatically males discharging sperms like those in Figure 28; use males producing spermatozoa like those on the left in Figure 127—not the male supplying sperms like those on the right.

5. Provide an ample rest period between pregnancies, especially in the dairy cow, bearing the dual sexual load of pregnancy and abnormal lactation. If in the prior pregnancy there has been abortion, a duration of pregnancy falling significantly below 279 days, difficult calving, retained afterbirth, etc., increase the pregnancy interval distinctly.

6. Carefully avoid overloading the sire.

When the avoidance of these, and any other transgressions of the laws of reproduction established through eons of time fail to assure satisfactory breeding results, vaccination, quarantine, slaughter and other alleged remedies may be considered.

Chapter XXII

MOLDS ASSOCIATED WITH INTERFERENCES WITH REPRODUCTION

MOLDS have occupied a minor place in the veterinary literature concerning the diseases of the genital organs. There is no adequate ground for believing them common. Apparently they have been somewhat overlooked because of the emphasis placed upon *Bacillus abortus*. Most reports of the association of molds with abortion have emanated from cases in which bacteria could not be implicated and the investigator has gone beyond in an effort to identify the cause. Jungheer[102] has recently recorded four cases, three from one herd, in which bacteria could not be incriminated and a mold was definitely identified. Jungheer cites numerous other reports of a similar character. Several species of fungi have been identified, such as *Mucor rhizopodiformis, Aspergillus fumigatus,* etc. The placental and other lesions are not macroscopically differentiable from those associated with the various bacteria. There is no means, aside from microbiological study, by which a diagnosis may be made, and no definite method of handling is proposed. Jungheer suggests the trial of iodine. The infection appears to be chronic and obstinate and the prognosis is apparently unfavorable. As in bacterial diseases of the ovum and uterus, so in molds, when serious lesions occur the endometrium can never be restored to ideal health. The female may again breed, in some cases satisfactorily, but the disease leaves a permanent mark upon the endometrium.

Chapter XXIII

PROTOZOAN DISEASES INTERFERING WITH REPRODUCTION

PROTOZOAN diseases are largely tropical. Since they usually pursue an acute course, their immediate peril to the life of the adult attracts the chief attention. Only two known protozoan diseases of the reproductive system have attracted definite attention.

Dourine (Beschälseuche, Maladie du Coit)

Dourine affects horses and asses, and presumably other solidungulates. Traditionally the disease has been known to the Arabs for centuries. It appears to be indigenous in parts of Africa and Asia, and in modern times has become widely disseminated in Asia and Continental Europe. It was definitely described by Ammon, the stud master at the Prussian Trakehnen stud, in 1796, following which date it has repeatedly caused important losses in the major European countries.

During the final quarter of the past century, when the Western states were being settled with great rapidity, and industrial development in the Eastern United States was proceeding with energy, the demand for horse power gave an epochal impetus to the breeding of work horses. Many thousands of draft animals were imported annually for breeding purposes, largely from France and Great Britain. Dourine broke out in DeWitt County, Illinois, so far as could be reliably determined, in 1885. No publicity was given the outbreak until 1886, when I was asked by the owners of some of the diseased animals to investigate the matter as a private practitioner. A diagnosis of dourine was at once made, the disease was reported to the Illinois State Board of Livestock Commissioners, and control measures advocated. The state authorities failed to act and the disease continued to spread. The area was actively engaged in the growing of horses for sale and there was an extensive traffic in horses with the rapidly settling Northwestern states. In 1887 I again reported the disease to the state authorities and strongly urged immediate action. I was then made officially responsible for the handling of the outbreak, and so far as known no diseased animals thereafter left the infected area. Prior to the official recognition of the disease, some animals, chiefly mares, had been sold and had gone to unknown destinations. Later dourine appeared in Iowa, Nebraska, South Dakota,

Montana, and across the international boundary in Canada. While the source of these outbreaks was in no case definitely traced, they were possibly due to some of the animals sold from the infested Illinois area prior to official recognition. The first of these outbreaks to be recorded was that at Gordon, Nebraska, reported by Faville[52] of the United States Bureau of Animal Industry in 1892.

A prominent figure in the outbreak was a stallion to which most of the diseased mares studied by Faville had been bred. The stallion had been bred, according to Faville, by a Mr. Dunham, a nationally known breeder at Wayne, Illinois, 125 miles from the Illinois outbreak, where dourine had been wholly unknown. He was sold into Wisconsin at two years old, presumably without having served mares. He was in the stud for five seasons in Wisconsin, where dourine has not been known. He was then shipped to Gordon, Nebraska, and placed in service early in 1891. All went well, according to Faville, until late spring, when he was permitted to serve two mares which were brought in from Wyoming. At the time of service they were evidently diseased and showing definite genital discharge. Both of these mares died soon afterward. After having served these two diseased mares, some of the mares later bred to him broke down with dourine. Twenty-one years later, in 1913, Mohler, Eichhorn, and Buck,[143] after recounting the outbreak in Illinois and its eradication in 1888, state: "but not before an affected stallion had been shipped to Gordon, Nebraska, thereby starting up a new center of infection in that locality." They evidently had in mind the stallion described by Faville, which, according to that official, was not shipped from Illinois to Nebraska, but from a dourine-free area in Illinois to Wisconsin, where he was in service for five years, causing no dourine, and then shipped to Nebraska where he contracted the disease.

Dourine stands alone, as the only known venereal disease due to trypanosomes. The parasite, *Trypanosoma equiperdum*, was discovered by Rouget in 1896. Natural transmission is by coitus only. Rarely it has been reported that geldings and mules have contracted the disease accidentally. It is not always clear, however, that the gelding or mule may not have copulated. Artificially the parasite may be transferred to ruminants, carnivora and the small laboratory animals.

Dourine is not so highly transmissible as many of the bacterial and virus diseases. If the individual be suffering only mildly, the incidence of transmission may drop as low as ten per cent; when the disease is very virulent the animal is often unable to copulate.

SYMPTOMS

The symptoms of dourine are extremely variable. In typical cases definitely specific symptoms appear in the external sexual organs within eight to fourteen days. In some cases, according to numerous observers, local manifestations of the disease are delayed for weeks

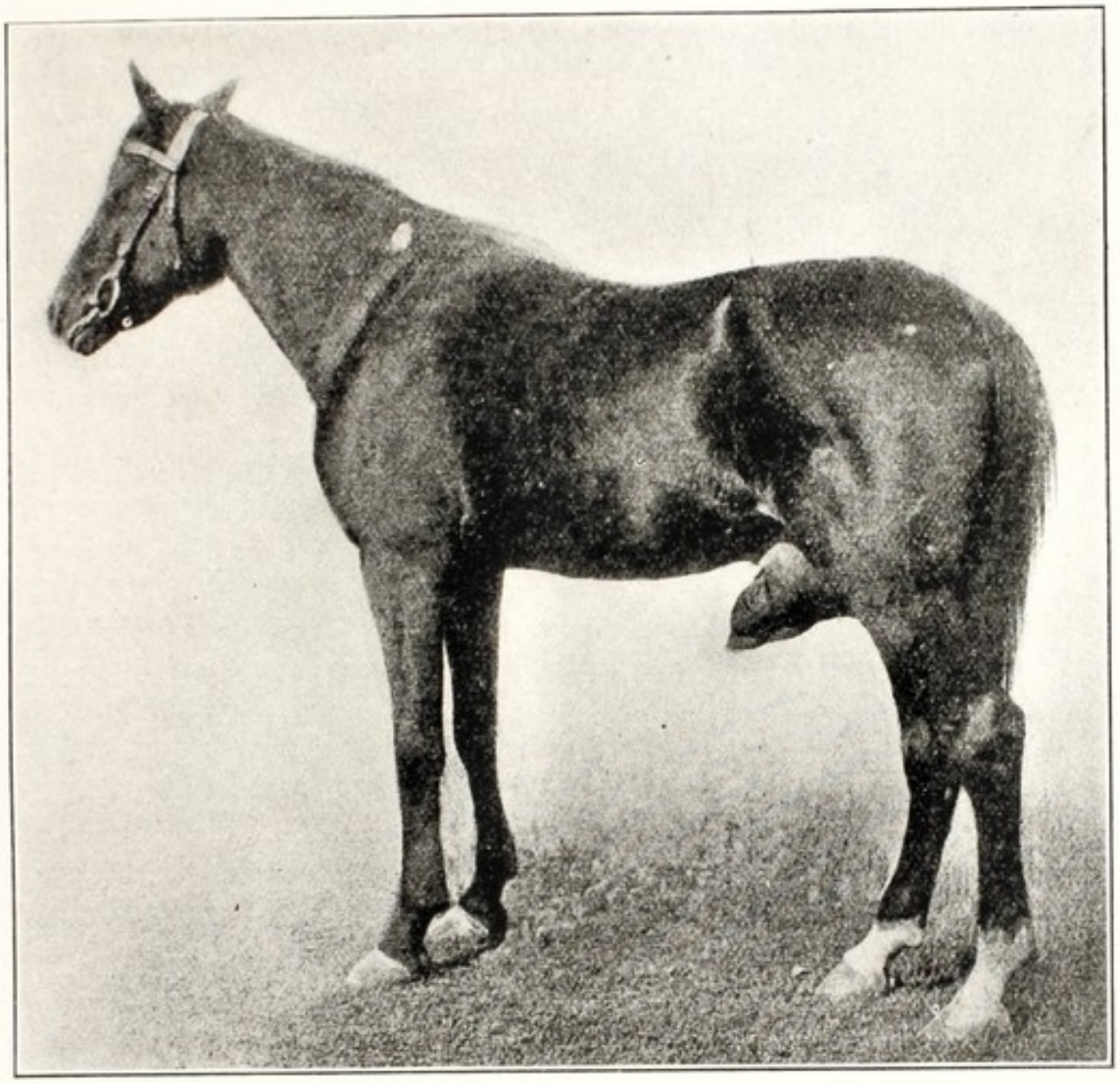


FIG. 98. Stallion in Early Stages of Dourine, Showing Edematous Sheath and Prepuce. (Baldrey)

or months although the animal may transmit the disease by coitus, while revealing no clinical signs.

The first symptom in the stallion is usually an edematous swelling of the sheath, scrotum, prepuce, and penis. As indicated in Fig 98 the penis protrudes from the sheath within its prepuce. The swelling may vary greatly in size and is virtually free from heat and pain. It is normally quite regular in outline. The swelling remains comparatively constant, with some tendency to increase. After some

weeks in many cases a specific leucoderma appears on the penis and may involve the prepuce, sheath, and scrotum. Sometimes leucodermic areas appear elsewhere on the body. The leucoderma is unlike other forms of depigmentation known to veterinary science. It is not preceded by vesicles, papules, ulcers, or other macroscopically visible cutaneous or mucous lesions.

The depigmentation is identical in character with that of the clitoris and vulva of the mare as shown in Fig. 99. The leucoderma usually begins upon the penis and gradually spreads to contiguous parts as an opaque-white veil, the margins being irregularly convex. Sometimes a few isolated patches of leucoderma appear near the border of the area, separated from it by a strip of fully pigmented



FIG. 99. Dourine in Mare Showing Typical Leucoderma of Vulva. (Foster in *Am. Vet. Review*)

skin. Later the pigment may disappear from the isolating strip and the two coalesce. No accounts have been found of histological study of the depigmented areas.

In their extensive monograph "Albinism in Man," Pearson, Nettleship, and Usher[155] describe and illustrate profusely the phenomenon of leucoderma in man. As in dourine of horses, so in man, as defined by Pearson, Nettleship, and Usher, there are no antecedent vesicles, papules, ulcers, or any recognizable type of dermatitis; the depigmentation begins unheralded and slowly expands, the margins being convex. As is the rule in equine dourine, so in human leucoderma, the focus of the leucodermic development is not infrequently the nipple, scrotum, penis, or vulva. The human phenomenon shows a considerable tendency to be symmetrical upon the two halves of the body, but in some cases is unilateral, and in yet others it is irregular. It is common in the tropics or in the subtropical zone and is most preva-

lent in Negroes and other dark races. The depigmentation so corresponds with the distribution of certain nerve trunks that numerous writers attribute it to a neurosis. There are interesting elements of resemblance in the leucoderma of man and in the dourine of horses. They differ radically in the fact that no antecedent infectious disease can be incriminated in man, while veterinarians can authentically attribute the depigmentation to the invasion of the system by *Trypanosoma equiperdum*.

The penis, as indicated in Fig. 98, is somewhat paralyzed early in the course of the malady. The paralysis usually fails to advance notably until the advent of the second stage of the disease, with marked disturbances of the cerebro-spinal nervous system. At this stage the penis may show any degree of paralysis. One of the Percheron stallions in the Illinois outbreak, after he had become greatly emaciated and showed well marked paresis, when caused to trot, swayed from side to side. His completely paralyzed and edematous penis dangled at full length from sheath and prepuce, so that when he swayed to and fro the penis swung back and forth, the glans striking against the middle of his flank.

Numerous observers record that for a time the sexual desire in the stallion is increased, and that he may continue to be quite amorous even after he is unable to copulate, because of either penial or posterior paralysis. Early in the course of the disease, and continuing for a considerable period of time, there is frequently seen, if the glans penis is examined, swelling of the mucosa of the urethra and a dirty thin discharge. As the disease advances the sheath and scrotum may swell to large proportions, the tissues become indurated, and abscesses of varying size form in the non-resistant tissues. These abscesses have no specific significance, being merely an expression of the low vitality of the parts which invites invasion by pyogenic organisms.

The early symptoms in the mare parallel in principle those observed in the stallion. The first symptom is usually an edematous swelling of the lips of the vulva. It is generally stated that there is simultaneous swelling of the clitoris. That is perhaps somewhat magnified. The edema of the labiae of the vulva causes vulvar paralysis and the sphincter apparatus fails to function properly, causing the lips of the vulva to stand apart at the lower commissure and expose the clitoris fully to view. While there is unquestionably some swelling of the clitoris, the other factor plays a highly important role in the exposure of the clitoris, as seen in Fig. 99. There is a variable discharge of dirty muco-pus from the vulva. The vulvo-

vaginal mucosa is usually swollen and irritable. The irritated state of the vulvo-vaginal mucosa leads to frequent urination with evidences of pain expressed by switching the tail, stamping the feet, and rapid opening and closing of the vulvar lips. It is generally held that the mare exhibits increased sexual desire—a sort of nymphomania. It is easy for one to err at this point because frequent urination, switching the tail and rapid opening and closing of the vulva may express sexual desire or urinary irritation and the one be mistaken for the other. Some mares express anger by urination, switching the tail and opening and closing the vulva. This group of phenomena is at times difficult to evaluate.

In a few weeks leucoderma begins. In dark-skinned mares the clitoris is generally pigmented. The depigmentation probably most often begins at the clitoris; it is usually first observed at the *rima pudendi,* at the line of demarcation between the skin of the vulvar lips and the mucosa. Thence it spreads outwards in all directions. Dorsally it often extends to, and encircles the anus. Ventrally it may extend along the perineum and may involve the mammae. If recovery sets in the area retreats from the margins. The last part to regain its pigment is the clitoris. The vulvar labiae are slow to resume their physiological form and function and the pigmentless clitoris, somewhat enlarged, and at times showing masses of smegma crusts about it, is fully exposed to view between the lips of the vulva and its lower commissure. It gives to the vulva of a young mare somewhat the appearance of the organ of an extremely aged, senile animal.

Beyond the local stages of the disease, when the systemic symptoms appear, the two sexes present the same symptoms, except that they may be aggravated in the stallion by frequent coitus or in the mare owing to pregnancy. Some writers observe that the course of the disease is more malignant in the stallion than in the mare. If the keeper of the stallion ignores the presence of the disease and causes him to continue to serve mares, the symptoms become intensified in harmony with a universal law that coitus aggravates a venereal disease. If taken out of service, he is probably confined and his resistance to the disease lowered. In the Illinois outbreak I saw no pregnant mares. It is by no means certain that no conceptions occurred; presumably there were conceptions, but the ova perished early and disappeared unseen. Watson[220] and other observers have studied pregnant mares affected with dourine. It is claimed that some have dropped healthy foals; others have aborted. When abortion occurs, as in abortion due to other infections, metritis and vaginal discharge follow in varying degree.

One of the outstanding symptoms of dourine is the formation of urticaria-like cutaneous swellings of varying size. They are circular or oblong elevations which may be as great as four to six inches across and may stand up above the general level of the skin for one-fourth to one-half inch. The borders of these plaques or "talerflecke" are abrupt, like urticaria or the sting of a bee. The larger plaques may have a depressed center. They may appear almost anywhere on the body, most commonly upon the sides, the buttocks, and the neck. They appear unheralded and have usually reached their maximum size when first observed. They consist principally of lymph or edematous liquid and when first formed contain many trypanosomes. The plaques, in their early stage, afford the most readily available source for obtaining the parasites from the living animal. The plaques persist for days or weeks and may then disappear almost as abruptly as they formed. The hair over the plaques stands erect and matted. The plaques are highly significant. While most forms of urticaria cause discomfort to the patient in burning or itching, those of dourine cause no irritation expressed by rubbing or biting the parts. They are not completely pathognomonic of dourine, but similar eruptions are described in other trypanosomiases, such as nagana, surra, and mal de cadera. In many countries dourine is the only trypanosomic disease known, and in such the plaques are characteristic of the one malady.

Emaciation is remarkably rapid in severe cases of dourine, despite an abundance of nutritious food. This symptom is also shared by the other important trypanosomiases known. Outside of this group of diseases, the emaciation observed in dourine, while the animal is feeding abundantly, is virtually unknown.

Inflammation of the eye is a somewhat common lesion in small laboratory animals inoculated with *Trypanosoma equiperdum*. The aqueous humor becomes clouded and contains the parasites. Such ophthalmia is rarely, if at all, observed in the horse.

There are usually present, in severe cases of dourine, various paralyses. The most common is the paralysis of the posterior part of the body. The horse drags the hind foot along the ground, or strikes the toe at the middle of the stride and knuckles over as a consequence of defective flexion of the tarsus. In other cases some have observed symptoms simulating stringhalt. When the animal is standing it shows nervous instability by repeatedly lifting a foot. Sometimes the obturator nerve is involved, and a straddling gait is present, while in other cases interfering occurs. Some investigators describe as the cause of these paralyses the presence within the nerve sheath of a yellowish

exudate. The spinal cord is reported as being seriously involved in fatal cases. The anterior limbs partake of the paresis in some cases. The motor nerve of the face may be paralyzed on one or both sides, thus drawing the lips to one side or causing both upper and lower lip to hang limp. Sometimes one ear is paralyzed and droops; in other cases the recurrent laryngeal nerve is involved, and roaring follows. These paralyses may appear suddenly, persist for an indefinite period and abruptly disappear.

The paralysis tends to grow apace in the severe cases and finally to render the animal unable to get up or stand when helped to its feet. Yet in some cases after several days of decubitis the diseased animal improves and is again able to get up and lie down. Most cases which go down die within a few days. Upon autopsy lesions are not found which account for the death.

DIAGNOSIS

The diagnosis of dourine presents several peculiar aspects. It has been greatly complicated in veterinary literature by including in its symptomatology another venereal disease of horses which, so far as may be determined at present, bears not the slightest relationship; other diseases due to trypanosomes present group symptoms, forms of paralyses and serological reactions which are very confusing; and the symptoms of the disease itself are quite erratic. There are other compensating features which largely offset these handicaps.

The earlier writers quite generally describe as a single disease two wholly distinct venereal infections: one, dourine, due to a trypanosome; the other the vesicular venereal disease considered due to a virus.

As an example, Hertwig[87] in 1842 contributed an excellent description of the two diseases as a single malady. Five years later, after a careful clinical study of the malady, Hertwig[88] in a second contribution, revised his views and regarded them as two distinct diseases. Throughout veterinary literature, a large proportion of the writers, while admitting the existence of the two wholly distinct maladies, continue to add to the symptoms of dourine those of the virus disease, and then describe the latter malady separately by taking from their description of dourine the symptoms of the genital pox. So Zwick and Fischer[280] in their extensive researches report no vesicular eruptions upon the genitalia of the naturally infected horses studied nor in horses or other species of animals artificially infected; apparently influenced by the writings of others, they mention vesicles as one of the symptoms of dourine. Watson[229] reports no vesicles in

the dourine outbreak in Canada, in which almost two thousand animals were destroyed. The Canadian outbreak was not definitely traced, but it probably slipped across the international boundary from the Northwestern states. Mohler[142] working in the outbreak on the other side of the border declares that vesicular eruptions upon the genital organs constitute specific symptoms of dourine, and holds that therefore dourine and genital pox cannot be clinically differentiated. He fails to relate having seen vesicular eruptions among the approximately two thousand animals destroyed; he artificially inoculated horses and mules and records no vesicular eruptions in them; he illustrates his contribution profusely and shows no vesicles or depigmented spots which could logically be attributed to antecedent vesicles. His colleague in the bureau, Faville,[52] working in the Nebraska outbreak, reports quite fully upon the symptoms observed and records no vesicular eruptions.

In the Illinois outbreak there were no vesicular eruptions. At that time I did not have access to extensive literature. Just beyond the confines of the infected area I examined a stallion and some of his mares which showed abundant vesicular eruptions upon the external genitalia, which from the literature at hand I believed, at first, to be dourine. They all quickly recovered and remained well, in sharp contrast to the animals with dourine less than ten miles distant.

Aside from the alleged occurrence of vesicles of specific character in dourine, no record is found of vesicular eruptions in diseases due to trypanosomes or other protozoa in domestic animals. So far as is known there is no barrier to the simultaneous presence in the same animal of dourine and genital pox. Since the latter is a free-booter infection which bobs up from unknown sources, no antecedent clinical cases are required: it simply arises unheralded when the environment, the details of which are unknown, favor its appearance.

When the leucoderma of the external genitals, as illustrated in Fig. 99, is present, with the gaping vulva and the prominent clitoris, the animal has dourine. These symptoms are not present in all cases. When the plaques or "talerflecke" are present they are highly characteristic. It may be necessary in some cases to exclude the possible stings of bees or some other very rare factor which may produce similar lesions. It is essential also that other trypanosomiases, like nagana and surra, be excluded. The paralysis and rapid emaciation are highly significant. There are individual cases of dourine which may defy clinical diagnosis, but such solitary cases rarely come up for diagnosis. In the history of dourine the primary diagnosis is a group identification; an outbreak of dourine is present and its identi-

fication is not difficult. After the outbreak has been diagnosed, the recognition of infected individuals has as its foundation the fact of coital exposure. As observed in the Illinois outbreak, dourine is safely diagnosable by clinical study. When the outbreak occurs upon the open range among semi-wild horses, where stallions and mares run together, the outlook differs.

The difficulty, or impossibility, of the accurate clinical diagnosis of dourine in solitary cases has not yet been overcome. No plan is known by which the horse breeders of a dourine-free area may effectively exclude a mild case of the disease. The control of the malady is far more effective within the infected area itself, where all diseased or exposed animals may be destroyed or held under rigid quarantine. Thus in the Illinois outbreak other areas were far more efficiently protected because of the measures taken within the infected area, than would have been possible by distant or adjacent communities. The diseased and exposed animals were identified and adequate precautions taken to render them safe.

The diagnosis of dourine by microbiological means is more difficult and less conclusive than in many other diseases. The trypanosomes are not readily discovered in some cases. Investigators report that as a rule the parasite may be found in the plaques at the time of their formation. They also occur in the genital discharges and in various tissues and organs, but there seems to be no place in the body of the living animal in which one may confidently expect to find the parasite at all times. The difficulty experienced in procuring the trypanosome from the tissues of the living horse are well illustrated by Mohler[142] who began search for the parasite in 1904 and found it seven years later in 1911, a quarter of a century subsequent to the Illinois outbreak, and fifteen years after Rouget first described it. While the discovery of the parasite is of great scientific interest, it has not solved some of the most important problems associated with the disease. The Bureau of Animal Industry of the U. S. Department of Agriculture apparently failed, at first, to accept as authentic the diagnosis of dourine in the Illinois outbreak, and in its first official publication, the report of Faville in 1892 upon the Nebraska outbreak, designated it as "Supposed maladie du coït." It was not until nineteen years later, in 1911, that Mohler had fully satisfied himself by finding the trypanosome, "thus confirming the diagnosis and also establishing the identity of the disease with that of Asia, Africa and Continental Europe."

The complement-fixation test as a diagnostic agent has been approved and disapproved by eminent investigators. Mohler, Eichhorn,

and Buck[143] expressed great confidence in the method in 1913 and were highly optimistic for the control of the disease in the northwest. After the lapse of thirty years of reliance of the complement-fixation test, dourine still exists in western areas. Upon the other side, Zwick and Fischer[280] found that some animals authentically known to be diseased from dourine tested negative and other horses unquestionably free of dourine but otherwise unhealthy were positive. The reports by different investigators are so conflicting that it is impossible to determine accurately the dependability of the test. Besides these complexities the complement-fixation test seems to be a group reaction involving surra and nagana. All in all it appears that, in the diagnosis of dourine, clinical observation continues to have distinct value, just as already noted when discussing genital tuberculosis: the tuberculin test fails in a numerically important number of cases.

PROGNOSIS

The prognosis of dourine seems to vary widely in different environment. It was not highly fatal in the Illinois outbreak, but accurate figures were not available because a considerable number of animals were destroyed which might have recovered if given the opportunity. I would estimate the possibility of recovery at fifty per cent. In other outbreaks the mortality has been almost total.

THE CONTROL OF DOURINE

Various means have been proposed for the prevention of the spread of dourine other than the control of the diseased animals themselves. As in other contagious diseases, efforts have been made to produce artificial immunity. There is little practical urge in this field: the destruction or quarantine of the diseased animals is more economical as well as more efficient. Some enthusiastic advocates of artificial insemination have suggested that its use might prevent the spread of dourine, but fail to indicate why the suspected or diseased animals should be bred.

Some interesting researches have been made in the field of chemical therapeutics, largely with arsenic and its more complex compounds such as salvarsan and neosalvarsan. There have been favorable reports of these experiments, but the remedies have as yet not attained extensive practical application. In the Illinois outbreak the physical recovery advanced slowly without interference and it has not been definitely shown that under medication they would have recovered more rapidly or surely. Neither has it been clearly learned when an apparently recovered animal is safe for breeding. An apparently

recovered mare may be quite efficient for work. When a stallion has been authentically condemned as affected with dourine his career as a sire is ordinarily terminated: owners of mares do not care to breed them to such an animal. Among semi-wild range horses, such as Mohler[142] and Watson[220] were dealing with, the individual value of the animals and the environment exclude medicinal treatment.

Under favorable environment, such as that in the Illinois outbreak, quarantine may be effective as a control measure. In that case the affected animals were well known to neighbors and the citizens were quite aware of the perils of the disease. The quarantine was limited to breeding, sale or removal from the infested area. So far as known no quarantined animal escaped from the territory and the breeding quarantine was violated but once, and cost the violator one stallion and one mare. Under other environment quarantine of a permanent character is inapplicable. The prompt destruction of the diseased is probably the safest and most economic course to pursue in almost all cases. The disease should be recognized early in the outbreak before many animals become involved, and handled vigorously. At this juncture the responsibility of the local practitioner is of vital importance. There was no private practitioner in the immediate area involved in Illinois. The horse breeders had not yet become interested in serious contagious diseases. Hence when dourine broke out it was allowed to spread insidiously for two years and in the meantime numerous animals were sold, probably among them some which were diseased.

The Trichomonad Disease of Cattle

During recent years a serious infectious disease of the genital organs of cattle, due to a trichomonad, has attracted much attention of veterinarians and dairy husbandmen. In 1928, Riedmüller[170] described the trichomonad briefly, ascribed to it definite pathogenic power as a cause of genital disease, and cited Mazzanti as having discovered the parasite in 1900. In 1932, Abelein[1] recorded the results of extensive clinical studies of the trichomonad disease, supported by experimental investigations and microbiological researches. Abelein's contribution is generally regarded as the essential foundation for clinical studies of this malady.

Looking backward, there appears ample evidence that the trichomonad disease is not of very recent origin. Hess[89] in 1909, while discussing infectious vaginitis (*Vaginitis follicularis infectiosa*) states: "We have repeatedly observed the interesting fact, that cows bred to certain old bulls which had been infected (with infectious vaginitis)

and were at the time apparently healthy, showed after 6, 9, 12 to 21 weeks after service, a purulent vaginal discharge, following the death of the young, and their maceration, constituting *Endometritis purulenta chronica.*" Hess thus vividly portrays the trichomonad disease in Switzerland while the controversy was raging between the advocates of *Bacillus abortus* on the one hand, and those of *infectious vaginitis* on the other, as the sole or chief cause of abortion and sterility. In America, Lothe[124] in 1928 presented a highly interesting report before the American Veterinary Medical Association upon the destructive prevalence of abortion and associated phenomena in a valuable herd negative to the test for *Bacillus abortus* infection. Lothe's paper was discussed by the leading experts upon contagious abortion, who limited their efforts to the defense of *Bacillus abortus* infection as the sole important cause of interferences with reproduction and left the questions raised by Lothe severely alone. Lothe had reported as an outstanding phenomenon the failure of numerous cows bred to one bull to come in estrum after coitus and their developing some months later a peculiar type of pyometra. It now appears quite certain that Lothe was dealing with trichomonad infection.

Immediately following the significant work of Abelein, several American veterinarians were studying the disease independently of each other. Emmerson[50] reported the disease in 1932 as existing in Pennsylvania or vicinity; McNutt, Walsh and Murray[131] reported a serious outbreak of the malady in Iowa in 1933; and during the same year, Cameron, Fincher and Gilman[23] reported a virulent outbreak in New York, believed to have been introduced by a bull imported from Iowa.

The history of trichomonad disease constitutes a highly interesting chapter in the evolution of hypotheses and knowledge regarding genital diseases of domestic animals. Abelein traces in a vigorous and illuminating manner the development of the theories regarding the causes of interferences with reproduction. He recounts the great ado over Isepponi's[99] contribution in 1887 regarding the "nodular vaginitis," which was enthusiastically supported by many authorities. Then followed Bang[8] with the epochal announcement in 1897 of the discovery, in *Bacillus abortus,* of the cause of infectious abortion in cows. Great disputes arose between the supporters of the two hypotheses, until finally, at the meeting of the association of expert investigators of the diseases interfering with reproduction, sitting at Munich in 1925, an authentic solution of the sterility-abortion problem was handed down to the clinical veterinarians. It was then and there declared that the nodular vaginitis of Isepponi was

not a cause of epizootic abortion. Abelein holds that the declaration failed to solve the problem among veterinary practitioners, and likened the situation to the swinging of a pendulum, now to one extreme, then to the other, and failing to come to rest on the center.

The history of the emergence of the trichomonad disease from obscurity is highly instructive. The discovery of vibrios, streptococci, paratyphus and numerous other bacteria associated with outbreaks of epizootic abortion was able to make scant impression upon the fable that abortions due to infection were all, or essentially all caused by one bacterium. Often the various bacteria found in abortions were associated with *Bacillus abortus*. When the trichomonad disease emerged from obscurity the clinician had before him a disease presenting new symptoms which had not received definite attention, although practitioners like Hess in Switzerland and Lothe in America had observed the signs without having been able to understand them.

The trichomonad disease presents one peculiar phenomenon for which as yet no solution has been offered by microbiologists: the disease is reported almost solely in Bang-free herds. One exception occurs in the contribution of McNutt, Walsh and Murray, in a manner which emphasizes, rather than denies, its preference for Bang-free herds. In Herd I, which was Bang-free, they recognized the protozoan parasite in thirty-six per cent of exposed animals and concluded that all were infected. In their Herd II only five per cent were found infected; in this herd some *Bacillus abortus* infection was present, but almost no breeding trouble could be attributed to it. In no case were trichomonads and *Bacillus abortus* found in the same animal. It may be that there is an antagonism between *Bacillus abortus* and *Trichomonas bovis* and the one needs be eliminated before the other may exert notable pathogenic power. In an unusually brief space of time, trichomonad disease has forged forward into a definitely important position as a peril to bovine reproduction.

The trichomonad disease is apparently quite widely disseminated in Continental Europe. It is known to exist in New York, Massachusetts, Pennsylvania, Wisconsin, Iowa and California and is probably present in the cattle of all states. The actual economic losses from it are not definitely recorded. McNutt, Walsh and Murray state that in one herd about one-third of the females eventually produced calves, but fail to state explicitly the number of females or the time covered. As nearly as may be judged, the duration of time was about two years, and if this is correct then there was one calf per annum for each six cows. The contribution of Cameron, Fincher, and Gilman leaves the impression that the losses were very severe.

SYMPTOMS

The symptoms in the bull are not characteristic. Preputial catarrh is present, but it differs little from the catarrh due to other infections. The catarrh advances and recedes as the parasites increase or decrease. There are no systemic disturbances. The clinical evidence of the presence of the infection is the transmission of the malady to the females with which he copulates.

The symptoms in cows are not always pathognomonic. In many cases there occurs within a few days after exposure a well defined vaginitis with a muco-purulent vaginal discharge. Abelein records characteristic nodules in the vaginal mucosa which give to the hand a sense of rasp-like roughness. This has not been observed by some other writers. It is not quite clear how one may differentiate between the nodular vaginitis of trichomoniasis as described by Abelein and the vaginitis of Isepponi, to be described later and supposed to be a wholly different malady. The trichomonad, so far as known, is deposited within the vagina from the surface of the penis and glans, in close proximity to the spermatozoa. They are not known to injure the spermatozoa prior to the ejaculation and their direct action upon the spermatozoa after coitus is unknown. It is known, or definitely believed, that a large ratio of copulations fail to produce recognized pregnancy. The trichomonads are endowed with motility similar to that of the spermatozoa. They swim actively like spermatozoa. If the liquid in which they are suspended is moving, they swim against the stream. Hence it appears possible that they swim actively toward the ovarian poles and being present when, or soon after fertilization occurs, may interrupt pregnancy at the beginning. In other cases the parasites destroy the fertilized ovum very early and it is expelled at one or two months. This is in sharp contrast to *Bacillus abortus,* which is non-motile, is believed to invade the uterus usually from the blood stream, and is very rarely associated with early abortion.

A very frequent and striking phenomenon is the early death and maceration of the ovum, with an accumulation of liquid in the uterus, which is designated pyometra. Estrum does not recur after service, there is no immediate vaginal discharge or other symptom of disease and the cow is presumed to be pregnant. The uterine seal apparently remains intact for some weeks, during which the pathological exudate within the uterus increases in volume and gives to the animal the general appearance of advanced pregnancy. Finally she fails to calve at the expected date and rectal palpation reveals a non-pregnant, flaccid uterus containing a large quantity of thin fluid. No

fetus is palpable and no uterine caruncles can be recognized. The uterine arteries are small and wholly wanting in the characteristics of pregnancy. The uterine seal may give way and a profuse discharge be present; the seal may be largely or wholly absent, and yet the contents remain within the uterus—there is complete atony of the organ. If a suitable soft rubber catheter or other appliance be introduced into the uterine cavity to constitute a siphon, one to several gallons of thin, yellowish or grayish flaky fluid may be drawn off. The fluid is regularly non-fetid, in sharp contrast to the fetid pus commonly observed when an ovum macerates in the presence of bacteria or when pyometra follows abortion or retained afterbirth due to or associated with bacterial invaders.

The trichomonad fetal maceration and pyometra are significantly unlike the phenomena so designated in bacterial disease. The so-called pyometra does not cause perimetritis with peritoneal adhesions. The uterine contents, which resist fetid decomposition within the uterus, retain their non-fetid character for several days after their removal from the uterine cavity if held in an open vessel at room temperature. During this period the trichomonads continue vigorous. There is little tendency shown toward the expulsion of the macerating fetus and the accumulating fluid surrounding it: apparently the trichomonad invasion exerts a specific paralyzing action upon the uterus so that it becomes inert. If the fluid is siphoned out, the uterus may resume its vigor sufficiently to contract and expel the macerated ovum and any remaining fluid.

Retained afterbirth is virtually unknown. The ovum generally perishes at too early a period in gestation for retained afterbirth to be probable. Although the details of the pathology have not been recorded, it appears conclusive that the macerating process affects primarily the fetal membranes and dissolves them, in sharp contrast to the sclerosis and calcification of the chorion and the retention of the fetal membranes associated with the invasion of the uterine cavity by bacteria.

The clinical diagnosis of trichomoniasis in isolated cases is often extremely difficult or impossible. As in dourine, the clinical identification of trichomonad disease seems quite practical as a group diagnosis and, since it is transferred as a rule by coitus only, the group is clearly defined and the symptoms of the various cases lead to a safe identification. The objection may be raised that the group diagnosis fails to supply needed evidence to prevent a diseased individual from penetrating a healthy breeding unit and infecting it. Thus far, however, essentially all diagnoses recorded in either dourine or trichomonad infection have been of groups. The pyometra of tricho-

moniasis, when typical, is apparently pathognomonic and may lead to an authentic diagnosis. Its post-coital character, the marked uterine paresis, the accumulation of great quantities of thin, yellowish flocculent pus in the uterus without antecedent parturition or retained afterbirth, and other peculiarities appear to justify a positive diagnosis. For example, a purebred Holstein-Friesian cow was imported into New York from a distant state prior to the discovery of trichomonad disease in America. She was purchased as pregnant after an examination by one of the foremost clinicians in the nation in this field of practice. She appeared well physically and the abdomen enlarged somewhat like pregnancy. No signs of estrum appeared and no genital discharge was present. Her time for calving came without any indications of approaching parturition. I saw her in Professor Udall's clinic. The uterus was greatly enlarged and flaccid. Without retraction the uterus could well be taken for that of advanced pregnancy; retracted into the pelvis with Albrechtsen forceps, the uterus was found to be enormously distended with a thin liquid; the uterine walls were flaccid and without palpable caruncles; and the uterine arteries were of approximately the normal size for the non-pregnant uterus, instead of being enormously enlarged, with a bounding, vibrant pulsation as in pregnancy. Some four or five gallons of thin, semi-transparent, yellowish, flocculent pus were siphoned out. The cow had been purchased in an area in which it was later learned that trichomonad disease had prevailed.

Such cases offer difficulties for the clinician. Cows are not searched for trichomonads as an aid in the diagnosis of pregnancy, nor is it known that the various laboratory methods of pregnancy diagnosis would hold in such cases. In fact there is no ground for claiming that the clinician cited erred in his diagnosis at the time of sale; she may well have been pregnant at that date. Without vaginitis, she would not at the present time be subjected to a search for trichomonads as a prerequisite to sale, and without vaginal discharge the trichomonads would not have been discovered. Today we should probably have diagnosed the case as trichomoniasis when we siphoned out the pus. In such cases a careful clinical diagnosis becomes highly important. At one examination a correct diagnosis of sixty days pregnant may be made; ten days later an ovum may be undergoing maceration and the uterus be of approximately normal volume for seventy days gestation, but the uterus is atonic and the uterine arteries of the caliber of non-pregnancy.

Once a clinical diagnosis has been made, it is frequently necessary, and usually advisable to check the decision by microscopic search for the protozoa. This is not always easy, owing to the fact that in

some cases there are few, if any trichomonads in the available discharges. If discharges are obtained which contain numerous trichomonads, they are not difficult of identification; they are similar in volume to spermatozoa and may be readily seen swimming actively by means of their flaggellata.

The control and eradication are to be based upon the exclusion of the affected animals from coitus and from attempts at artificial insemination. Semen from an affected bull cannot be obtained in such a manner as to authentically bar contamination; the affected cow should not be permitted to become pregnant.

There is no specific remedy—no immunization, no chemical destruction of the protozoan. The disease is without great peril to the life of the affected animal. With the exception of the so-called pyometra in the female, the patient tends to recover its general health in so far as clinical observation may determine. Recovery of the bull may be facilitated by douching the sheath, prepuce and penis. Very weak disinfectants may be used in the douche, but the chief reliance is to be based upon the mechanical cleansing. Possibly there may follow some benefit in the cow by douching the uterus and vagina with physiological salt solution.

When pyometra is present, with or without a macerating ovum, the first consideration is the evacuation of the uterine contents by siphoning. This is often followed by the expulsion of the macerated ovum. Later heat develops and the vaginal tract is largely cleansed. If estrum fails, the corpus luteum of pregnancy may be advantageously removed. If deeply embedded so that it cannot be safely dislodged by digital compression, the capsule of the ovary should be punctured with the ovarian scalpel (Fig. 43), after which the yellow body is readily removed by compression. When the pyometra is abundant, it must be siphoned out before attempting the removal of the corpus luteum.

Precisely how much is to be gained economically by handling the trichomonad disease remains to be determined. Some animals which have been diseased breed again, but the ratio of reproductive recoveries is at present unknown. In a sense the results of handling the pyometra of trichomonad infection may be better than in postpartum pyometra associated with retained afterbirth and partial or complete necrosis of the uterine caruncles.

Walsh, McNutt and Murray[216] tabulate fifteen infected cows which had aborted or suffered from pyometra. Six had been killed, two were non-pregnant, and seven had conceived at periods of two to nine months after apparent recovery. At date of report none had calved. Their data are not accurately measurable, but the inference is war-

ranted that, if they were ordinary grade cows, little or no profit came from handling. The report of Cameron, Fincher and Gilman[23] leaves the economic outcome of handling uncertain. In both reports insufficient time had elapsed to render the matter clear. Europeans report recoveries, but the ratio of these to the total infected and the delay in breeding is not readily determined.

As in dourine, so in the trichomonad disease, the effective prevention of the spread of the disease from state to state or herd to herd is dependent upon the efficiency of the handling of the malady at the point of origin. In the Cameron-Fincher-Gilman outbreak the suspected carrier was imported from a distant state upon an official certificate of freedom from "contagious abortion," but that did not prevent the bull from causing an abundance of sterility and abortion among cows and the ruin of several potentially valuable bulls. In fact the certificate of freedom from *Bacillus abortus* infection merely misled the buyer into believing the bull genitally healthy. As a consequence of this mistaken confidence, a second herd was contaminated and his cows which failed to conceive by him were fearlessly bred to other bulls, thus assuring the further spread of the infection. Similarly in the McNutt-Walsh-Murray outbreak, the bull, being free from "Bang's disease," was considered genitally healthy and it was deemed quite proper, after he had well distributed the disease in one herd, that he should do likewise in another. The effective control must be *within* the infected herd.

The intra-herd control of the disease needs be based upon the immediate suspension of breeding until the extent of the infection has been authentically determined. Coitus should on no account be permitted with any exposed animal until ample time and opportunity shall have shown clearly that the animal is not infected. There is no agglutination, complement-fixation or other easy test: the decision needs rest upon clinical study of the outbreak and the microscopic search for the parasite. Once the animal is known to have been infected, the question of breeding after apparent recovery should be carefully weighed. Animals near the border line of economical worthlessness should be discarded. Valuable individuals should be granted reasonable delay after apparent recovery, and the breeding should then be so planned that the peril of renewal of the outbreak should be eliminated. A bull which has apparently recovered should be tested carefully upon cows which have been previously bred to him, or upon a limited number of test cows; a cow which has apparently recovered should not be bred to the herd sire used upon cows of known health, but should be mated with a test bull not in general use.

Chapter XXIV

VIRUS DISEASES OF THE REPRODUCTIVE SYSTEM

IT has previously been noted that such virus maladies as sheep pox, hog cholera and foot-and-mouth disease frequently cause destructive abortion in pregnant females. Other viruses invade the reproductive organs alone, so far as may be determined at present, interfering with conception and perhaps interrupting established pregnancy. The field has been only very meagerly surveyed and there is at present no dependable foundation upon which a reliable conclusion may be based regarding the number of species of viruses which may invade the genital system without causing significant disease of other organs.

The Vesicular Venereal Disease. Coital Exanthem. Genital Pox. Bläschenausschlag

There occurs, not infrequently, in horses and cattle an acute coital infection characterized by profuse vesicular eruptions in and upon the copulatory organs of both sexes, which are succeeded by ulcers and cicatrices. Friedberger and Fröhner[65] state that the same disease, or one which cannot be differentiated therefrom, occurs less frequently in goats, sheep and swine, and that attendants upon the infected animals sometimes have similar eruptions on their hands.

In horses and cattle it is one of the most highly transmissible infections known to veterinarians. In my experience the transmission has been essentially universal in all exposed animals. The disease apparently exists in all lands. It is far more common in Continental Europe than in America, the reasons for which are unknown. Friedberger and Fröhner state that in 1886 there were reported in Germany 248 affected horses and 3944 cattle. I have observed during sixty years two outbreaks in horses and one in cattle, involving a total of about thirty animals. Dr. Fincher reports one outbreak in cattle during eighteen years in the ambulatory clinic.

It is not definitely known that the virus is identical in all species. It is stated by some authorities that the disease is experimentally interchangeable between horses and cattle. The symptoms, course and termination are so alike that separate descriptions are not necessary. Since the malady is spread naturally by coitus only, it does not pass from one species to another. Rarely the disease may be accidentally transmitted, e.g., by an attendant in grooming.

The period of incubation is brief, usually three to six days post coitum. After such interval small papules in great abundance appear upon the copulatory organs, which promptly develop vesicles containing transparent, yellowish lymph. These soon rupture and leave behind an ulcer of 5-10 mm. in diameter, which heals in ten to fifteen days with a depressed center, in which the epithelium loses its pigment and in dark-skinned animals, especially horses, the circular, depigmented areas present a striking appearance. The eruptions appear in repeated crops for a few days. These characteristic symptoms are observed most notably upon the generally dark epithelium of the penis, sheath, clitoris and the skin of the vulvar lips. Within the vagina and vulva the lesions cause marked irritation, the parts are painful to the touch, vaginitis is evident with muco-purulent discharge, and frequent urination with evidences of pain. Some writers record increased sexual desire; this may be partly erroneous, the observer mistaking frequent, pathological urination for estrum. The penis of the stallion, being covered by a thin skin which secretes sebum, differs from the naked penis of the bull and modifies somewhat the degree of irritation, although in principle the lesions are alike. There is no fever, inappetence or other signs of systemic injury. If uncomplicated, the lesions heal in about two weeks. The depigmentation commonly persists for several months.

There is some conflict of opinion concerning the economic importance of the disease. Most writers dismiss the question rather lightly by stating that the loss is confined to the delay in breeding, but fail to state precisely what they mean. Ideally the animal recovers from the disease in two or three weeks so that an estrual period is not necessarily missed.

Reisinger[163] and some others contend that it causes epizootic abortion in cattle, but the contribution by Reisinger in 1928 fails to clearly exclude the trichomonad disease in the outbreaks studied. Abelein believes Reisinger was dealing with the protozoan disease and that the virus of the vesicular venereal disease was a chance complication. The description of the disease observed by Reisinger corresponded in all important clinical symptoms with the trichomonad disease as described by Abelein *et al.* At that period no search was being made for trichomonads as a cause of epizootic abortion. The available literature fails to make it clear whether the coitus which transmits the infection may also beget pregnancy. Like others I have failed to make accurate observations in this respect; my impression is that identifiable conception does not generally occur at the coitus when the infection is transmitted. The gonads of the dis-

eased animals are not apparently affected and the ova and spermatozoa so far as known are healthy and fertile. Fertilization probably occurs, but the ovum is perhaps promptly killed by the virus. There is room for the inference that, since the disease continues clinically for but two or three weeks, any destruction of ova probably is completed within that time.

Rarely the disease may become severe and chronic, resulting in persistent vaginitis or metritis. I have observed one such case, in a mare. It is stated by numerous writers that no immunity is gained through an attack of the disease.

The diagnosis is comparatively simple. As stated while considering dourine, the vesicular malady has been confused by numerous writers with that malady. There is scant reason in my judgment for the confusion. Sometimes it appears that some have confused the granular venereal disease or nodular vaginitis of Isepponi with this disorder in cattle. The nodular enlargements in the vulvar mucosa are usually somewhat transparent and when viewed hastily may well be mistaken for very small vesicles. These nodules, however, are only 2-3 mm. in diameter. They may have their summits damaged by friction but no vesicles develop and rupture. The suddenness of the onset, the rapid and benign course, the characteristic vesicles which rupture quickly leaving distinct ulcers which heal promptly, and finally the white circular depigmented ulcer sites render the diagnosis clear. While in Continental European states a given outbreak is apparently traceable in most cases to some antecedent transfer of an infective individual to a new breeding unit, this is not generally true in America. As a rule in this country the outbreak is without known antecedents. It might be said that the disease, as a clinical entity, arises spontaneously. Apparently the virus exists in a saprophytic form until some unknown force suddenly arouses its pathogenic powers and there promptly follows one of the most highly transmissible, and characteristic infectious diseases known to veterinary science.

In Continental European countries the disease is reportable and subjected to official control. In America official control is rarely, if ever attempted. When an outbreak occurs in an intelligent community and a proper diagnosis is made, the self interest of the owners of the diseased animals usually suffices for the eradication. Exceptions may arise and official quarantine be essential to proper control. As soon as the disease is identified, all diseased and exposed animals should be withdrawn from breeding. Perhaps the greatest danger lies in community bull associations. If the disease breaks out in cattle of one owner, other members of the association may try to evade

loss by sending their cows to another bull, which may involve mating a diseased cow with a healthy bull, thus causing a new outbreak. All cattle in the association should be excluded from breeding until it is known to be safe.

There is little actual call for treatment. The disease regularly runs a rapid, benign course and recovers spontaneously. Cleanliness of the affected parts probably hastens recovery somewhat. The ulcerating surfaces may be washed with physiological salt solution, with some mild antiseptic. In those rare cases complicated with severe vaginitis or metritis, treatment may be applied as suggested by the symptoms.

Virus Abortion

Dimock and Edwards[42] expressed the belief in 1932 that some highly destructive epizootic outbreaks of equine abortion were due to a virus. They discussed the subject at greater length in 1936,[43] recording numerous serious outbreaks of equine abortion in which they failed to incriminate bacteria, and they definitely concluded that in these enzootics a filterable virus was responsible. The disease presented the same symptoms—or want of symptoms—observed in association with *Salmonella abortivo-equinus*. The mares appeared well until without warning they aborted, after which they recovered promptly and were fertile. Precisely how fertile they fail to state. Since other contributions indicate that in the affected area there are about fifty foals for each one hundred mares bred the previous year, it may be inferred that approximately one-half the mares thus aborting, conceived a few weeks later.

Autopsy of the aborted fetuses revealed in some cases an accumulation of serous exudate in the pleural cavity. The most characteristic lesions observed were "numerous small white degenerated areas in the liver." They do not say in what way this differs from the lesions in *Salmonella abortivo-equinus*, or in streptococcic abortion. Transmission experiments were made with mice, rabbits, guinea pigs and mares. The experiments with mice and rabbits were essentially negative; the guinea pigs were already infected with *S. aertrycke*, from which they sometimes abort. Three guinea pigs, inoculated by unstated avenue, aborted. Dimock and Edwards exposed twenty-two pregnant mares, of which six aborted. The source of the mares was not stated. Inferentially they were obtained in the Lexington, Kentucky, area where six abortions among twenty-two mares is not unusual.

Miessner and Harms[138] studied an outbreak of equine abortion in a stud of mares in which seventeen of twenty-three aborted. Since no

bacteria could be found, the authors concluded they were dealing with a virus disease. Neither they nor Dimock and Edwards record having made search for protozoa. Altogether it appears quite plausible that they were dealing with a form of virus abortion. Should their conclusions be accepted, it is not at all clear how, in the presence of paratyphus or streptococcic infection, the abortion virus may be eliminated. If virus abortion is admitted for mares, then it becomes probable that many of the abortions in cattle for which no cause has yet been identified may be attributed to a virus.

Dimock and Edwards followed rigidly the common rule of totally ignoring such primary causes of genital diseases as the breeding of a foaling mare on the ninth day, as emphasized by Jennings;[101] the mating of mares with stallions producing spermatozoa like those in Figures 28 and 128, and other primary forces which endanger physiological reproduction.

The avenue and date of invasion are unknown. No reliable method of handling has been announced. Isolation is not available because no means of diagnosis are known before the abortion has occurred. Dimock and Edwards state that they have drawn blood from mares which had recovered after virus abortion and injected it into pregnant mares, which did not abort, but they did not know that the mares in question would otherwise have aborted.

The subject is a highly interesting problem which merits careful study.

Chapter XXV

UNCLASSIFIED GENITAL DISEASES

Infectious Vaginitis. Nodular or Granular Venereal Disease

ISEPPONI[99] recorded in 1887 extensive clinical studies regarding severe losses from bovine sterility and abortion in the Canton of Chur, Switzerland, which he attributed to an infectious vaginitis. He observed in the vagina, or rather in the vulva, of cows and upon the penis of bulls a number of nodular or granular elevations in the mucosa, associated with vulvar or preputial catarrh. Occurring ten years prior to Bang's discovery of *Bacillus abortus*, when great concern was developing rapidly because of the destructive prevalence of sterility and abortion, the report of Isepponi aroused great interest, and many of the leading authorities of Continental Europe supported his views. When Bang made his highly important discovery, wide conflict of opinion arose. At the Ninth International Veterinary Congress, at The Hague, in 1909 the two best known writers upon sterility, Hess of Switzerland and Albrechtsen of Denmark, supported vigorously the views of their fellow countrymen, Isepponi and Bang. The conflict of opinion is not so loud now, but it still quietly persists. Some continue to regard infectious vaginitis as an important disease; some consider it modestly injurious; other high authorities deny that the malady causes sterility or abortion. Isepponi regarded it as a specific infection of cattle. The same phenomena in a comparatively mild form are common in sheep, goats and swine. It is not known that they are absent in carnivora.

The wide conflict of opinion regarding the significance of the malady—if it is a malady—apparently lies in the vast variations in its intensity as seen by different observers. The essential clinical symptoms in the cow consist of the appearance of few or many nodular elevations in the vulvar mucosa. When mild there may be visible very few nodules grouped about the clitoris, as seen in Fig. 100. Such animals show no signs of disease beyond the essentially universal discharge of muco-pus in small quantity which dries in inconspicuous brownish or black crusts upon the vulvar tuft of hairs. See Figures 31, 32. In these milder cases the nodules are transparent and suggest vesicles. This has apparently led, at times, to confusion with the vesicular venereal disease in which the nodules do not form vesicles and remain somewhat permanent.

In the more severe cases, probably the type studied by Isepponi,

the nodules may be extremely numerous, as in Fig. 101. In such cases the nodules tend to arrange themselves in closely packed rows along the summits of the longitudinal folds of the vulvar mucosa. They may retain their transparency. Usually when many are present the surrounding mucosa becomes deeply injected and swollen. A vascular girdle forms about the base of each nodule, unless they are

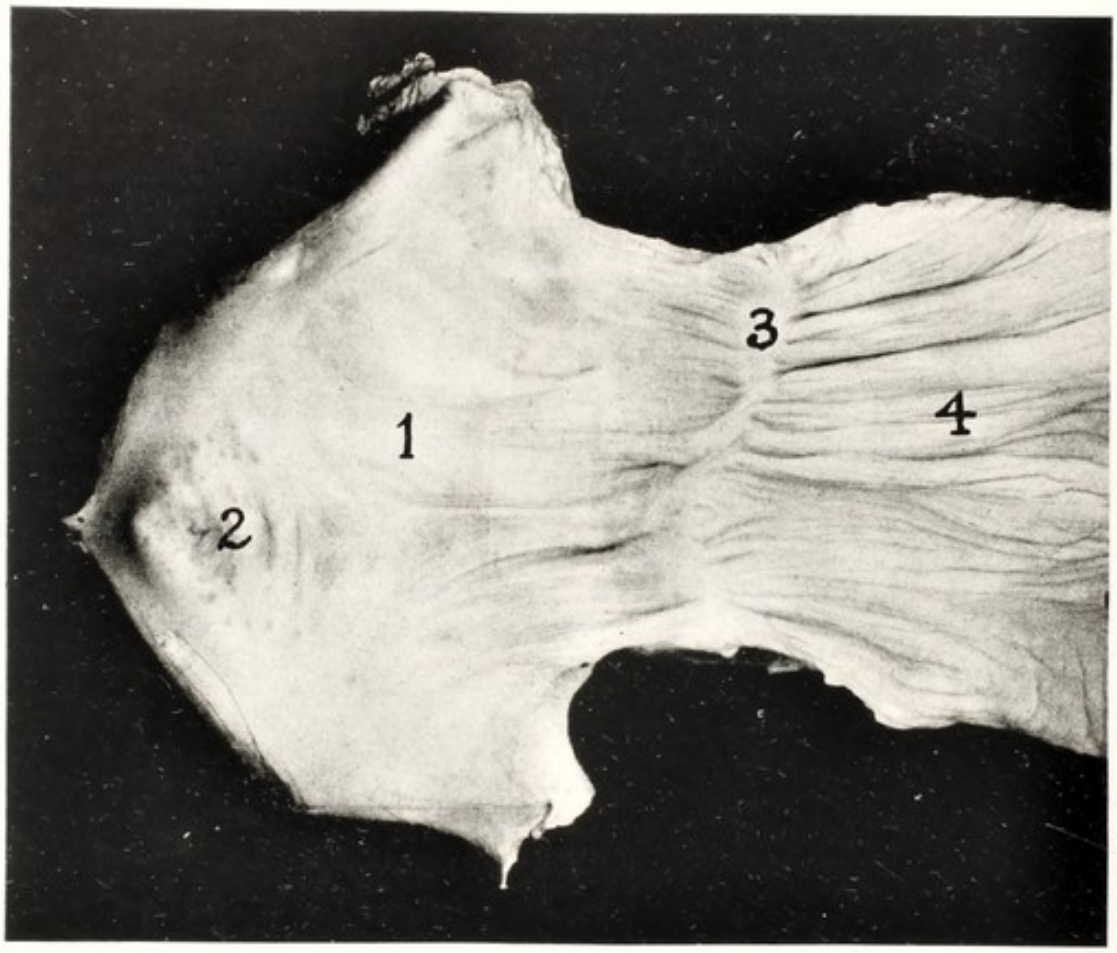


FIG. 100. Mild Nodular Venereal Disease (Infectious Vaginitis) in Heifer. 1, Vulva; 2, clitoris surrounded by nodules; 3, hymeneal line; 4, vagina.

in immediate contact, and the mucosa in general assumes a distinctly inflamed appearance. A definite muco-purulent discharge occurs of a volume which the clinician is forced to regard as disease. If the examiner grasps the vulvar labiae and gently draws them apart, the muco-purulent exudate stretches across the vulvar cavity in white strands. The vulvar mucosa is extremely sensitive in such cases; the parts bleed upon very slight friction or pressure; and urination causes pain.

The penis of the bull presents analogous lesions, as shown in Fig. 102. The bull calf in most dairies when a few weeks old shows definite preputial catarrh, as evidenced by the incrustation and blacken-

ing of the preputial tuft of hairs as in Fig. 33; if the male calf is fed exclusively on boiled milk, the tuft remains clean as in Fig. 34. Severe balanitis develops now and then in young bulls, the glans penis becomes intensely inflamed, the nodules are very abundant, and the parts become so sensitive that the animal refuses to copulate.

The intensity of the symptoms varies widely due to causes not always clearly recognizable. The nodules apparently increase some-

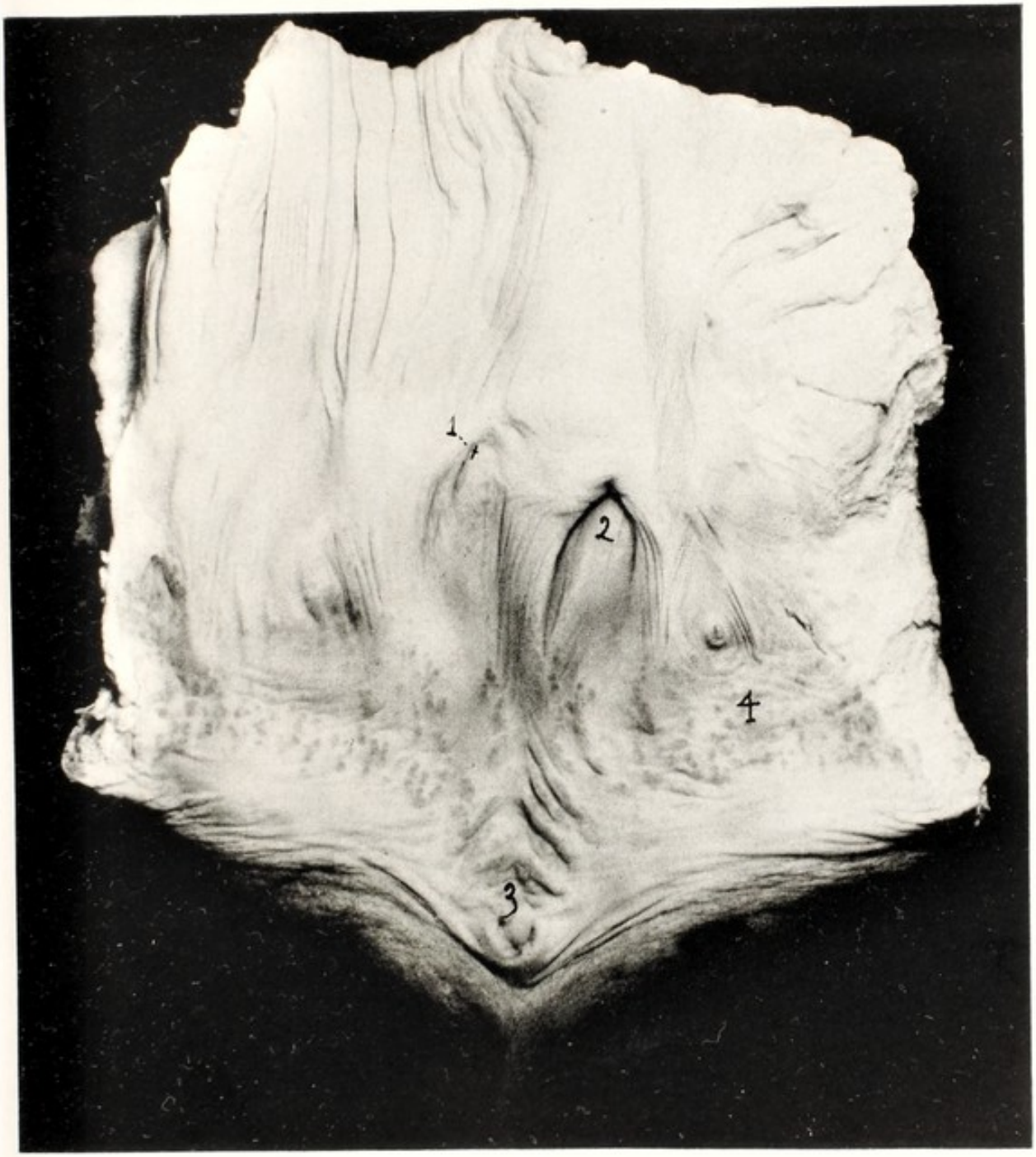


FIG. 101. Advanced Nodular Venereal Disease (Infectious Vaginitis) in Cow.

1, The meatus urinarious; 2, abnormal development of the vulvar end of the Wolffian duct (Gartner's duct) to form a large, blind sac; 3, the clitoris surrounded by nodules: 4, band of nodular enlargements extending around the vulva.

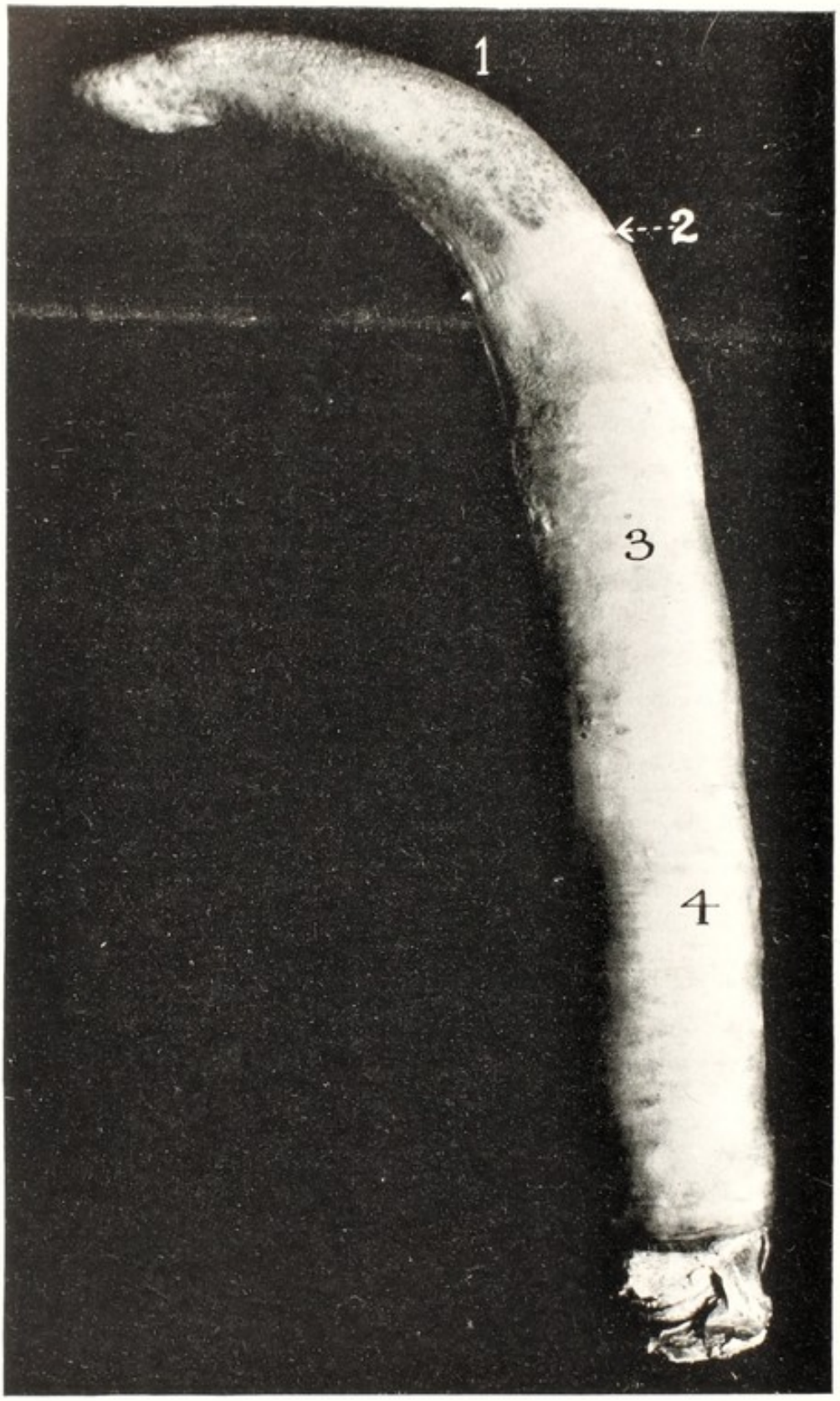


FIG. 102. Penis of Bull Showing Nodular Veneral Disease (Infectious Vaginitis).

From same bull as Fig. 123 (diseased testicles) and 137 (diseased seminal vesicles and ampullae). 1, Glans penis showing confluent nodules; 2, fornix; 3, the reflected prepuce showing solitary nodules; 4, the reflected sheath.

what in the vulva with the advent of puberty and they multiply enormously from coitus. For example, I studied the genital organs in a lot of 80 virgin Hereford heifers in the abattoir amongst which was one which evidently through some accident had copulated and become pregnant. All except the pregnant animal were almost wholly free from nodules, as in Fig. 100, while the vulva of the one pregnant heifer, according to my estimate, contained as many nodules as the

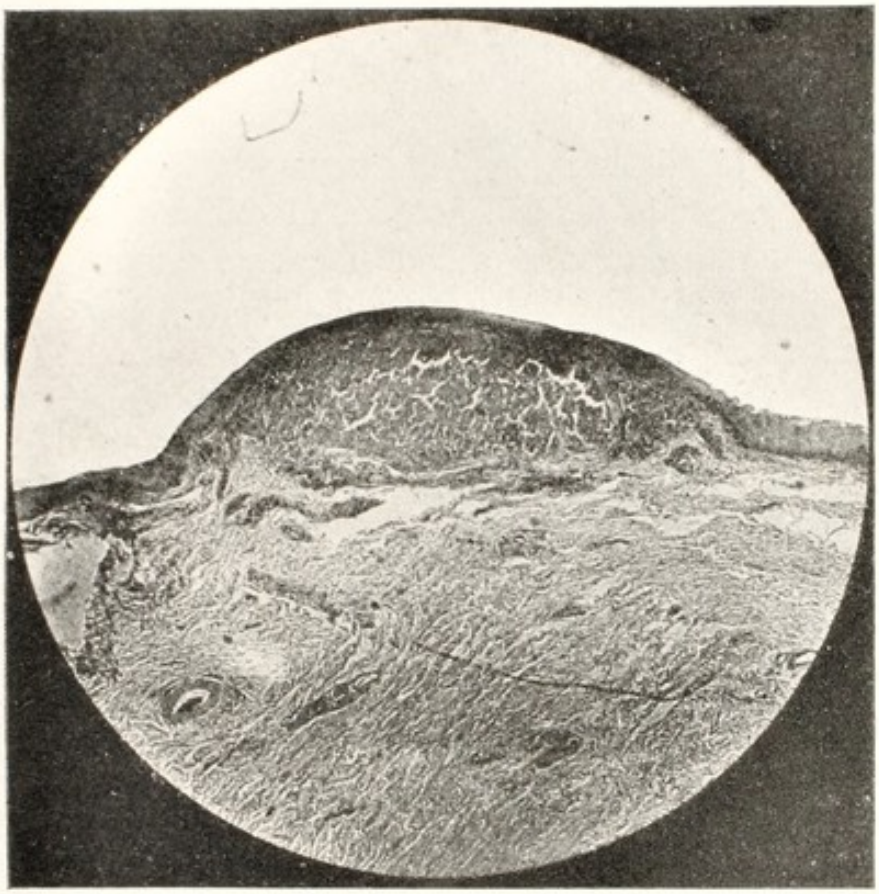


FIG. 103. Section of a Nodule of the Nodular Venereal Disease from the Vulvar Mucosa. (Thoms)

79 others combined. Her vulvar mucosa was swollen, intensely inflamed and covered with masses of exudate. Under the conditions coitus seems responsible for the enormous multiplication of the nodules. Since the heifer had passed mid term, it was clear that the exacerbation of the disease was enduring and not a mere temporary irritation of coitus. Similarly in dairy herds where genital diseases are severe, if a group of virgin heifers in which breeding has been begun be inspected by drawing apart the lips of the vulva it is not at all difficult to identify all those which have copulated: the numbers of nodules will have increased enormously, the vulvar mucosa is highly irritated, and there is a very marked exudate of muco-pus.

When parturition or abortion approaches, the vulvo-vaginal mucosa becomes edematous and the nodules are less prominent or invisible. Similarly in the presence of pyometra or other serious disease of the vagina or uterus the consequent edema may obscure the nodules. The elevations stand out most prominently in heifers which have not calved or aborted. They continue to be recognizable in old age, but are fewer in number and less conspicuous.

Sheep and swine do not ordinarily show great numbers of nodules, marked irritation of the vulvar mucosa, or notable exudate. They present a condition closely simulating that seen in Fig. 100, and would not be classed as clinically diseased by most observers. The pathogenicity of the disease, or phenomena which some call disease, and others deny, arouses much conflict of opinion. In some herds where sterility, abortion and allied phenomena are destructively present, the nodular venereal disease is unusually virulent. It has not been determined, however, whether the interferences with reproduction are *caused* by the nodular disease, or merely *coincidental*. Hence at the time when *Bacillus abortus* reigned supreme as the cause of sterility and abortion, many writers emphatically denied the power of the nodular infection to interfere with reproduction. Apparently the more positive supporters of *Bacillus abortus* based their views upon cases like Fig. 100, which certainly constitute the vast majority of all cattle. When the clinician finds the vulva of a heifer thickly covered with nodules, the mucosa highly inflamed and bleeding at touch, and covered with thick, tenacious pus, or when he sees a young bull with such severe balanitis that he cannot, or will not copulate, he is compelled to classify the animal as diseased. The interference with reproduction in the bull cannot be denied. Clinicians largely, if not generally, would also classify the severe cases in heifers as disease. Inasmuch as the disease appears to be milk-borne in new-born calves, the etiological factor must primarily invade the system via the alimentary tract and reach the reproductive system through the blood stream. If that be correct, the etiological factor in a form at present unknown is presumably able to enter the udder. There is no definite proof that it may invade the intra-uterine young. If such invasion occurs, it would seem that the development of the disease could not be evaded by feeding the calf only boiled milk. On the other hand, Liebetanz[119] claims that the etiological factor does enter the intra-uterine young and causes gastro-enteritis and pneumonia in the new-born calf.

According to some observers the nodules appear somewhat rarely in the caudal end of the vagina, very rarely farther forward, and ap-

parently not at all in the uterus. There have been as many names proposed for the disease as there are opinions regarding its significance. While it has been perhaps most frequently designated as *vaginitis,* that is owing to the rather common habit of thinking of the vulva and vagina together as vagina, while as a matter of fact the nodules in the female are almost wholly confined to the vulva, a distinct organ arising from a wholly different source in the embryo.

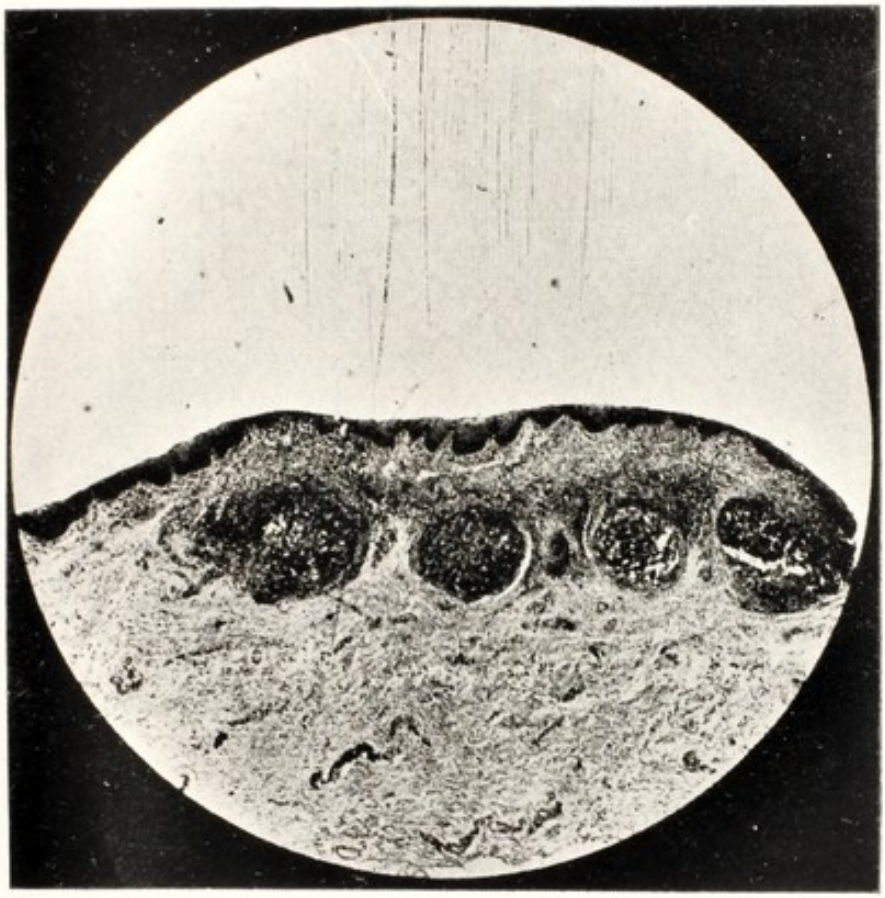


FIG. 104. Section Through a Series of Four Nodules, Causing a Broad Elevation of the Mucosa. (Thoms)

It is also inept to speak of the malady as *vaginitis,* when it affects equally the glans penis and prepuce.

The cause of the disease has not been extensively studied. Ostertag[154] conducted limited researches and obtained cultures of a streptococcus from the vulvar discharges. This he introduced into the vulvae of heifers which he believed free from the infection, and concluded that he had produced the disease. From these he recovered the streptococcus in pure culture. It is not clear whether Ostertag regarded an animal like Fig. 100 as healthy. Neither was the streptococcus definitely identified. It was possibly related to the mastitis streptococcus or to the streptococcus described by Webster[222]

as the cause of epizootic sterility based upon an infectious cervicitis transmitted by the bull. Clinically the disease does not behave in the usual manner of a bacterial infection. Ordinarily when lesions are caused by bacteria, and recovery sets in, the lesions subside volumetrically; in the nodular malady the nodules disappear numerically, and those persisting are about the same size as when the infection was at its height. Blaha,[14] after comparatively extensive studies, reports the finding, upon the summits and within the nodules of, vast numbers



FIG. 105. Histological Section Through a Series of Nodules from the Roof of the Vulva, Without Macroscopic Elevation of the Mucosa. (Thoms)

of sickle-shaped bodies, having nuclei located at one side. These appeared to Blaha very much like the trachoma bodies as described by Prowazek. He concluded that the invaders were protozoa. Such conclusion is in agreement with the clinical character of the disease.

The histology of the nodules has not been extensively studied. The most complete study is that of Thoms[195] who cites Bermbach and Nevermann as stating that the malady causes as great destruction as foot-and-mouth disease. Thoms regards the infection as exclusively coital and believes that the period of incubation is as brief as twenty-four hours post coitum. He describes the nodules as being especially

abundant about the clitoris (which is the common rule) and in well marked cases so numerous that they give to the organ a mulberry-appearance. In the vulva they are arranged in rows upon the longitudinal folds of the mucosa. The nodules, 1 to 2 mm., Figs. 104-106, consist of masses of round or elliptical lymphoid cells. Sometimes capillaries enter the cell heaps. There is round cell infiltration about the nodules.



FIG. 106. Histological Section Through a Mass of Nodules Causing a Knob-like Elevation of the Vulvar Mucosa. (Thoms)

The prevention of the nodular venereal disease calls for precautions during calfhood and at the time of mating. If Liebetanz[119] be correct, that the etiological factor enters the fetus in utero, then it becomes necessary to go back to the period of conception and follow the law of mating only healthy animals. But, as stated above, if such animals as illustrated in Fig. 100 be diseased, then the mating of healthy cattle is essentially impossible. So, as I see the problem, such animals should not be classified as *diseased* but as host to the causative parasite in such numbers and avirulence that no disturbance of function is visible. Such is the case in essentially all sheep and swine, in well raised dairy cattle and in almost all beef cattle. As

indicated in Figures 32 and 34, calves may be grown, according to my experiments, upon boiled milk and kept free from the nodules. But experience indicates that the calves which are doomed to develop the nodular disease are quite largely in such a state of health that they cannot endure boiled milk. The call for better growing of calves, as will be discussed more fully later, is most urgent in those which demand raw milk. It is practical and economical, however, to so grow calves upon raw milk from genitally diseased cows that the nodular disease is reliably held to the avirulent state as shown in Fig. 100 and illustrated in the calves in Fig. 35.

If both sexes have been grown healthfully as calves, so that the nodular disease does not exceed the type shown in Fig. 100, there is no known danger from ordinary coitus. When the phenomena assume a definitely pathological character, as indicated by very numerous nodules, infected vulvar mucosa which bleeds at touch and a vulva containing muco-purulent exudate—or when the penis of the bull is extremely nodular, preputial catarrh evident and coitus difficult—precautions at the time of coitus are demanded but are not wholly effective.

The balanitis of the bull should be vigorously handled both upon his own account and that of the females with which he is to be mated. In severe cases the penis, prepuce and sheath are to be cleansed before and after coitus. The douching of the parts should be effected by means of an ordinary hospital irrigator fitted with a seven-foot rubber catheter of the diameter ordinarily used for horses. The object may be attained in the absence of the long catheter by attaching a sufficiently long piece of rubber tubing to the irrigator and splicing upon this by means of a piece of glass or metal tubing, an ordinary soft rubber horse catheter. The douche should consist of warm physiological salt solution (approximately 1 oz. salt to one gallon water) to which may be added with benefit 0.25 per cent Lugol's solution of iodine or 0.50 per cent carbolic acid. Powerful disinfectants are to be avoided. The irrigator should be suspended two or three feet above the back of the bull and the catheter inserted into the sheath for a distance of seven to ten inches. The insertion is facilitated by the application of oil or vaseline to the end of the tube. Some of the douching fluid should be allowed to flow slowly from the catheter before it is inserted into the sheath, in order that all air be forced out; otherwise the escape of air bubbles into the sheath is liable to induce resistance by the bull. When the catheter has been introduced, the fluid is permitted to flow freely into the sheath while the operator compresses the sheath opening and prevents the escape of the douche. This causes distension of the sheath, eliminates its numerous folds

and causes the fluid to come in contact with every part. After the sheath has become fully distended, the operator should suddenly release the compression and permit the escape of the liquid. The process should be repeated sufficiently to assure thorough cleansing of the parts. When the balanitis is very severe and coitus difficult or impossible, the animal should be kept apart from females and the douching frequently repeated.

In those herds where the virgin heifers are quite intensely affected and muco-purulent exudate is present in the vulva, the vagina and vulva may be advantageously douched, similarly to the douching of the bull, prior to coitus. The physiological salt solution alone may be used immediately prior to copulation, or the weak antiseptic may be used an hour or two prior to coitus so that the iodine or carbolic acid will have been absorbed or expelled and not injure the spermatozoa. That will not wholly prevent the coital irritation. Within five to ten hours post coitum, after the spermatozoa shall have had ample time to have passed the cervix, the application of the douche with weak antiseptic is highly beneficial and tends to prevent the great exacerbation commonly following the mating. The douche may then be repeated daily for several days.

The Venereal Tumors of the Dog

VENEREAL GRANULOMATA. LYMPHOSARCOMA

There is observed in dogs a specific type of venereal disease not otherwise seen among domestic animals, the precise nature of which



FIG. 107. Venereal Tumors of Dog Involving the Glans Penis and Prepuce.



FIG. 108. Early Stages of Venereal Tumors of Vulva of Bitch.

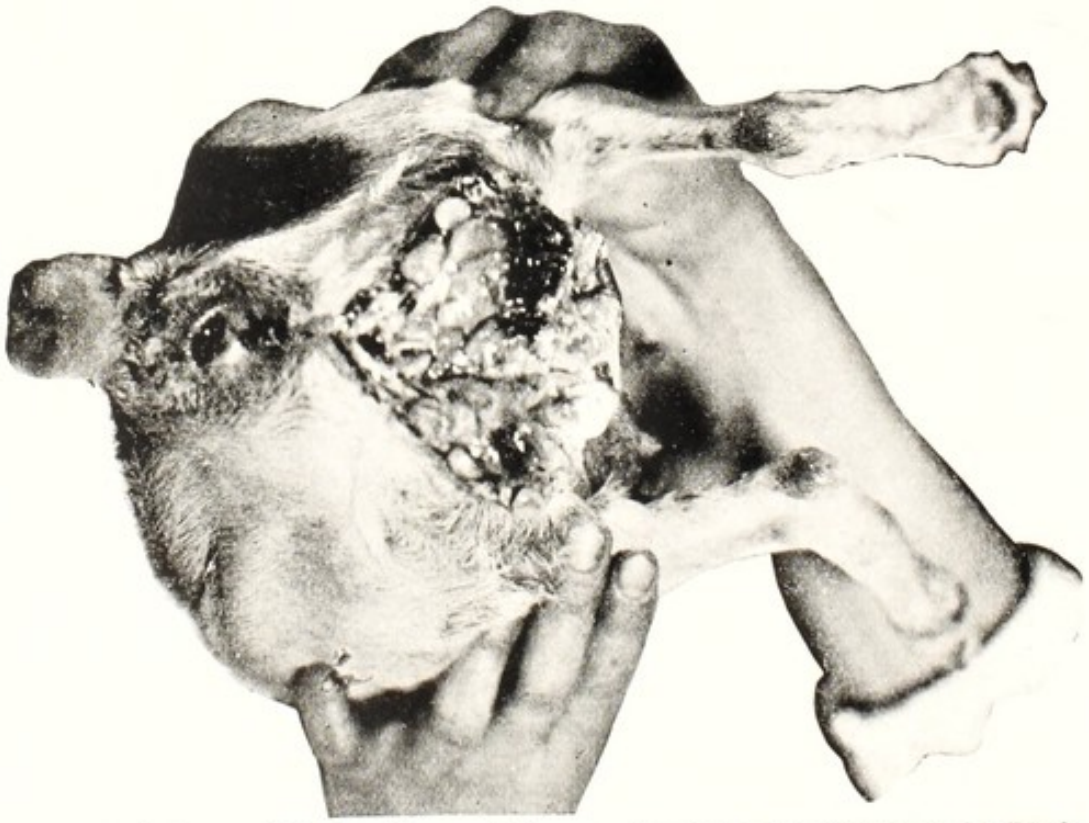


FIG. 109. Advanced Canine Venereal Tumors Involving the Vulva of the Bitch.

has not yet been determined. It is marked by the presence upon the copulatory organs of new-growths variously designated granulomata, lymphosarcoma, alveolar sarcoma, epithelioma, etc. The malady is transmitted essentially by coitus alone, though it may be conveyed experimentally, and rarely transferred by accident. It affects chiefly the penis and prepuce of the male as indicated in Fig. 107 and the vagina and vulva of the female as indicated in Fig. 108. The disease sometimes spreads to adjacent tissues and may cause metastatic neoplasms of the inguinal lymphatics.

A few days after the infecting coitus there appears a bloody discharge from the prepuce or the vulva, accompanied by swelling of the parts. If the penis be exposed, there are observed grayish red vegetations chiefly upon the glans or at the preputial ring, with which the glans is in contact when the penis is retracted. The new-growths may appear at times upon other parts of the mucosa. The disease of the female is accompanied by analogous symptoms—a bloody discharge from the vulva and neoplasms of the same type as seen in the male, located largely along the floor of the vulva, and when large protruding from the vulva as a sarcomatous, bleeding mass as in Fig. 109. The vulvar discharge is usually fetid.

There is no unanimity of opinion regarding the infecting agent. Beebe and Ewing[9] after extensive studies believed it a virus. Numerous investigators have found a spirochete in the tissues, but there is division of opinion regarding the pathogenic power of these.

Histologically the tumors consist of large round or polygonal cells which multiply rapidly and tend to push aside the contiguous tissues rather than penetrate them.

The tumors are malignant and do not yield satisfactorily to treatment unless handled early. The chief reliance is placed in excision, followed by suturing or in some cases by cautery. Castration has been said to favorably affect its course. The exclusion of diseased animals from mating is too evidently imperative to warrant discussion.

SECTION VI

THE DISEASES OF VARIOUS GENITAL ORGANS

DISEASES OF VARIOUS GENITAL ORGANS

THROUGHOUT the preceding pages it has been the constant aim to reveal in so far as possible the fundamental causes underlying interferences with reproduction. Aside from a comparatively small ratio of unavoidable accidents, it has been assumed that the possession of such knowledge by the breeder will automatically enable him to avoid the losses due to sterility or low fertility.

Unfortunately, the knowledge in this field is very far from complete, and there is everywhere much conflict of opinion. Moreover there are constant changes of opinion in essentially all phases of the problem, and it should be clearly understood that the character of the interferences with reproduction constantly changes. Not only are new causes discovered by students in the field, but the changes in animal husbandry create new problems in sexual pathology.

The etiology of the diseases of the individual organs of the reproductive system has not yet been so fully determined in all cases that they may be wholly prevented. The abundant failures to maintain genital health make mandatory efforts to cure or ameliorate genital diseases. The veterinary practitioner and the breeder should early and clearly learn that when any genital organ becomes importantly injured by disease it cannot be restored to its ideal physiological state. During the early years of the aroused interest in the pathology of reproduction many fabulous cures were announced, and some traces of those fables are yet visible in veterinary literature. In 1909 Albrechtsen[4] stated "I have *cured* 90% of sterile cows," without clearly defining either sterility or cure. Similar statements are made by many writers, but they fail significantly to follow their *cured* animals through life. To some, a sterile cow is *cured* if she again becomes pregnant, dies from parturition, aborts, has retained afterbirth, succumbs to pyelo-nephritis or otherwise brings financial disaster to the breeder. Experienced clinicians and breeders are beginning to observe that a large ratio of animals which have been subjected to active interferences because of sterility, and thereafter conceive, prove inferior breeders when followed over a long period. There are still veterinarians who *cure* retained afterbirth in cows, but when a considerable number are followed for some years, the cures depreciate notably in their financial success.

The position is taken that when a breeding animal definitely fails in reproduction, owing to disease or defect of one or more of the reproductive organs, whether the phenomenon be designated as steril-

ity, abortion, stillbirth, premature birth or other name, the animal involved should be regarded as "a ship upon the rocks," and any handling viewed as attempted salvage. The animal may, and fortunately does, in many instances, recover sufficiently to profitably produce young, but it should not be expected to breed as well as one which has been successfully guarded against genital disease. The remedies are not retroactive and are powerless to restore the diseased tissues or organs to their primitive state. The treatment may halt the disease process and enable important repairs to be made. The skillful handling of the genital diseases of domestic animals constitutes a highly important field in clinical veterinary medicine. It must increase in importance until animal husbandmen cease to advance the incidence of genital diseases through errors in feeding and breeding. In the meantime the veterinary practitioner and his client should strive with increased earnestness to learn more definitely the etiology of genital diseases in order that these may be eliminated and the losses avoided.

Chapter XXVI

THE DISEASES OF THE MALE REPRODUCTIVE ORGANS

THE male may be of low fertility or wholly impotent because of diseases of other organs than those of reproduction. Such diseases may lower or inhibit the production of spermatozoa. An indefinite list of diseases may prevent coitus, such as painful affections of the posterior feet or limbs, and the presence of umbilical or ventral herniae. Some have recently advocated the overcoming of such disabilities by artificial insemination in horses and cattle, the semen being obtained by the massaging of the ampullae of the vasa deferentia. This has already been discussed and disapproved while considering artificial insemination.

Brief references have been made in Section I to the importance of the male in physiological reproduction. In the evolution of knowledge concerning the pathology of reproduction, the male has been assigned a minor role. The early writers, like Zschokke,[279] note that some males are unable to copulate and that others do not ejaculate spermatozoa with their semen. Such animals are comparatively few, are usually promptly recognized and discarded, and constitute a relatively minor source of loss. In the main, abortion, sterility, retained afterbirth, etc., were deemed female maladies. Slowly this hypothesis has been forced to give way to the uncovering of facts, such as those presented in Tables III, IV, V, and VIII, which show that the male may be responsible for the various phenomena of disease shown by the females which he has served. There remains much conflict of observation and opinion. I have taken the stand that the male is as frequently responsible for sterility or pathological reproduction as the female. Richter[169] considers this estimate too high. The conflict of view is probably more a question of definition than of fact. Perhaps there are more absolutely sterile bulls in the area with which I am familiar, than have been observed by Richter, but we are not widely apart in our estimates. The totally impotent males are quickly recognized and offer no complex problem.

I have been far more concerned about those sires shown in the tables cited in the above paragraph, which were capable of inducing pregnancy, but in which the pregnancy is a liability which leads toward disaster in the form of abortion, pathological duration of pregnancy and sub-normal young, lowering the fertility and value of

the progeny and repressing, instead of advancing the quality of the family or breed.

Numerous contributions have been made to current veterinary literature upon various phases of male sterility in the different species. Richter[166] contributed a highly informative monograph in 1919 upon the sterility of male goats, with ample citations to prior publications in the field, showing clearly the great importance of the reproductive health of the male in goat husbandry. In the first edition of this treatise, 1921, it was definitely held that the health of both sexes was equally vital to physiological reproduction. In 1923[240] I placed greater emphasis upon the importance of the male in genital pathology. This was followed in 1925 by the work of W. W. Williams and A. Savage[266] upon the morphology of the spermatozoa as compared with the breeding powers of bulls. Other writers added important observations. In 1934 Professor Lagerlöf[114] carried the studies in this field a long step further by correlating the breeding histories of bulls and the morphology of their spermatozoa, with the micro-pathology of the testicles.

Among dairy cattle, where genital disease causes the greatest financial losses, the sire is expected to serve twenty-five to one hundred cows and his health is of equal importance to that of all his cows combined.

Compared with the literature upon the physical diagnosis of the genital diseases of the female, the clinical diagnosis of the diseases of the male genital organs is almost blank. The confusion is intensified by the fact that writers upon veterinary anatomy have only very meagerly described the male genital organs. Most unfortunately the descriptions of the normal male organs are not always in substantial agreement with what is ordinarily observed by clinicians. For example, Sisson[180] illustrates the ampullae of the vasa defferentia of the bull as passing back to the prostate, wholly exposed dorsally. On the contrary, it is shown at B in Fig. 139 that in a presumably healthy bull, the bases of the ampullae, 3,3, are completely covered dorsally by the vesiculae seminales, 2,2, while in the grossly diseased bull, A. the bases of the ampullae are also covered by the vesiculae seminales.

The clinical examination of the male genitalia reveals much which is of interest. The penis of the stallion is readily grasped by inserting the hand into the prepuce, through the sheath, and the organ is easily withdrawn, where it may be freely inspected. Phymosis is rarely present and readily identified by palpation. The penis of the bull, or other male ruminant, is not so readily exposed. The sheath and prepuce

are too small to admit the examiner's hand. The penis is completely retracted and is normally exposed only by such erection as eliminates the sigmoid flexure. It is almost necessary to cast the animal in order to fully expose the penis. Then the sigmoid flexure may be firmly grasped through the skin, and constant force exerted toward the sheath-opening, until the penial retractor becomes exhausted and protrusion of the penis occurs. The exposed organ may then be grasped and a detailed examination made. Such examination facilitates the recognition of the nodular venereal disease (infectious vaginitis) and tuberculosis of the glans, both of which have been described. It also reveals the coital rupture of the prepuce and other miscellaneous injuries, defects, or diseases. As a rule such defects have already been suspected because of the behavior of the animal in copulation.

The examination of the testicles is readily done upon the standing animal, when properly secured and prudent precaution taken to prevent kicking. Unless the clinician has had abundant experience, the examination is made far more convenient and accurate if a bull known to be fertile can be used for comparison. Their normal size varies with the age and size of the bull. Their form is somewhat ovate in health, as indicated in Fig. 120, not of the cylindrical character shown in the diseased testicle in Fig. 139. The testicles of the bull normally hang perpendicularly at essentially the same level.

Defectively descended testicles of the bull or other ruminant male may be tilted or lie horizontally, which indicates congenital defects of significance. Varying degrees of hypoplasia, inflammation, and degeneration cause the testicles to be below normal in volume, and their consistency abnormal; they may be either abnormally hard or soft. The mobility of the testicles within the scrotum is said to be lowered by adhesions between the visceral and parietal layers of the peritoneum. This is not very notable in testes such as shown in Fig. 139, because most of the adhesions are by elongated connective tissue strands which do not bind the two layers firmly and closely. Even when they are bound closely together, if the connective tissue between the cremaster and dartos is unaffected, the mobility of the gland is not notably affected so far as palpation reveals. It is to be remembered that the visceral and parietal layers of peritoneum, the latter embraced within the terminus of the cremaster muscle, are of intra-abdominal origin.

Descent of the testicle is a somewhat misleading term: the process involves the descent of the cremaster muscle and the peritoneal sac, within which the testicle later moves down into the pouch consisting

of the dartos and skin. The scrotum thus consists of an extra-abdominal portion (skin and dartos) and an intra-abdominal part, the cremaster and parietal peritoneum. Between these two portions there is an abundance of loose connective tissue so that the testicle with its parietal sac and the cremaster may move freely within the darto-cutaneous sac. If inflammation is sufficiently virulent to invade the areolar tissues between the two portions, as is commonly true in acute orchitis, the gland becomes fixed within the darto-cutaneous sac. Very serious degenerative changes, with evidences of scrotal peritonitis and adhesions between the two peritoneal layers, may develop as in Fig. 139, without readily detectable immobility of the testicle.

Serious diseases of the epididymis largely reveal themselves by palpation. The palpation of the intra-pelvic portions of the male genital system is no less important than the rectal palpation of the female organs, but has been almost totally neglected. Referring again to Fig. 139 for illustration, one may palpate the ampullae, 3,3, which are often enlarged, as in A. The enlargement is not only transverse, but also longitudinal. If B. is accepted as normal, it also appears that in disease the ampullae may lose their conical, tapering form and cease bluntly at their anterior ends to be continued by the vasa defferentia. I have observed the ampullae extremely sensitive in the stallion while the disease was in the active stage. This is probably generally true in stallions and bulls during the active stages of disease. The seminal vesicles, 2,2, are quite readily palpated. Normally they are fairly equal in size, as in B. and lie above the neck of the bladder. Their bases cover the urethral ends of the ampullae and their bodies extend forward alongside the ampullae in the form of a triangle with its open base anterior. When inflamed they become enlarged, are frequently grossly unequal in size and vary in consistency. They may be edematous and unduly soft, as they were in A. of Fig. 139, or they may be hard, unduly sensitive or fluctuant owing to suppuration. They frequently become extensively adherent, as indicated in Fig. 137. In some cases the pathological excretions escape into the urethra, while in others the excretory duct is blocked, and the resulting abscess ruptures into some contiguous organ. All my cases have ruptured into the rectum, as in Fig. 138. In such cases the sclerotic, thickened walls of the abscess remain, and the floor of the rectum is firmly adherent to the remnants of the seminal vesicles and to the ampullae of the vasa defferentia. At the point in the ventral wall of the rectum where the abscess ruptured, a readily palpable, indurated cicatrix persists.

The comparatively small prostate gland, Fig. 139 B. 1, is freely palpable and such enlarged prostate as shown in A. in the same figure, is readily identifiable.

The teratology of the male subsidiary glands has not been studied. The diseased bull, A. Fig. 139, was apparently defective at birth. The base of the left ampulla lies upon, instead of alongside the right organ and the right seminal vesicle is bent outward until it is almost at right angles to the ampullae and pelvic urethra. This served to cause confusion while palpating the pelvic organs in the living animal.

Thus the rectal palpation of the accessory male sex glands becomes highly significant, and invites careful study by the veterinarian having to do with genital diseases.

Much as in the diseases of the female genital tract, so in those of the male, there is a marked relationship between the entire list of the organs. Thus in Fig. 139, the testicles, the ampullae of the vasa defferentia, the seminal vesicles and the prostate were all involved. Again the bull from which Figs. 102, 123, and 137 were taken suffered from general infection of the entire genital system. The spermatozoa inevitably participated. Usually some one organ or pair of organs is predominantly diseased. The attention is concentrated upon the single organ and the existence of disease in other units of the system is undetermined. So we describe orchitis, epididymitis, inflammation of the ampullae of the vasa defferentia, semino-vesiculitis, etc., without recording the presence or absence of disease in the other units of the system. At present it is not known in what ratio of cases a given unit of the male genital system may be diseased, without the participation, in some degree, of the other organs.

The avenue of invasion of the male genital organs is generally unknown. Bacillus abortus, orchitis and semino-vesiculitis are apparently believed due to invasion via the blood stream. The literature is otherwise silent concerning the avenue of invasion. There is equal silence regarding the basic causes of the invasion. Indirectly highly significant facts tend to form a safe foundation for conclusions. The diseases of the male genitalia are by far most frequent in dairy bulls, perhaps next in thoroughbred stallions, with the male goat occupying third place. The frequency runs essentially parallel with the degeneration of the breed, authentically referable to the high-pressure breeding, feeding, and handling practiced. These conditions are expressed in two closely linked phenomena—congenital defects (teratology) and congenital weakness (pathology). The bull illus-

trated in Fig. 139 was quite certainly born with anatomically abnormal genital organs, which logically branded him as a sexual weakling, highly vulnerable to various invaders.

To these are added, in dairy bulls and goats, the artificial feeding of the young animal, resulting in low or indifferent health during the period in which the genital system is completing its evolution. Then follows early and heavy breeding, close confinement and other items in handling which serve to depress the vigor and increase the vulnerability to invaders.

When diseases of the male reproductive organs have reached a stage where they may be readily identified clinically, they are remarkably and discouragingly defiant of all efforts at cure or amelioration. Any hope for their effective control, as the problem now appears, is dependent upon prophylaxis. The description of the various diseases and defects of the organs has its chief value in opening the path for the timely discard of the animals as sires. Exceptionally they may be cured or the disease ameliorated.

The Diseases of the Testicles

TESTICULAR HYPOPLASIA

Testicular hypoplasia is frequent in dairy cattle and goats. Little is recorded regarding its occurrence in other species or in beef cattle. A bull coming under my observation, said to have been born a single, had small, soft testicles well descended in the scrotum. He was of fair size, in apparently good physical health, but had the conformation, horns and voice of a castrate. Sexual desire was wholly absent. There was complete arrest in the development of the tubuli seminiferi in the testicles. Richter[166] records similar arrests in male goats. Such extreme types are uncommon.

Testicular hypoplasia of dairy bulls is associated with varying degrees of general, as well as of testicular development. The sexual desire is usually normal, and coitus is performed readily and naturally. The testicles are generally subnormal in size and soft to the touch. There is every possible degree of hypoplasia. If the defect is mild, the bull may show low fertility, the testicles may be normal in volume and consistence and the diagnosis be dependent upon a study of the stained spermatozoa, the low fertility of the bull and the histological examination of the glands.

In such rare cases as the one mentioned above, the hypoplasia is first revealed by an absence of sexual desire. In other instances the bull arrives at breeding age with ample sexual desire, copulates freely, ejaculates spermatozoa like those shown in Fig. 128 *b* (stallion) and

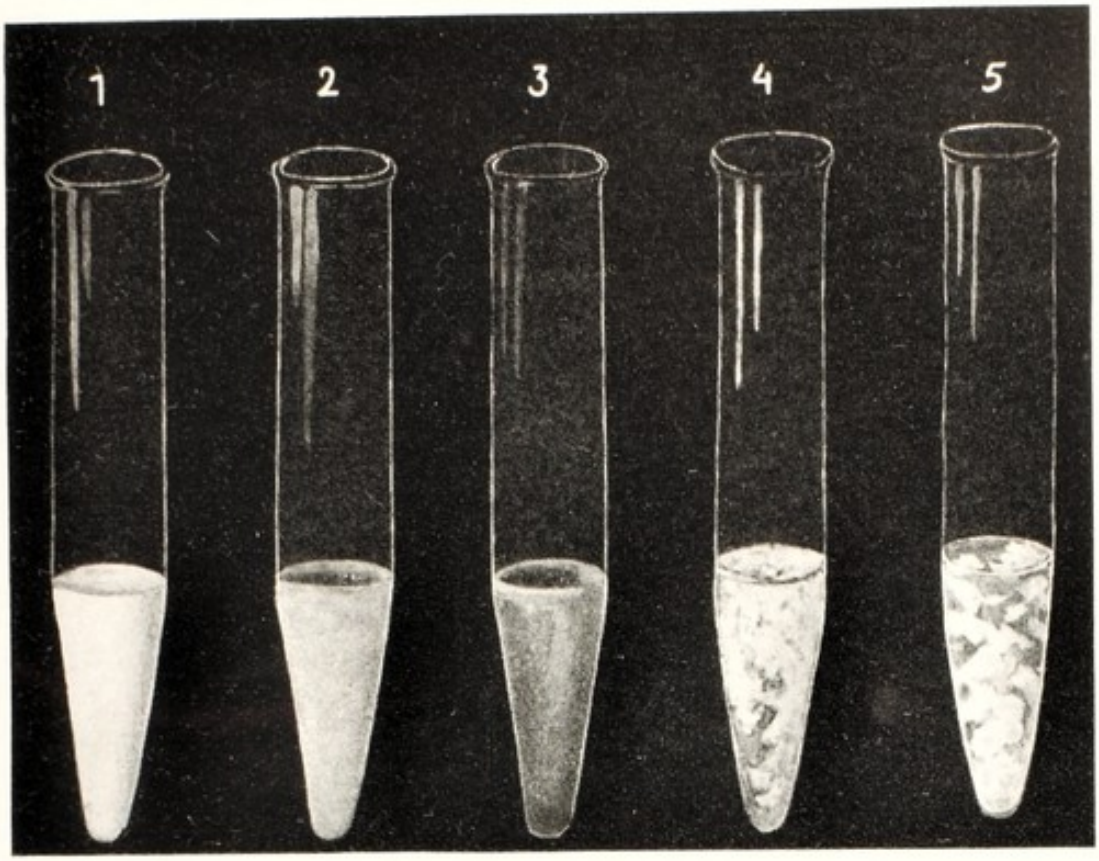


FIG. 110. Semen from Healthy and Diseased Bulls.

1, Semen from a normal bull with about 1,000,000 spermatozoa per cmm.; 2-3, Semen from bulls with testicular hypoplasia—2, about 200,000 sperms per cmm.; 3, about 25,000 per cmm.; 4, 5, semen from bulls suffering from purulent inflammation of the seminal vesicles—note the clumps of pus in the otherwise transparent semen. (Lagerlöf)

is totally sterile. In the milder cases, where some tubuli seminiferi are normal, or near normal in development, there is subnormal fertility of varying grades, and the stained spermatozoa reveal a corresponding ratio of defective cells. Hypoplasia becomes evident in the young bull when first placed in service. At this time the volume of semen is physiologically low. Healthy semen is opaque-white: in hypoplasia it is frequently watery, somewhat transparent, and may be of yellowish color, as indicated in Fig. 110. The semen of healthy bulls contains 300,000 to 1,000,000 spermatozoa per cu. mm., while that of the hypoplastic animal may be destitute of sperms and rarely reaches the normal number of the healthy animal. The spermatozoa, when present, in hypoplasia, are sometimes devoid of motility, and usually of subnormal motility. When the spermatozoa are fixed and stained, they are regularly of abnormal morphology, the ratio of abnormal sperms increasing as the degree of hypoplasia increases, as indicated in Fig. 111. A large proportion of morphologically defective spermatozoa take stain imperfectly, indicating physiological, as well as anatomical defectiveness.



FIG. 111. Spermatozoa from Three Diseased Bulls Showing Abnormalities in Morphology and Staining Affinity.

a and *b* are from a bull of satisfactory fertility for eight years, when he broke down and got 10 visible pregnancies from 190 copulations. Bull *c* was purchased at two years as an association sire. His semen was examined prior to purchase and numerous motile spermatozoa being present he was pronounced fertile upon the basis of motility. He came from a herd free from tuberculosis and *Bacillus abortus* infection. During his first seven months he served 44 cows without a recognizable pregnancy: all conceived to a fertile bull. His stained spermatozoa showed 57% pathological, 30% of which had protoplasmic drops about the neck. Testicular hypoplasia and pathological spermatogenesis. Bull *d* suffered from obstinate diarrhea as a calf. He made 116 services with six recognizable conceptions. Testicular hypoplasia, probably caused by the calf diarrhea. (Lagerlöf)

The histological study of the testicles shows endless variations in the arrest of development of the seminiferous tubules. Thus in a bull aged two years, which had been used extensively without having produced a pregnancy, the semen from which contained no spermatozoa, and many giant cells, Lagerlöf found that one-half the seminiferous tubules had been completely arrested in their development, while the other half had several layers of germinal epithelium, but no spermatozoa were formed, the spermatids degenerating into

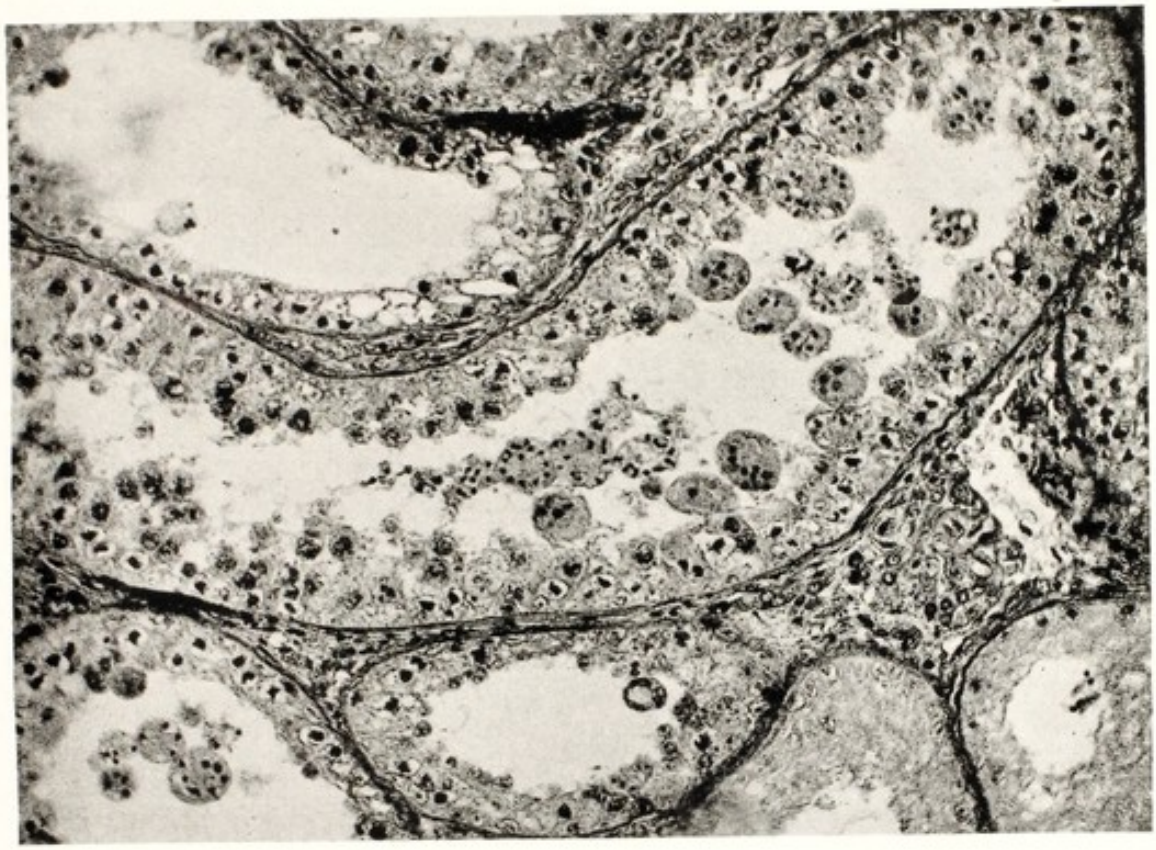


FIG. 112. Histology of Testicular Hypoplasia in a Young Bull.

Some of the tubuli seminiferi show degenerative change and the formation of giant cells. He served sixteen cows without a pregnancy. Compare Fig. 26*c*. (Lagerlöf)

giant cells as indicated in Fig. 112. Histologically the hypoplasia passes from absolute arrest with aspermatogenesis, by imperceptible gradations, to the normal. The fertility of the animal corresponds with the histology of the glands, passing from absolute infertility step by step through every grade to physiological reproductive power. Over 25 per cent of the bulls of low fertility or totally sterile studied by Lagerlöf had testicular hypoplasia. It constitutes by far the chief cause of low fertility or absolute sterility of young bulls when first placed in service.

Richter[166] in his excellent monograph upon sterility of male goats reports that of twenty sterile animals studied, eight were due to

testicular hypoplasia. Richter cites Lang as having diagnosed hypoplasia in fourteen out of thirty-eight sterile male goats. The aplasia either is congenital or occurs very early postnatal. Richter reports that in most cases the goat had normal sexual desire. One animal had no sigmoid flexure of the penis and could not therefore copulate. Other cases were unable to copulate because of too short a penis or inadequate erection. Penial defects are not known to be commonly associated with testicular hypoplasia in bulls. Lagerlöf mentions no cases. I have observed a young bull with a misshapen glans which caught in loose folds of skin at the navel and barred the protrusion of the penis, but I did not learn that his testicles were hypoplastic: perhaps they were. He was distinctly teratological.

Both Lagerlöf and Richter, and many authors which they cite, attribute testicular hypoplasia largely to heredity. With this view I quite agree in the main. Lagerlöf[116] reports "in a polled breed of cattle in Sweden (a highland breed) a widespread hereditary form of testicular hypoplasia occurs. In this breed the left testicle is, as a rule, hypoplastic, while the right one is generally normally developed. This form of hypoplasia occurs in approximately 25 per cent of all males of the breed. In the cows there occurs at the same time a hypoplasia of the left ovary, likewise to the extent of about 25 per cent." When the hypoplasia is moderate and the fertility low, the spermatozoa are inevitably subnormal, the fertilized ovum weak, and the last system to be formed in the embryo, that of reproduction, is in peril of arrest in its development.

Lagerlöf[115] cites Blomberg as having studied a medium sized herd of valuable pedigreed cattle in which sterility had been prevalent. The management later purchased an especially promising bull, which proved highly satisfactory. One of his sons was installed as herd sire, although half- or full-brother to most of the females. Ten of his get, examined for sale, proved to be aspermatogenic and had to be discarded. Some of his get bred for a time and later became infertile. The hereditary testicular hypoplasia was outstanding. Until the primitive law "like begets like" is superseded, the breeder who relies upon a sire of low fertility, will obtain progeny of yet lower fertility.

It does not appear warranted to ascribe all genital hypoplasia to heredity. Any serious impairment of the health of the young endangers the orderly completion of the genital system. As has been stated in Chapter IV and illustrated in Table X, genital hypoplasia had clearly arisen at least chiefly from subnormal health of the calves. Accordingly the cause of testicular hypoplasia may become potent in three separate chronological eras: 1, it may be based upon a defective

spermatozoon or ovum; 2, it may originate in the embryo; 3, it may arise at any postnatal date prior to puberty. Either two or all three eras may be involved.

Male animals affected with testicular hypoplasia in an important degree constitute a serious peril to successful reproduction. Those young bulls in which the hypoplasia is so complete that they produce no pregnancies are soon recognized and discarded. Lesser degrees of hypoplasia, with subnormal fertility, are far more important. Some cows become pregnant, leading too often to the belief that the female, not the male, is at fault. With only a portion of the females failing to become pregnant, the young sire is continued in service. Each failure constitutes an economic burden upon the breeder, and repeated services to the failing female throw an abnormal burden upon the male, rendering him less fertile and more dangerous. As in other types of low male fertility, so in hypoplasia, logically many fertilized ova perish and disappear unseen, and the female is in estrum at an abnormal period.

Prevention consists of the exclusion from the breeding herd of animals having testicular hypoplasia and the intelligent care of newborn young from birth to sex maturity. Once the hypoplasia is established, there is no remedy.

Richter observed, in addition to the cases of aplasia or hypoplasia of the testicles of young male goats, many which were sterile or of low fertility when first placed in breeding, due to spermatic blocking or embolism of the coni vasculosi, and cites Wester as having made similar observations. Upon autopsy, the testicles show swellings in the head of the epididymis of varying size, some as large as a hazel nut or larger, having a yellowish or pale brownish color. Upon section the swellings are found to contain a cheesy mass, not calcareous, but suggesting tuberculosis to the inexperienced observer. The cheesy, or smegma-like mass is readily squeezed out. Microscopically it consists of countless spermatozoa. They do not reach the tail of the epididymis. The *vasa efferentia* are apparently closed and retain the spermatozoa, distending the canals and causing degeneration and atrophy of the testicular tissues. Both Richter and Wester apparently believe that some disease, not infectious, acts upon the canal walls and results in the obliteration of their lumen. Since these phenomena occur in unused males, it would appear not impossible that the *vasa efferentia* were teratological and had acquired characters not unlike the blind tubules of the allied *Ducti aberrantes* and the paradidymi of the embryo. The appearance of these lesions is shown in Fig. 113. Richter cites Schwartz as having

observed similar lesions in a sterile bull which had been fertile. The hypothesis that the spermatic blocking of the ducts is teratological and in a sense hereditary would suggest their analogy to the stenosis of the ducts of Mueller near their cranial ends, with distension of the defective tube on the ovarian side of the occlusion as described by Fincher and Williams[53] and illustrated by Fig. 6. Richter believes that twin or multiple pregnancy does not cause genital hypoplasia in goats. Apparently this is based upon the Keller and Tandler, and

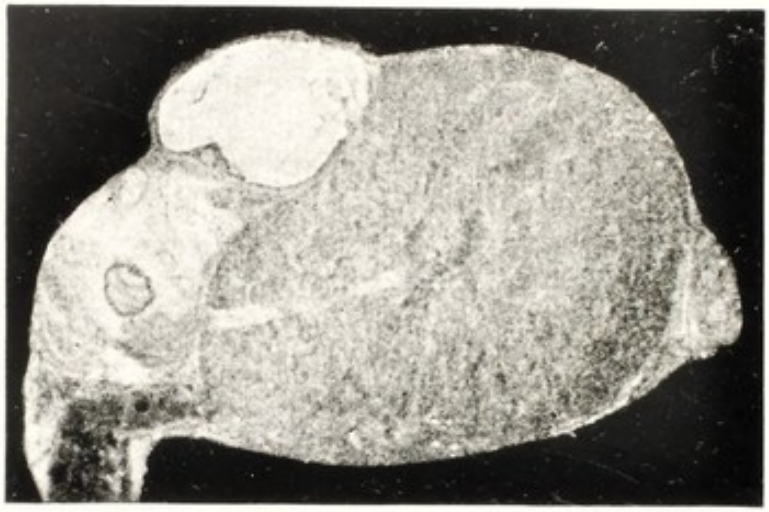


FIG. 113. Sperm Blocking of Vasa Efferentia in Testicles of Goat. (Richter)

Lillie hypothesis regarding bovine free-martins. From my viewpoint, as expressed in Chapter III and illustrated in Fig. 21, twin and multiple pregnancy in sheep and goats often imperil proper nutrition to some fetuses. This may interfere with proper development of the genital organs.

ORCHITIS

Orchitis occurs in all species of domestic animals. The causes are imperfectly known. Prior to the enormous increase in the diseases interfering with reproduction, orchitis was largely ascribed to traumatic injury. I have studied one pair of testicles from a bull in which there were numerous small shot. The presumption was that the bull had trespassed upon the property of an irate neighbor. As a rule both testicles are involved in orchitis. This inferentially denies traumatism as a cause of orchitis except in such cases as gunshot wounds. Abscessation associated with *Bacillus abortus* occasionally affects but one testicle of the bull with sufficient intensity to cause abscess, but as a rule the other testicle is involved to a lesser degree. The character of orchitis varies widely. The most outstanding types may be profitably considered.

ACUTE ORCHITIS

The clinical evidences of acute orchitis are usually well marked. The scrotum is swollen and hot. If the scrotal skin is white or of light color, there is redness. The affected gland is swollen and tender. The peritoneal coverings of the gland are involved and soon become adherent, so that the testicle is not freely moveable within the scrotum. The temperature may be elevated, and the appetite and general vigor depressed.

The course of the disease is rapid and the prognosis is highly unfavorable for the fertility of the patient. It does not, as a rule, imperil the life of the animal. The density of the tunic of the testicle binds the parenchyma so firmly that necrosis is invited. The milder cases usually lead to the degeneration of the seminiferous tubules with permanent loss of spermatogenic power. When abscesses form, they are said to rupture in some types, but in *Bacillus abortus* orchitis of bulls the abscesses are usually chronic and firmly encapsulated.

Orchitis occurs in the course of various contagious diseases. Tubercular orchitis occurs in bulls and boars, as indicated in Fig. 69. Its accurate diagnosis depends upon the section of the diseased gland.

That type of equine influenza known as epizootic cellulitis or "pink eye" is commonly associated with orchitis under conditions which tend to intensify the basic disease. During my private practice in Central Illinois, my clients imported annually from France some hundreds of Percheron stallions. During the long and arduous transportation by ship and train, infectious diseases often spread among them in virulent form. Upon several occasions the epizootic cellulitis raged and upon their arrival at destination almost all animals, in an entire cargo of fifty or more, showed marked clinical evidences of orchitis. When some of the stallions later proved sterile the loss was naturally attributed to the influenza orchitis. At that time little was known concerning the etiology of infectious cellulitis. It is now assumed to be a virus disease, which suggests an interesting analogy to mumps in man, not infrequently complicated with orchitis. While discussing abortion it was noted that mares affected with this form of influenza largely abort. The prevention and handling of the orchitis constitutes part of the therapy of the general disease. Richter believes that the absolute sterility observed in stallions after influenza, and in bulls following foot-and-mouth disease, is perhaps due to occlusion of the *Ducti efferentia,* the final result being like the semen-blocking in young goats.

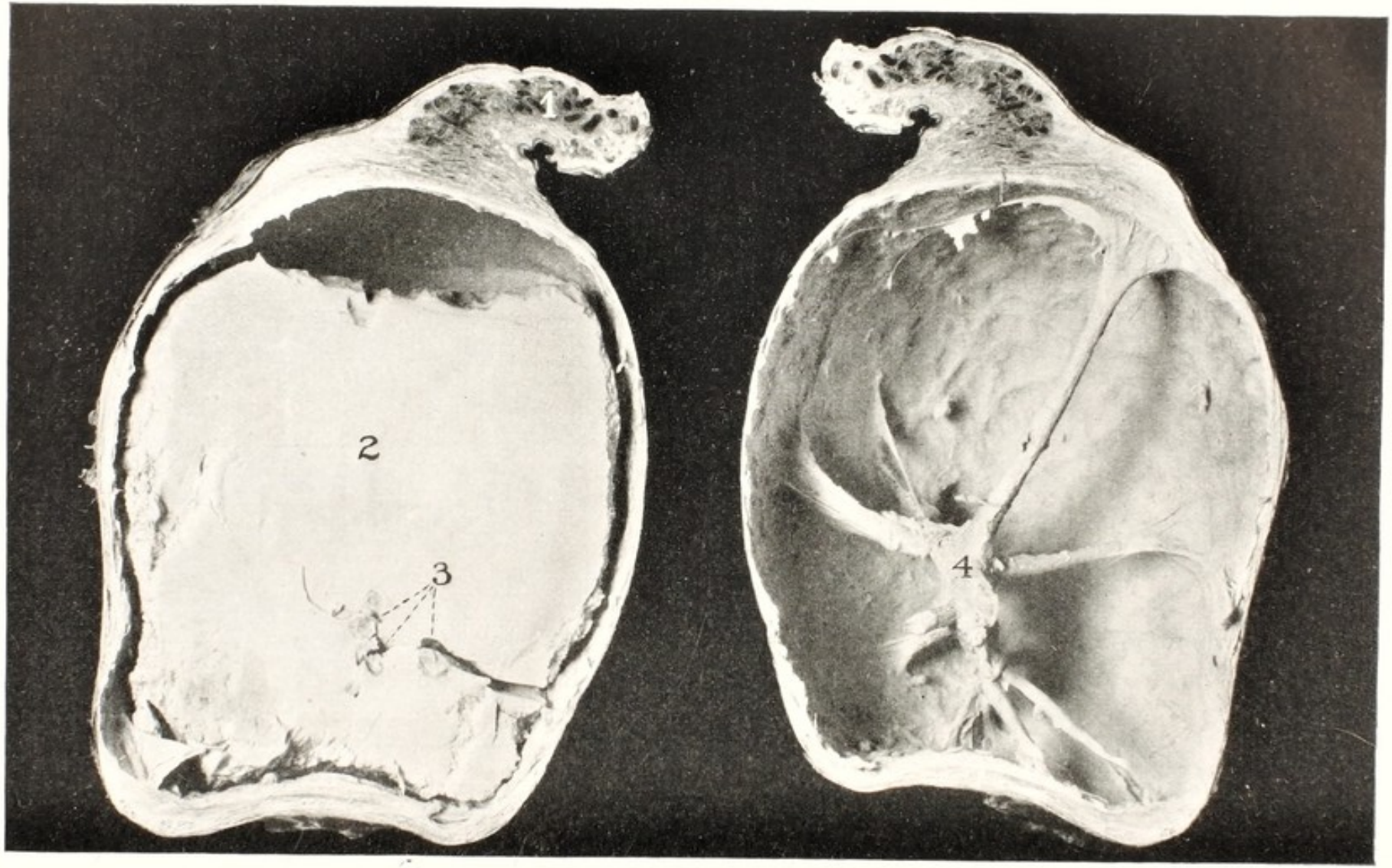


FIG. 114. *Bacillus Abortus* Orchitis of Bull, Resulting in an Abscess Eight Inches in Diameter.
1, Spermatic cord; 2 pus; 3, testicular remnant; 4, fibrous skeleton of testicle remaining after removal of pus.

Bacillus abortus orchitis is not rare in the bull and may occur in other ruminants and in swine. The disease commonly results, in the bull, in abscessation of one testicle. According to the available literature and limited personal observation, the orchitis develops suddenly. The temperature is sharply elevated, the appetite depressed, the scrotum markedly swollen and the affected gland sensitive to pressure. The dense fibrous envelope of the testis usually becomes thickened and the parenchyma necrotic, resulting in an abscess without marked tendency to rupture. If the abscessed testis be removed, the temperature quickly drops, the appetite returns, the general condition becomes normal and the bull readily copulates, usually ineffectually. Although only one gland is generally clinically diseased, the other apparently participates in the disease. Udall, Fincher, and Gibbons[202] report a case of *Bacillus abortus* orchitis in a two-year-old bull. After some months of rest following the attack and when the active disease had abated, the bull was caused to serve repeatedly five cows, two of which eventually conceived and were reported to have calved normally. Upon autopsy the affected testis measured 8 x 4.5 inches in diameter and consisted largely of an enormous fibrous capsule containing pus and detritus. In other cases the fibrous capsule is only moderately thickened and is distended with an enormous volume of pus and glandular detritus (Fig. 114).

Lagerlöf[116] has observed in some cases that the ampulla of the vas deferens of the affected gland is enlarged and sensitive to manipulation per rectum. This is possibly general and has usually been overlooked.

The agglutination test and guinea pig inoculation are positive. The general custom is to consider the diagnosis complete in the presence of these two facts. In some cases, where search has been made, other pyogenic bacteria have been recognized, but neither their frequency nor their significance is known.

The period in life at which *Bacillus abortus* orchitis may originate has not been accurately determined. It has been recorded generally in sexually mature bulls. It is known, however, that aborted fetuses contain that organism in various tissues. There are reasons for inferring that the infection may enter the fetus and persist after birth. The diseased bull calf coming under the observation of Udall, myself, and others[243] and designated as Case 86 was clearly diseased at birth. When he came under our observation at ten weeks of age, he was definitely affected, besides other lesions, with abscessation of the epididymes and vasa defferentia, as indicated in Figs. 115 and 116. Bull No. 2 in Table II was of very low fertility from the first. At

some time prior to investigation he had suffered from abscessation of the seminal vesicles as indicated in Fig. 138, and since such abscesses are commonly attributed to *Bacillus abortus* invasion, the inference is justifiable that the bull was born with that infection, or had some other invader present which lowered his resistance to the abortion organism.

The prevention of *Bacillus abortus* orchitis rests upon the general

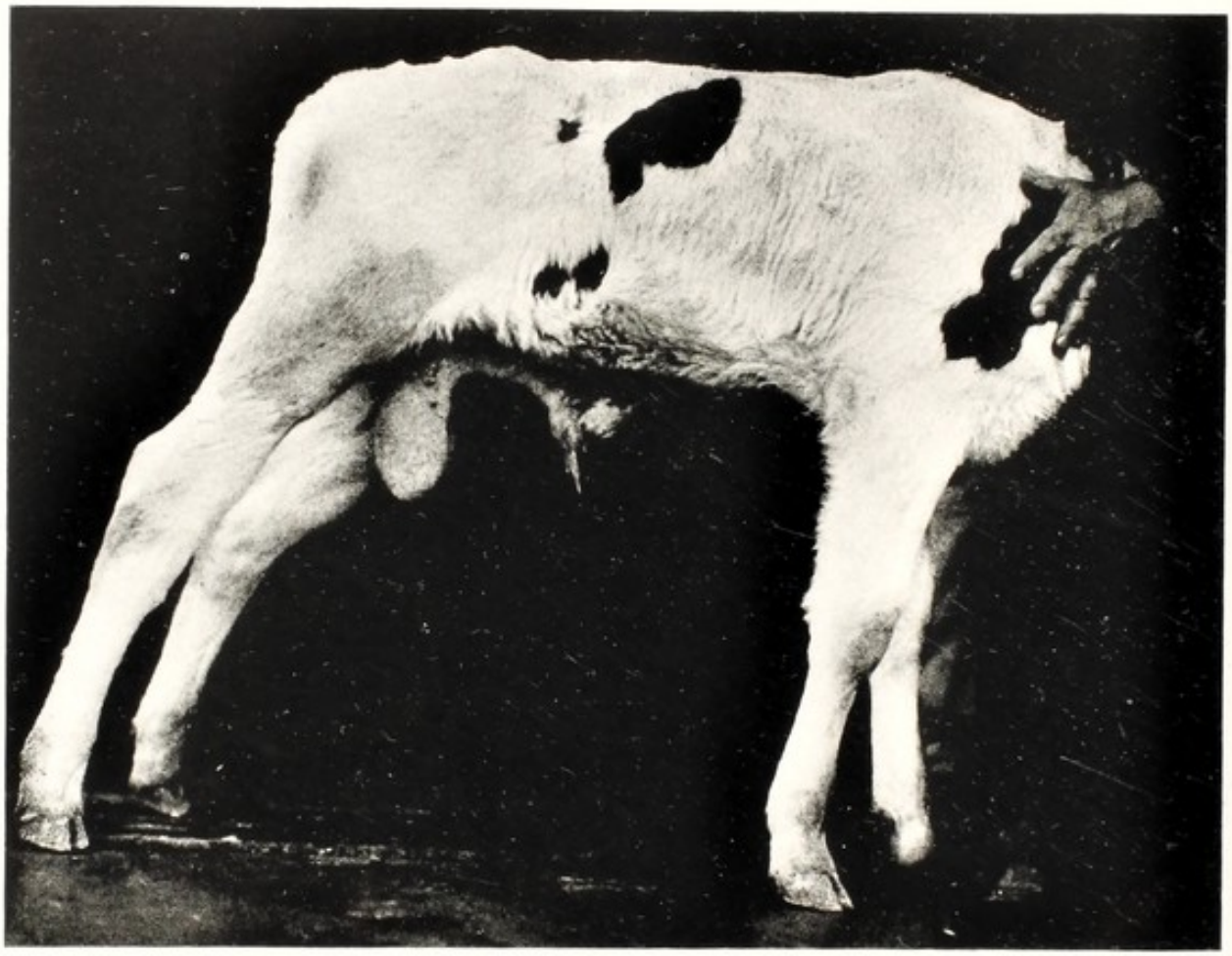


FIG. 115. Congenital Purulent Infection of the Umbilicus and Urino-Genital System of a Calf.

The animal was a firstling out of a sterile heifer bred for over one year before conception. The calf was carried about full term, was born weak and constipated. Photographed at three months. The umbilical area is much soiled by profuse discharge of pus: the enlargement of the scrotum is owing chiefly to abscessation of the epididymes and of the vasa defferentia. (Cornell Veterinarian)

protection of the animal against that infection. Once established, there is no remedy for the restoration of fertility. The recovery of the general health, preparatory to slaughter for food, may be hastened materially by the removal of the diseased testicle.

Hart[81] reports an extensive outbreak of orchitis in a flock of rams. A number of fatalities occurred. In some cases the entire testicle was destroyed. The entire genito-urinary apparatus was involved—

testicles (chiefly), kidneys, bladder, prostate gland, urethra, and penis. Those of the band which had apparently recovered, or which had not shown clinical evidences of the disease, were later used as sires, followed by serious losses because the ratio of lambs dropped

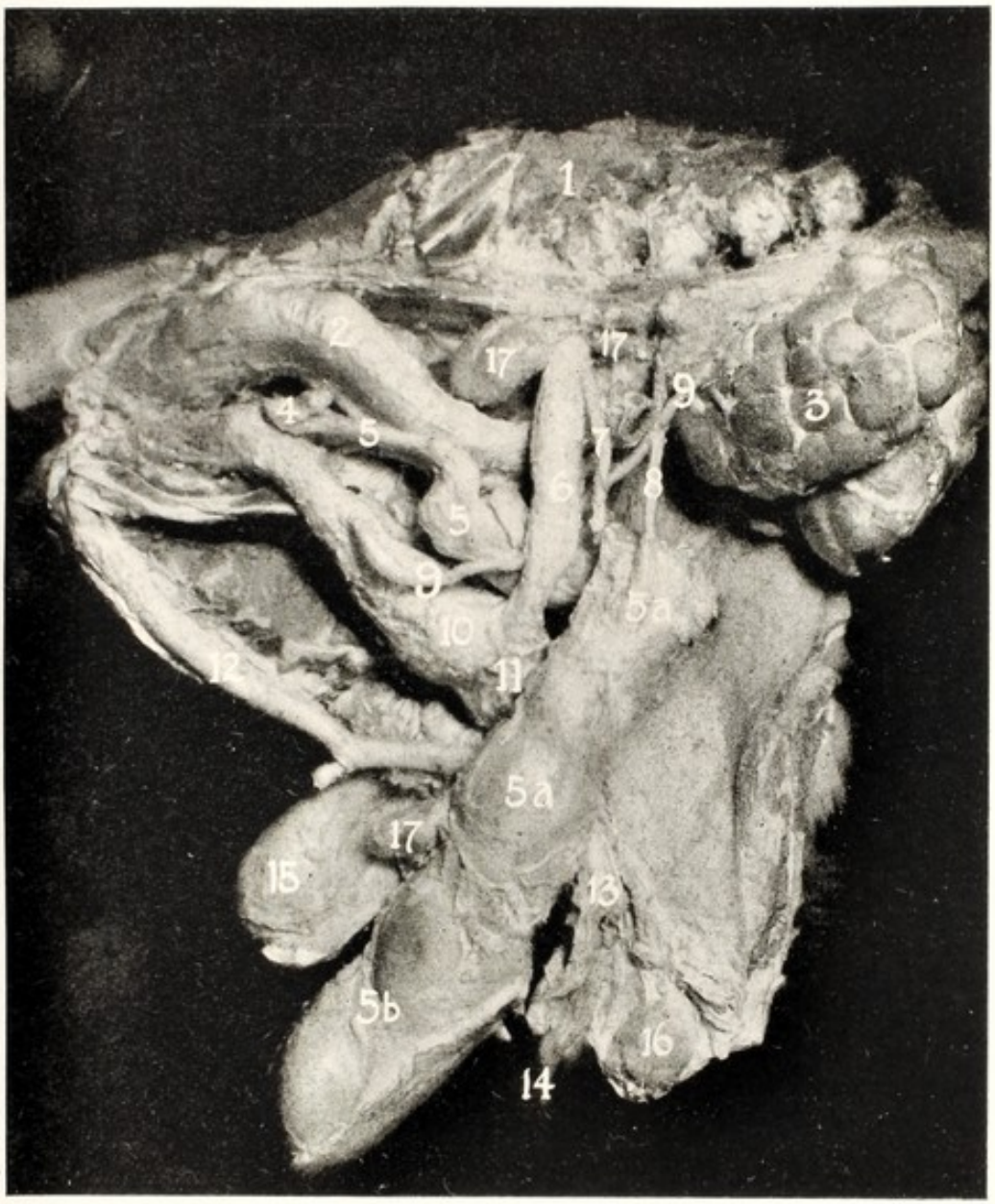


FIG. 116. Section Through the Pelvis of Calf Shown in Fig. 115, Viewed from the Right.

1, Sacrum in sagittal section slightly on the right of the median line; 2, rectum; 3, right kidney; 4, right seminal vesicle, approximately normal in volume; 5, 5, caudal end of right vas deferens, approximately normal in size at left, enlarged and distended with pus at right 5; 5*a*, 5*a*, right spermatic cord, greatly distended by abscess of vas deferens; 5*b*, enlarged right scrotal sac containing abscess of epididymis and vas deferens; 6, right umbilical artery; 7, small right testicular or spermatic artery; 8, main spermatic artery; 9, right ureter; 10, urinary bladder; 11, abscess involving the termini of the urachus and umbilical arteries; 12, penis; 13, sheath; 14, preputial tuft of hairs; 15, left scrotal sac distended by pus in epididymis; 16, umbilical abscess; 17, lymph glands.

Upon section, both testicles appeared macroscopically intact. Had the animal survived they would probably have atrophied or undergone abscessation.

to about 33 per cent of the anticipated. It is not stated that the ewes suffered from the malady.

Wallraff[215] records an outbreak of purulent orchitis in horses, cattle, and goats. The disease had existed in his locality in Freudenstadt for a number of years. At least it was known in 1832, fourteen years prior to the date of his contribution. For a year prior to his report, the disease had been common and caused very considerable losses among breeding bulls. It involved animals of all ages, attacking many calves when only 10 to 20 days old. The disease began with light fever and loss of appetite. The animals stamped their hind feet as if afflicted with colic. Presently the testicles became more or less swol-

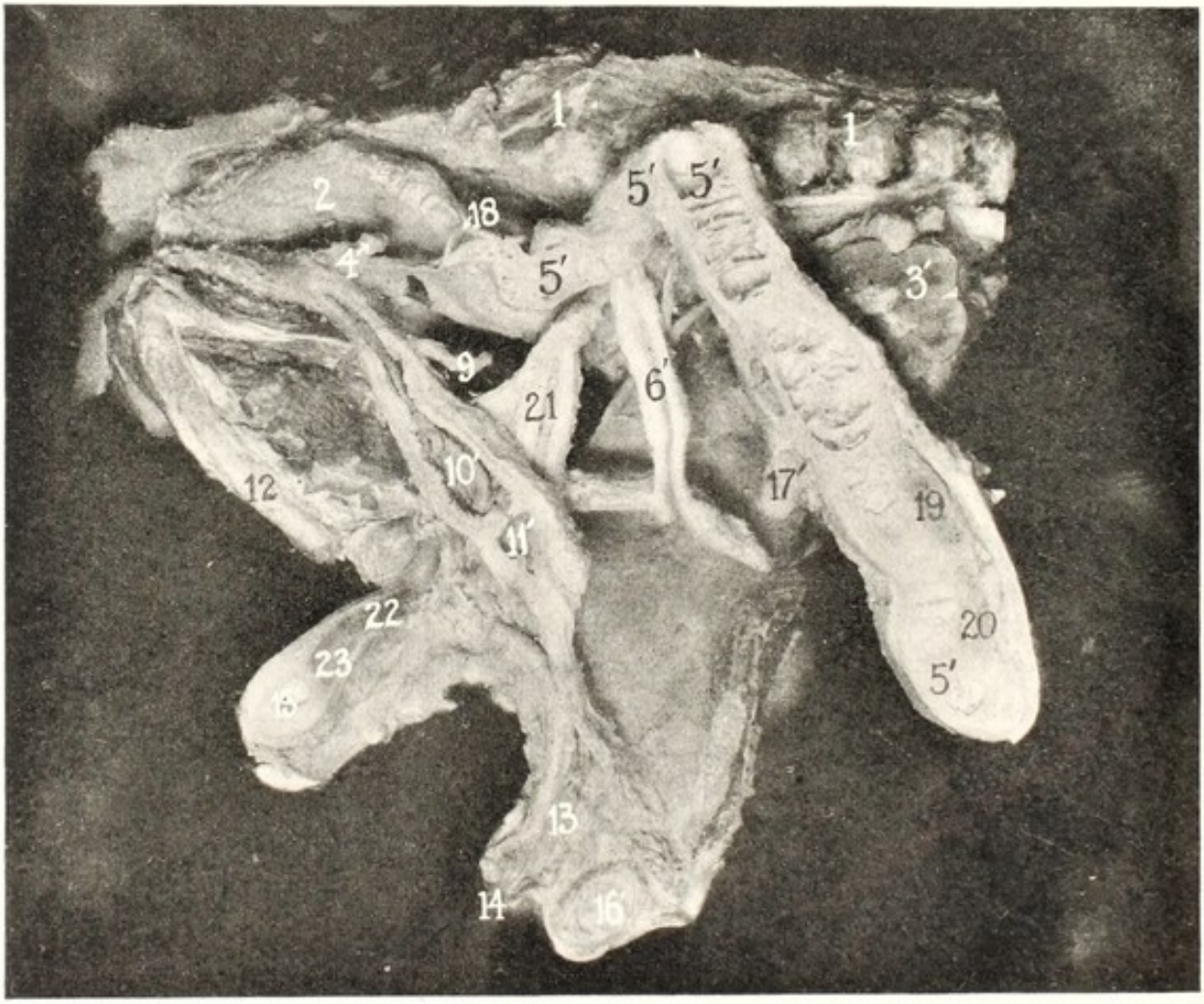


FIG. 117. Same as 116, with most of the organs in sagittal section. They are marked as in 116 with (′) added. The right ureter has been severed, and right umbilical artery and right vas deferens and testicle displaced forwards in order to expose underlying parts. 18, A section from the normal left vas deferens placed upon the abscessed right organ as check; 19, right spermatic blood vessels; 20, right testicle in sagittal section; 21, right umbilical artery in sagittal section showing a fibrinous cast in its lumen. It will be seen that the lumen of the right umbilic artery, 6′, is empty. Apparently it had contained an identical cast which had become dislodged and passed into the artero-urachal abscess, 11′, thence through the urachus, into the urinary bladder, 10′; 22, vascular mass of left spermatic vessels; 23, left testicle.

len, the scrotum red, hot, and painful upon manipulation. This condition continued for three to five days. Then, upon one or both sides of the scrotum, a small opening appeared, from which at first a stinking discharge, mixed with blood, escaped. After one of two days

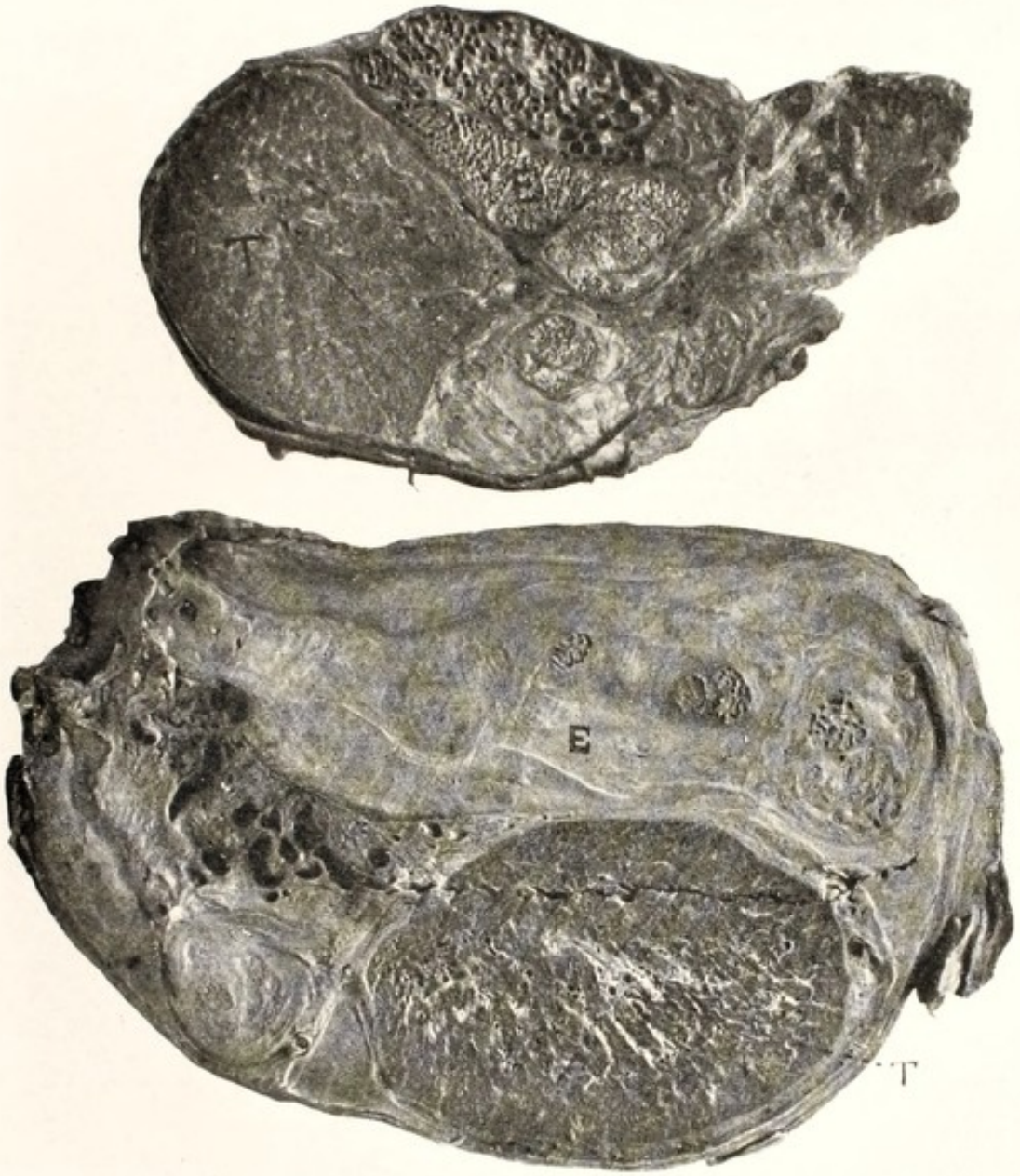


FIG. 118. Equine Orchitis and Epididymitis.
T, Testicle; *E*, epididymis.

this discharge was superseded by ordinary pus. This continued for five to fourteen days, until the testicle was wholly destroyed. Wallraff uniformly observed the swelling and suppuration in the left testicle first. As a general rule, after one testicle was destroyed, the other also underwent destruction later, though in some cases one of the testicles escaped.

The disease had little danger for the life of the animal. After the

destruction of the testicle, the animal recovered without any very great enlargement of the scrotum. In one case, when the abscess in the testicle ruptured, the bull almost bled to death. Wallraff failed to recognize the cause. For a time he thought it was hereditary, and continued to believe that it might be so.

While there seemed to be no great danger to the life of the patient,



FIG. 119. Orchitis and Epididymitis in Stallion.

1, Epididymis; 2, testicular tissue; 3, hemorrhage in the epididymis; 4, hemorrhage in the testicle.

there was very serious loss in condition and also, in all or nearly all the cases, the breeding life of the animal was brought to a close. Consequently, as a rule, when the disease was observed early, the animal was sent to the butcher as the most economic course. Once the disease was established, castration apparently proved the most direct method of handling.

The occurrence of enzootic orchitis as recorded by Hart and Wallraff injects into the problem interesting questions. Wallroff was puzzled owing to the peculiar type of the orchitis and to the fact that it was confined to a limited area about Freudenstadt (Baden). Animals sent out of Freudenstadt in good health remained well: those imported into Freudenstadt became diseased. The cause was not discovered. Similarly, the orchitis studied by Hart is not known to occur

outside a small area in California. Hart found *Bacillus pyogenes* in the pus, cultures of which injected into the testicles of healthy rams caused parallel lesions. *Bacillus pyogenes* is relatively common, but is not known to cause enzootic orchitis in other species or in other areas.

Orchitis in the stallion does not tend to the formation of an abscess, in which respect it is unlike the orchitis of the bull. The scrotal walls in the stallion are much thinner than those of the bull, and palpation

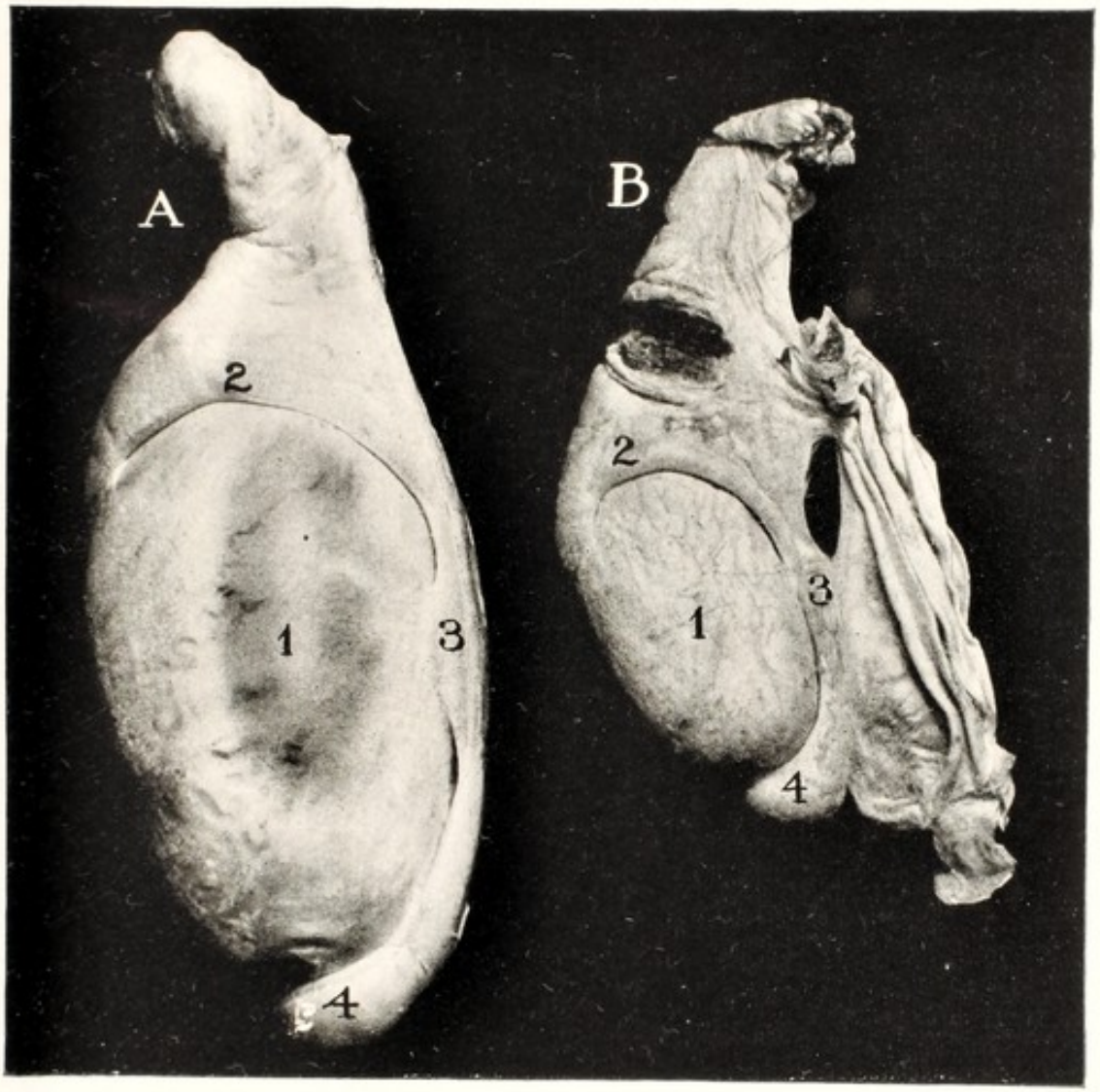


FIG. 120 Degenerative Orchitis in a Young Bull.

A, Normal testicle as check; B, advanced degeneration of testicle of sterile bull. 1, Testicle; 2, head of epididymis; 3, body of epididymis; 4, tail of epididymis.

of the testicles is thereby facilitated. The glands become adherent in the scrotum quite early in the disease. The epididymis generally participates in the disease and its comparative abundance of connective tissue permits it to swell more freely than the gland proper. With the adhesions, the enlargements of the testicle and its epididymis become blended in such a manner that it may become difficult or impossible to differentiate the two structures by clinical examination,

as may be understood by studying Figs. 118 and 119. The volume of the testicles proper is much reduced in these cases, while the size of the epididymes is greatly increased. Except in the cases of influenza orchitis mentioned, the causes of orchitis in stallions are usually unknown.

CHRONIC ORCHITIS

Chronic orchitis is most often observed in stallions, bulls, and goats. It may appear at any time after the advent of puberty. It is sometimes present in young bulls when they arrive at breeding age. In bulls chronic orchitis is not usually ascribed to *Bacillus abortus,* as is the case with acute orchitis. It seems not improbable that the testicular hypoplasia, already described, constitutes the most important and common cause of its appearance. A male having mild testicular hypoplasia may be fertile when first placed in breeding. With many seminiferous tubules so hypoplastic that they cannot produce spermatozoa, the other tubules are logically subnormal in function. When the animal is placed in breeding, the glands cannot safely carry the expected load. The dairy bull and the male goat have been artificially fed when young and the normal development of the reproductive system repressed.

Overbreeding certainly endangers the integrity of the testicles and tends constantly to cause a low type of chronic inflammation of the glands. This is quite spectacular in the case of many young bulls placed under high pressure in hand breeding, and even more notable when bulls of one or two years are used in pasture breeding. Again at the other pole of reproductive life, as age creeps upon the sire, he may have acquired fame through his get and it is desired to obtain the greatest number of progeny possible. So in the stallion illustrated in Fig. 121, and his semen in Fig. 28, when he had become famous, he was apparently overbred, and became dangerous, when, with conservative use, he might have continued valuable.

The clinical symptoms are not always clear. In Figs. 118 to 121 the glands are definitely atrophied without marked enlargement of the epididymes. There may be, as in Fig. 119, quite definite sclerosis of the tunica albuginea and an increase in the connective tissue between the seminiferous tubules. Adhesions between the two peritoneal tunics may be absent or very slight. Such testicles are palpably hardened and atrophied. Sometimes, as in Fig. 121, the testicles are atrophied, and the epididymes enlarged, with the peritoneal coverings adherent, obscuring the outlines of the structures and making it difficult or impossible for the examiner to distinguish between testicle and epididymis.

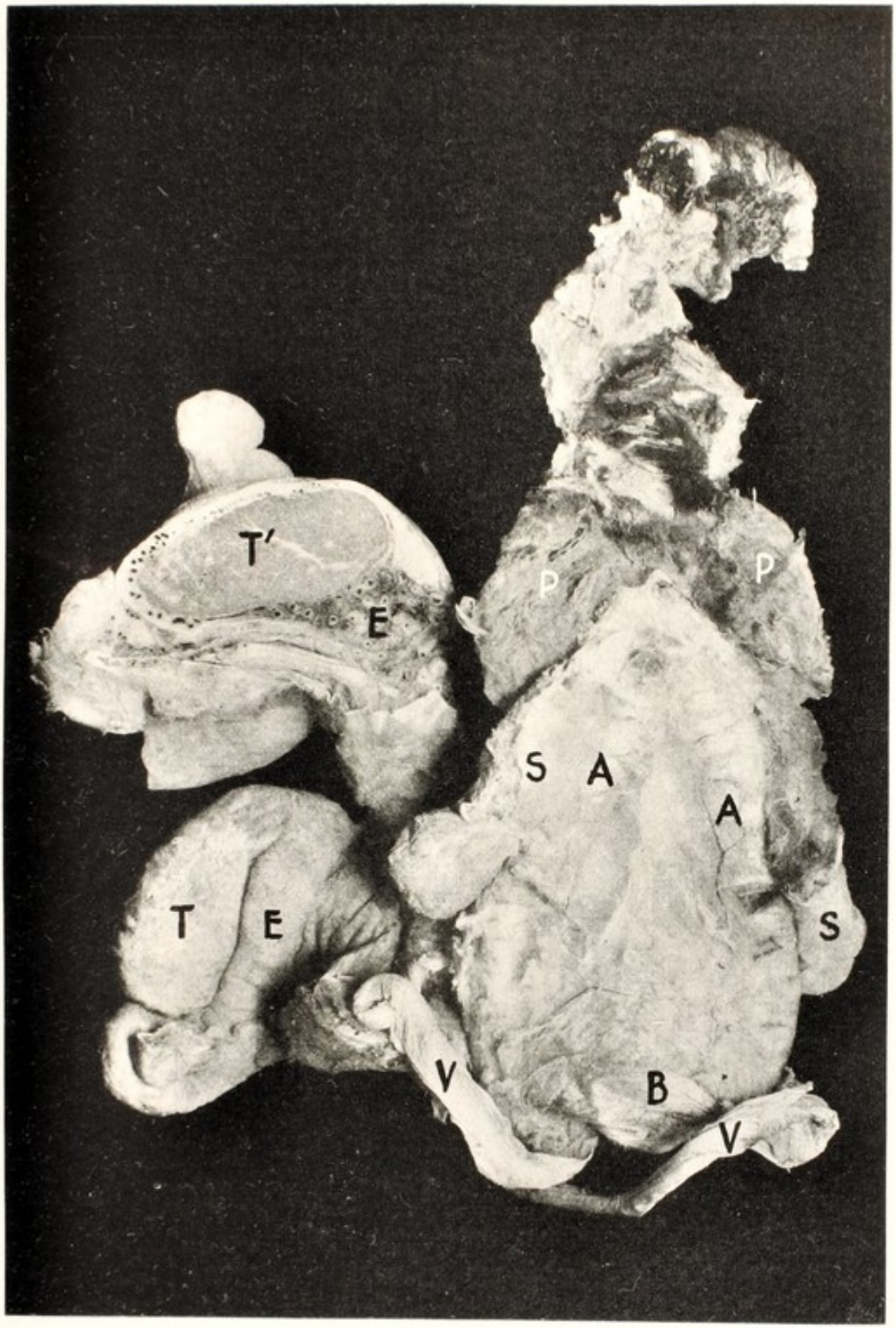


FIG. 121. Genital Organs from Diseased Stallion, Same Animal as Stallion 1 in Table III.

His spermatozoa are shown in Fig. 28 and the ampulla of his vas deferens in Fig. 131. A, A, Ampullae of vasa defferentia; *B,* cephalic end of urinary bladder; *V, V,* vasa defferentia; *E,* epididymis; *E',* section through epididymis; *T,* testicle; *T', sagittal* section through testicle; *S, S,* seminal vesicles; *P, P,* prostate glands. *(Cornell Veterinarian)*

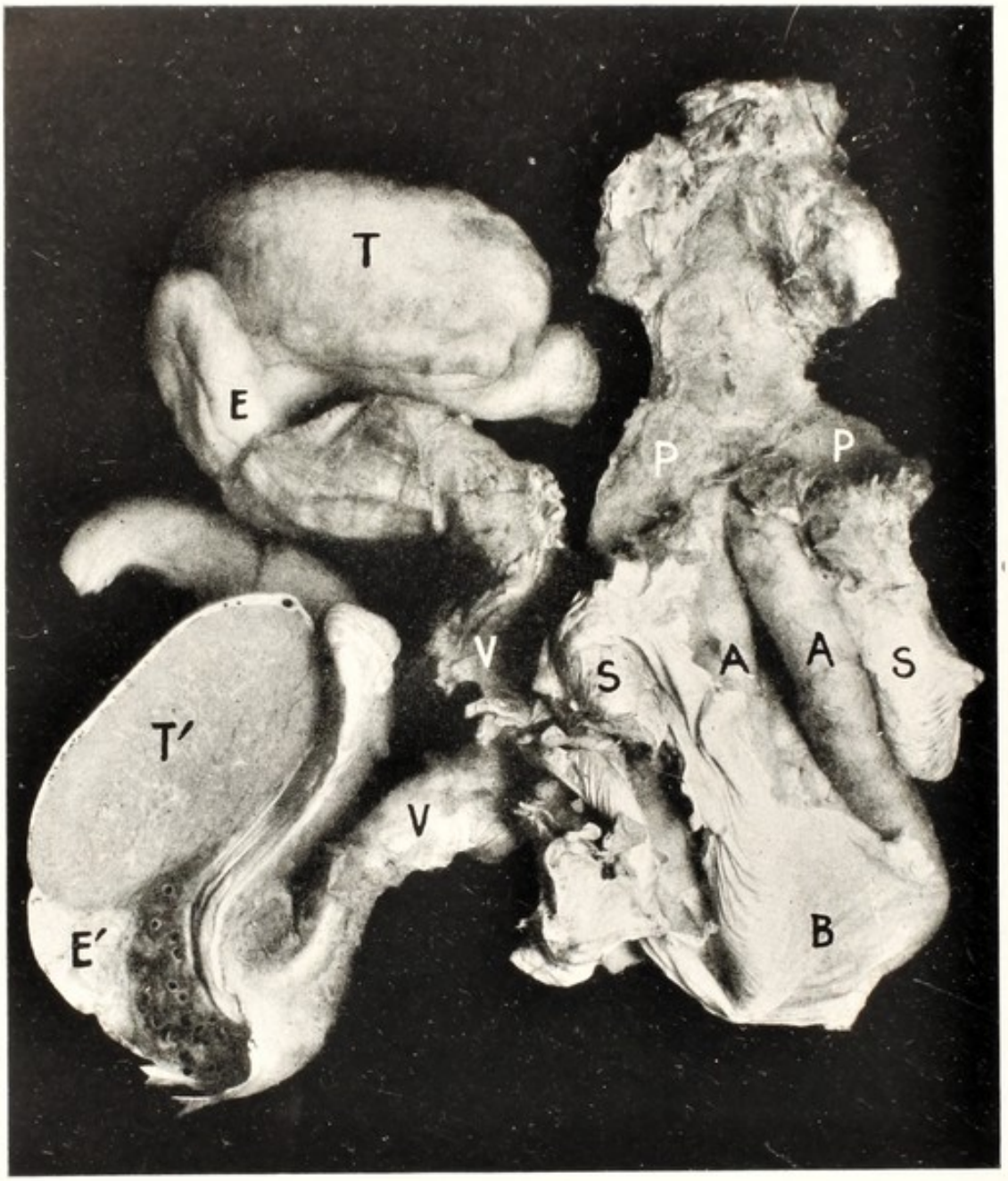


FIG. 122. Healthy Genital Tract of Stallion for Comparison With Fig. 120: lettering as in preceding. *(Cornell Veterinarian)*

Sometimes, as in Figs. 119 and 121, chronic orchitis is associated prominently with inflammation of the ampullae of the vasa deferentia and seminal vesicles. Such association is ample evidence of the presence of infection. The identity of the invader is not generally determined.

Further evidence of chronic orchitis is available by a study of the semen. In extreme cases no spermatozoa may be ejaculated. The stallion whose testicle is illustrated in Fig. 121 ejaculated a few very defective spermatozoa, as shown in Fig. 28. Not many months prior to his destruction he was of very low fertility, as shown in Table III. The defects in the spermatozoa do not reveal authentically the precise

character of testicular disease; the faulty morphology may have come from orchitis, from testicular hypoplasia or from degeneration of the tubuli seminiferi from undetermined causes.

Histologically the epithelium of the tubuli is variously damaged. All layers may be involved and the lumen may be narrowed or destroyed. Round cell infiltration occurs in the interstitial tissues and there may be important increase in the connective tissue.



FIG. 123. Chronic Orchitis in Aged Bull. *Same animal as Figs. 102 and 137.*

A, Left testicle; *B*, normal gland as check; *C*, right testicle. 1, Globus major of epididymis, greatly enlarged and closely adherent to testicle; 2, body of epididymis; 3, testicle; 4, tail of epididymis; 5, 6, 7, (*B*) globus major, body and globus minor respectively of healthy testicle; 8, globus major inflamed and enlarged; 9, parietal peritoneum of scrotum, firmly adherent to the testicle, which has been dissected away and turned back. *(Cornell Veterinarian)*

Richter[166] describes the frequent occurrence of calcareous degeneration of the seminiferous tubules in the testicles of sterile goats, as illustrated in Fig. 124. Webster[222] records that among 187 abattoir bulls studied in New Zealand, 31 per cent showed calcification of the tubuli seminiferi.

When chronic orchitis has become definitely established there is no known method of handling by which satisfactory fertility may be

restored. If diagnosed early, which is not usual, sexual rest with prudent feeding and handling may halt or ameliorate the disease. Castration tends to hasten the restoration of physical health.

TESTICULAR DEGENERATION

DEGENERATION OF THE TUBULI SEMINIFERI

In the preceding sections it has been noted that, in diseases of the testicles which do not lead to total necrosis, changes occur which disturb or destroy the production of spermatozoa. These have been attributed to the invasion of the glands by infective forces which

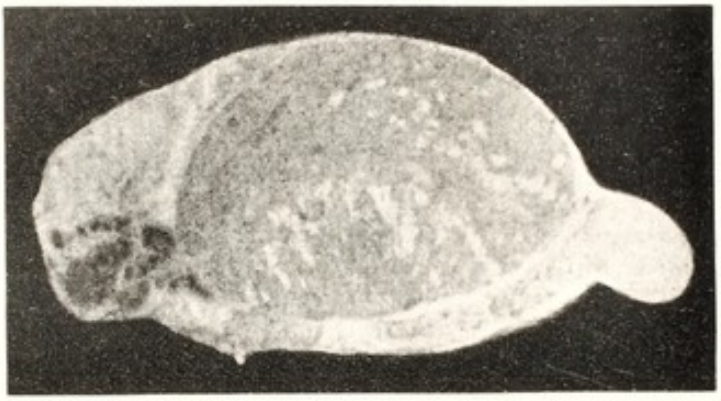


FIG. 124. Calcareous Degeneration of Testicle of Goat. (Richter)

have led to changes in the epithelium of the tubules and inevitably of the sperm cells which they produce. There are other cases in which the seminiferous tubules lose their power to produce physiological spermatozoa, the precise cause of which is unknown. There is not known to have been a prior invasion of the glands by bacteria, protozoa or viruses. It seems best to classify these as *testicular degeneration*. Some of the forces causing such degeneration have been determined. Roentgen rays applied to the testicles depress or destroy their power to create spermatozoa.

Lagerlöf[114] remarks with reason that, of all important organs in the body, the testicles are the most sensitive to their environment, and that their function of spermatogenesis is repressed or interrupted for apparently very trivial reasons. He has well shown that by the insulation of the scrotum of the bull in a manner to raise the temperature nearly to that of the rectum, the fertility of the animal is promptly depressed and soon wholly suspended; the spermatozoa produced are defective morphologically, in motility and in all features characteristic of physiological sperms, as is indicated in Fig. 125. The degeneration proceeded rapidly from the normal at *a*, to *b*, *c*, and *d*, at six, seventeen, and eighteen days respectively after the in-

sulation on alternate days during seven days, or a total insulation of ninety-six hours. When one testicle was removed at eighteen days, degeneration of the tubuli seminiferi was well marked. Fifty days later, when the bull was slaughtered, the remaining testicle had recovered almost completely and was producing good spermatozoa. At the same date Phillips and McKenzie,[156] pursuing similar experiments upon the ram, produced parallel results. They submitted evidence which they believed showed that the dartos serves an important purpose by contracting or relaxing according to the atmospheric temperature, causing the testicles to be drawn up closely against the body in cold weather or drop away during hot weather, thus serving to regulate the temperature of the glands.

The testicles logically obey the general law that overwork causes fatigue, and eventually degeneration. In reproduction the overexertion may be either sexual or physical. Race horses are not normally fertile while on the turf. After their retirement to the stud they display low fertility for some months. Sometimes the stallion is wholly sterile when taken out of training and placed in the stud, and it may be difficult to determine whether the sterility be due to severe training, to influenza, or other causes.

Excessive sexual load may render any sire impotent or greatly lower his fertility. Holding a male out of breeding for a long period of time does not cause a notable lowering of fertility. The causes of testicular degeneration are largely unknown. I observed a thoroughbred stallion which had been highly fertile for some years. Located in the tropics, he was allowed a commodious paddock with abundant exercise throughout the year. Suddenly, without having shown signs of disease, he was absolutely sterile at the opening of the breeding season. After he had served some thirty mares, all of which returned, I examined his spermatozoa, which were abundant and all morphologically defective. The mares all conceived when bred to another sire.

Too little is known, regarding the basic causes of degeneration of the seminiferous tubules, to enable the practitioner to make a thoroughly accurate diagnosis as correlated with the cause, or to give a dependable prognosis. Lagerlöf removed one testicle from a bull after the glands had been subjected to the degenerating influence of scrotal insulation and the animal had become impotent, his spermatozoa largely non-motile and morphologically defective. Histologically the testicles had undergone important degenerative changes in the epithelium of the tubuli seminiferi. After the one testicle had been removed and the scrotal insulation withdrawn, regeneration gradually occurred, as shown in Fig. 125, and the bull again became fertile.

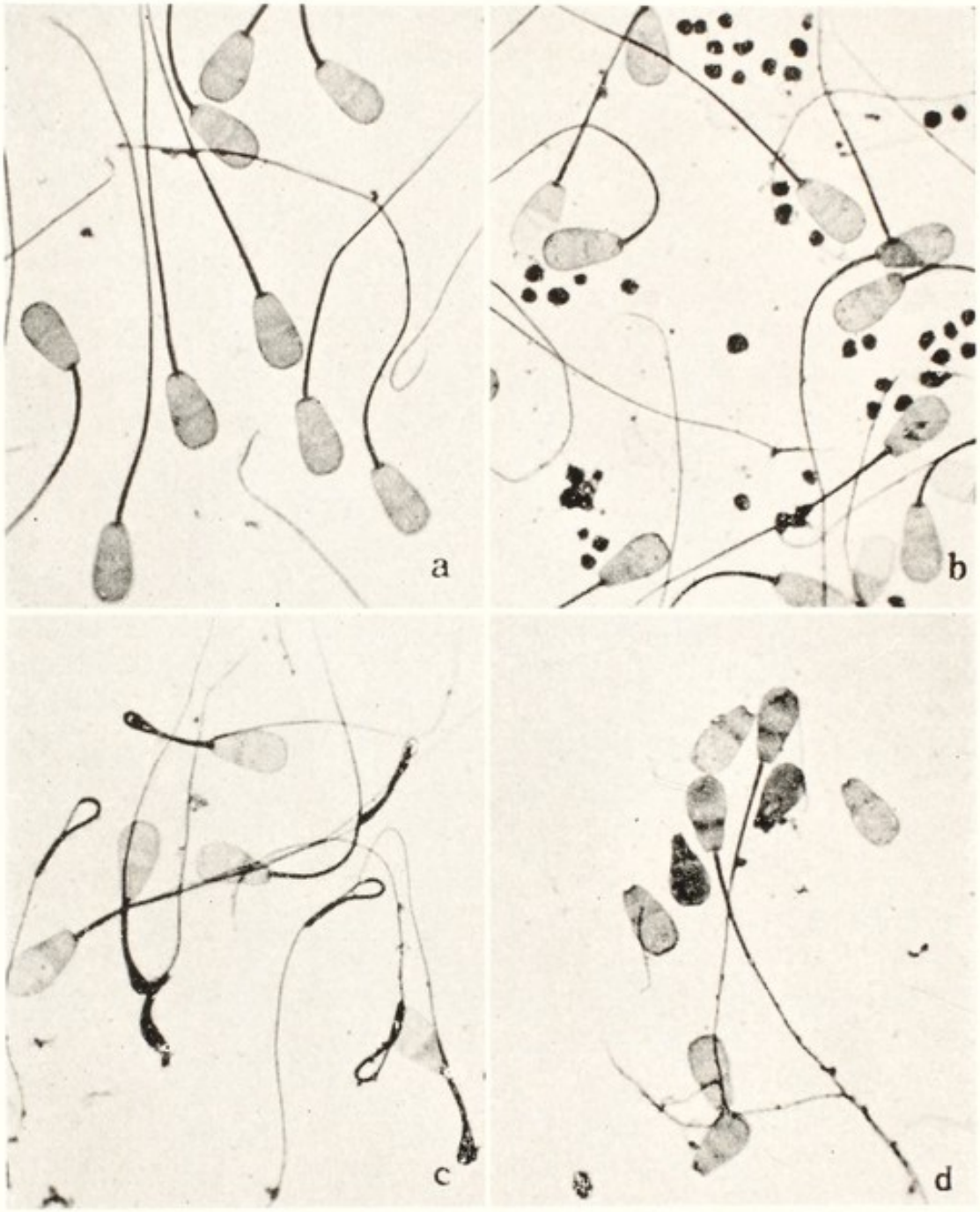


FIG. 125. Spermatozoa of Bull Subjected to Experimental Degeneration of the Testicles by Scrotal Insulation.

a, Normal spermatozoa before beginning experiment; *b*, six days after beginning of experiment *(Note the abundance of free protoplasmic droplets)*; *c*, after 11 days; and *d*, after 12 days. (Lagerlöf)

This justifies the assumption that, if the basic layer of the epithelium of the tubuli seminiferi remains intact, the damaged gland may recover. The recoveries are apparently limited to cases in which the testicles have retained approximately their normal volume. If the glands have become definitely atrophied, there seems no good basis for a favorable prognosis. Generally, so far as the literature indicates, the outlook for recovery is not good.

The Pathology of the Semen and of the Spermatozoa

Any testicular defect or disease modifies the quantity and quality of the semen and its spermatozoa.

The study of the semen and spermatozoa constitutes the most accurate basis known for the determination of the reproductive efficiency of the male before the final test upon a sufficiently large group of females involving one or more breeding seasons. The one known important exception is the trichomonad disease of cattle. Apparently this protozoan does not invade the testicles, at least in the early stages of the disease, in such a manner that the spermatozoa are damaged, nor do we have evidence that such protozoa attack the spermatozoa during their passage through the epididymis or the vas deferens. Instead the protozoa apparently attack and destroy the fertilized ovum prior to the recognition of pregnancy, and the phenomenon is classed as sterility, or it later causes the death of the recognized embryo and is called abortion. Those infections designated as *unclassified* and including the nodular venereal disease of ruminants and swine and the venereal tumors of dogs are not known to invade the testicles and injure the spermatozoa. These constitute a minority of offenders in the realm of reproduction.

The semen for study may be obtained by any of the methods already mentioned while discussing artificial insemination. It is usually most conveniently obtained by mating the sire with a female and obtaining the semen from the vagina.

The volume of semen discharged at coitus by a stallion varies between 50 and 150 cc. That of the bull varies widely according to various observers; it may be as low as 1 to 2 cc. or as high as 20 cc. The size and age of the bull are not usually recorded. Lagerlöf places the usual amount at 2 to 5 cc. In the first edition of this treatise it was placed at 6 to 8 cc. Probably most examinations of the semen of bulls are made upon young, partly grown animals, in which a small volume is to be expected.

The spermatozoa are densely packed in the vas deferens, and at coitus are diluted by the liquid secretions from the seminal vesicles, prostate, and Cowper's glands. These latter constitute the major volume of the ejaculate.

Healthy semen is opaque white, as indicated at 1, Fig. 110. Semen from bulls suffering from extreme testicular hypoplasia, and hence consisting almost wholly of the secretions from the seminal vesicles, is usually very thin, watery, and more or less transparent, as in 2 and 3 of Fig. 110. When purulent disease of the seminal vesicles is

present and the pus is discharged as a part of the semen, the ejaculate becomes coagulated irregularly as in 4 and 5.

Testicles suffering from hypoplasia, orchitis or degeneration sometimes fail to produce spermatozoa. Usually they produce a subnormal number with a notably high ratio of morphologically defective sperms. Morphological defects become established prior to the detachment of the spermatozoa from the epithelial membrane of the tubuli seminiferi. Disintegration of the spermatozoa may occur later in the epididymis or vas deferens, but definitely defective morphology, double heads, immature cells with protoplasmic nodules on the body, coiled tails, narrow heads, etc., are formed before the cells become detached from the spermatogenic membrane. It has been believed by some that disintegration of normal spermatozoa sometimes takes place in the ampulla of the healthy vas deferens when the male has been sexually inactive for a prolonged period. Unfortunately it has not been made clear that the males studied were ideally healthy. When the ampulla of the vas deferens is the seat of disease, spermatozoa detained therein for a considerable period would logically undergo disintegration.

We are not greatly concerned at this point with those males which fail to produce or to ejaculate spermatozoa. They are readily eliminated. The earlier writers placed essentially all emphasis, when spermatozoa were ejaculated, upon their abundance and motility. Undeniably a male which ejaculates only non-motile spermatozoa is absolutely sterile, and no further discussion is needful. Others have placed much emphasis upon the ratio of motile spermatozoa to the total number present. While this procedure admittedly has value, it becomes superfluous, so far as known, if a study of the stained spermatozoa is applied. Non-motile or otherwise badly diseased spermatozoa do not have affinity for differential stains. Hence, when properly stained, the non-motile cells and those having other perilous defects are clearly revealed, as in Figs. 28, 111, and 126.

For the study of stained spermatozoa it is preferable that the staining be done immediately after the semen is obtained from the male. A drop of the semen is placed upon a slide and, with a second slide held delicately at an angle of about 45 degrees, the liquid is thinly and evenly spread over the surface. This enables the examiner to find in all cases some areas in which the spermatozoa are favorably frequent. The chief danger lies in the smear being too heavy so that the sperm cells are heaped together too densely.

As soon as the smear has been made, it should be thoroughly dried, either in the open air or at room temperature or, should con-

ditions render it desirable, the drying may be hastened by moderate heat—100 to 150 degrees F. The staining should then follow without delay.

If for any reason the semen is to be retained for a considerable period before the sperms are to be stained, it is far best to postpone making the smear, and instead place the semen in a vial with thymol in sufficient quantity to prevent decomposition. It may thus be retained for an indefinite period, when the smears may be made and stained.

Various stains have been recommended, which it seems quite superfluous to describe, since the interested reader doubtless has command of the principles involved. The staining technic introduced by W. W. Williams[266] especially for the spermatozoa of the bull has been fully endorsed by Lagerlöf and other leading workers. The primary stain consists of a mixture of eosin and carbol-fuchsin (1) followed by methylene blue (2) as a counterstain.

STAIN 1.

Saturated alcoholic solution of eosin (bluish)	1 mil.
Ziehl-Neelsen carbol-fuchsin*	2 mil.
Alcohol 95%	1 mil.

* The Ziehl-Neelsen carbol-fuchsin is made as directed by Mallory and Wright, Pathological Technique.

Mix the eosin and carbol-fuchsin in a dry watch glass. Immediately on mixing, there form an insoluble precipitate and a metallic film. Filter the mixture and add to the filtrate the 1 mil. of 95% alcohol. This stain loses its efficiency after two or three days.

STAIN 2.

Loeffler's methylene blue	1 part
Distilled water	4 parts

After having made the smear of semen and dried it, immerse the slide in a 0.5% solution of chlorazene (chloramine-T or sodium paratoluene-sulphochloramine) for 5 to 7 minutes to dissolve any mucus present. Wash film gently with water and then with 95% alcohol.

Apply stain 1 for 3.5 to 4 minutes. During this period a precipitate tends to form in the staining solution. This is to be prevented by the addition of more of the stain from time to time, with a dropper, permitting the excess to run off. Wash the film by immersing in a vessel of water and moving gently.

Counterstain with stain 2 for 8 seconds. Wash the slide in distilled water and dry.

After locating a favorable field under low magnification, the study



FIG. 126. Spermatozoa from two genitally diseased bulls designated by Williams and Savage[266] as cases 242 (1-6 incl.) and 63 (7-12 incl.). The conception rate of the cows served by them was extremely low and the abortion prevalence in the few pregnant cows was destructively high.

of the stained sperms is best made by means of moderately high power with oil immersion.

Each portion of the spermatozoon presents an unlimited variety of defects. As in the embryo, so in the spermatozoon there are numerous teratological defects, such as double heads with one tail, a single head with two tails and other types of defect which indicate teratology rather than pathology. It is not known, and there is no good reason to infer, that such distinctly teratological sperms ever fertilize ova. Like most definite monsters they are probably infertile. There occur in the literature a few recorded instances in which a sire begot an extraordinary ratio of monsters, but there has been no authentic linkage of this phenomenon with teratological spermatozoa. The conclusion is quite justifiable, however, that, as in monstrous young, the prevalence of teratological sperms in the semen of a male constitutes evidence that the spermatogenesis is faulty and that the presence of the monstrous cells constitutes according to their frequency a badge of inferiority, and should be regarded as having the same significance as pathological sperms.

The tail of the spermatozoon is essential to the migration of the cell through the uterus and oviduct, to meet the ovum when discharged from the ovary and fertilize it. The head of the spermatozoon penetrates the ovum and the tail and middle piece are discarded. Unless the tail and middle piece be perfect, fertilization is not probable.

There is frequently observed in the spermatozoa of bulls a spheroidal mass or ring of protoplasm surrounding the neck of the spermatozoon, as shown on the right in Fig. 127. The body is apparently normal during the earlier stages of spermatogenesis but should physiologically disappear before the spermatozoon becomes freed from the epithelium of the tubuli seminiferi. Accordingly it constitutes an arrest in development of the cell, to which Lagerlöf applies the designation of *unripe*. As is well shown in Fig. 127 they frequently move caudally and become lodged for a time at the juncture of the body and tail. Less commonly they are observed surrounding

---

1. a, Thickened middle-piece; head with narrow nucleus; 2, enlarged fused anterior and posterior end knobs and end rings, filiform middle-piece; 3. f, filiform middle-piece; e, eosinophilic body surrounding anterior end of middle-piece; 4. s, spheroid swelling at middle of middle-piece within sheath; 5. s, spheroid swelling of end ring; 6. e, acidophile body attached to middle of middle-piece; 7. abaxial middle-piece; 8. x, abaxial middle-piece with tail on one side and abaxial rudimentary neck and body on the other; 9. x, abaxial neck; s, enlargement of posterior end knob; 10. types of arrested nuclear development; 11. types of arrested nuclear development, z, note enlargement of anterior end knob; 12. e, eosinophilic bodies attached to anterior end of body.

the tail, and are often seen lying free in the semen. The same defect is not rare in equine spermatozoa, as shown in Fig. 128*b*.

Such spermatozoa are often, if not usually motile, but all evidence indicates that they are impotent to fertilize. As a rule this defect does not occur alone: the heads are not uniform in their contour and they do not take stains like the physiological cells, and detached heads and other defects are numerous. This arrest in development of the male cells is almost constant in testicular hypoplasia, is com-

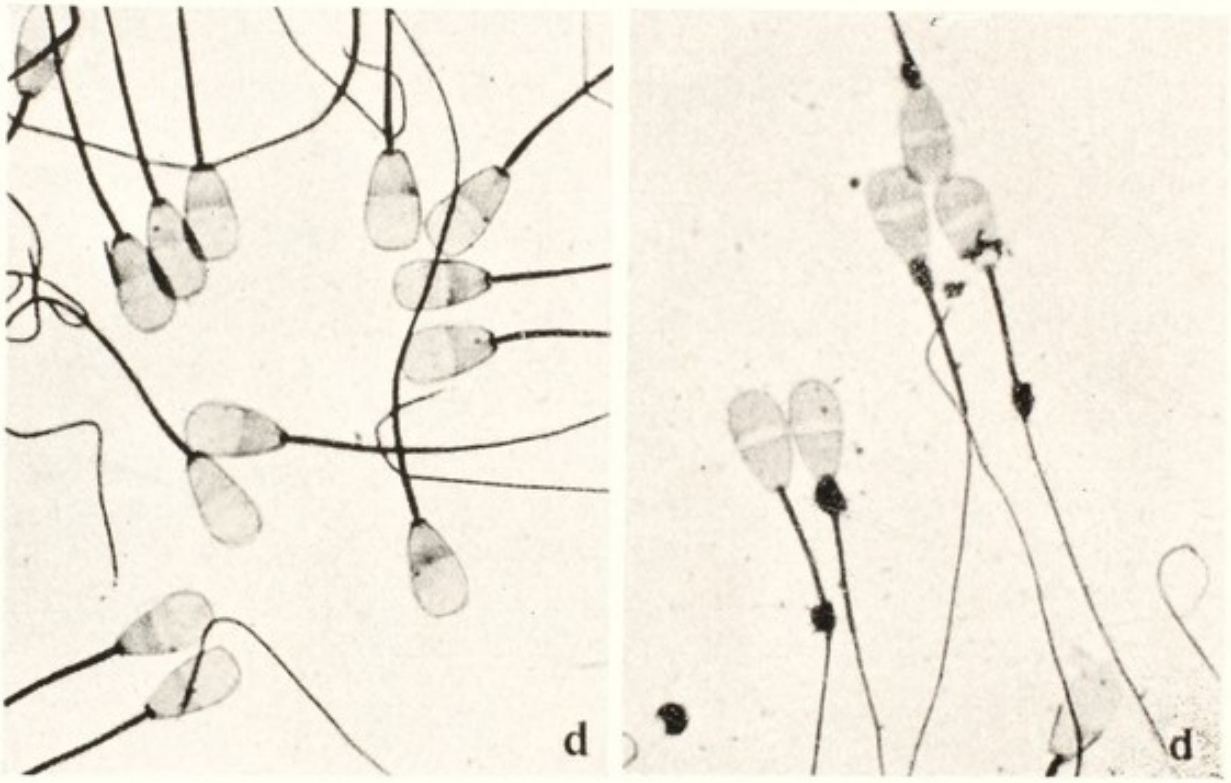
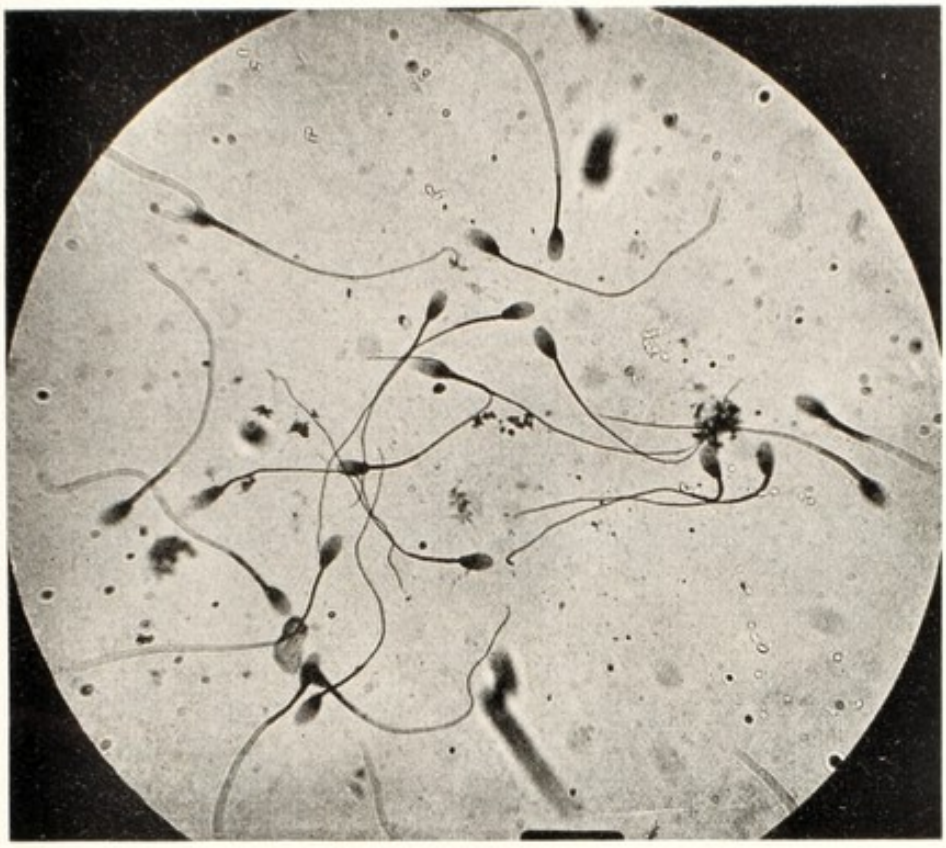


FIG. 127. Normal and Unripe Bull Spermatozoa.

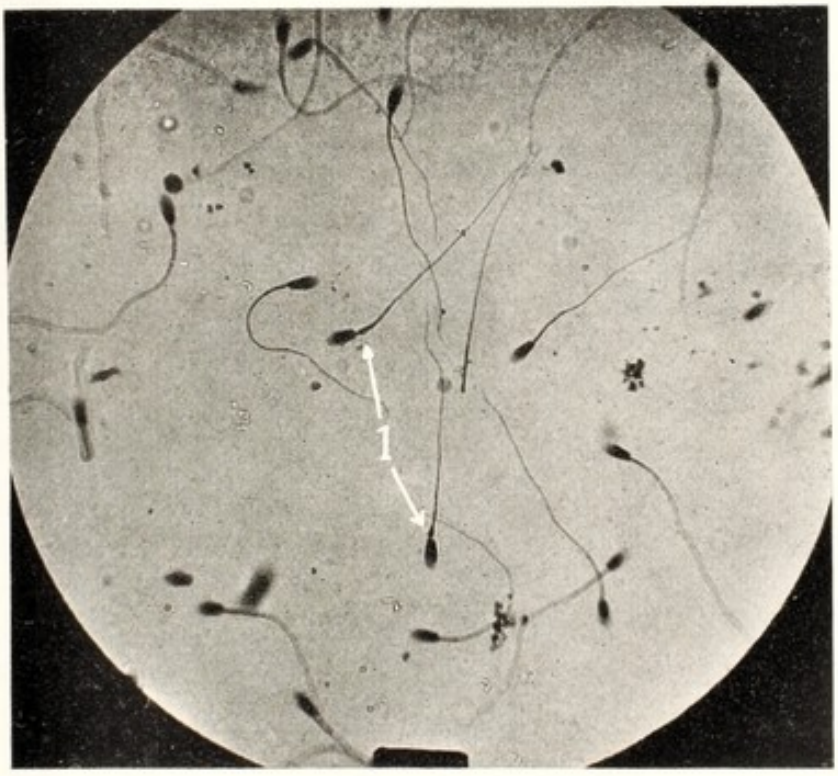
On the left, normal spermatozoa: on the right, immature cells with protoplasmic droplets about neck or at juncture of middle piece with tail. *At two years the latter bull served ten cows repeatedly without a conception. Testicular hypoplasia.* (Lagerlöf)

mon in various types of testicular degeneration, and abounds in the spermatozoa produced by testicles submitted to experimental degeneration by insulation, as recorded by Lagerlöf.

Not infrequently the tails of a large ratio of spermatozoa are abruptly folded at their juncture with the middle piece, as shown at *a*, while in other cases the middle piece and the tail form a series of coils about the head as in *b*, of Fig. 111. The types of defect in the middle piece and the tail are extremely variable. The folding and coiling of tails do not completely destroy their motility, but do inhibit their efficiency. The cells have an undulatory movement, but remain fixed in their location, powerless to migrate toward the ovum.



a



b

FIG. 128. *a*, Spermatozoa from Healthy Stallion; *b*, Spermatozoa from Stallion of Low Fertility.

Note, at 1, two cells with ring of protoplasm about neck. *(Cornell Veterinarian)*

A large ratio of heads, as indicated in Fig. 111, may become detached from the tails. A given field may contain many detached heads with few or no separated tails, or the reverse may be true, as shown in an unique specimen supplied by Webster (Fig. 129) in which all the heads are detached and the tails agglutinated in great masses. The detached heads are usually abnormal in form, size, and staining affinity.

Instead of the physiological discard of the tail at the time of union of the ovum and spermatozoon head, the detachment occurs prematurely, prior to ejaculation. No evidences of decay are observed in the separated parts. When spermatozoa die, the heads do not

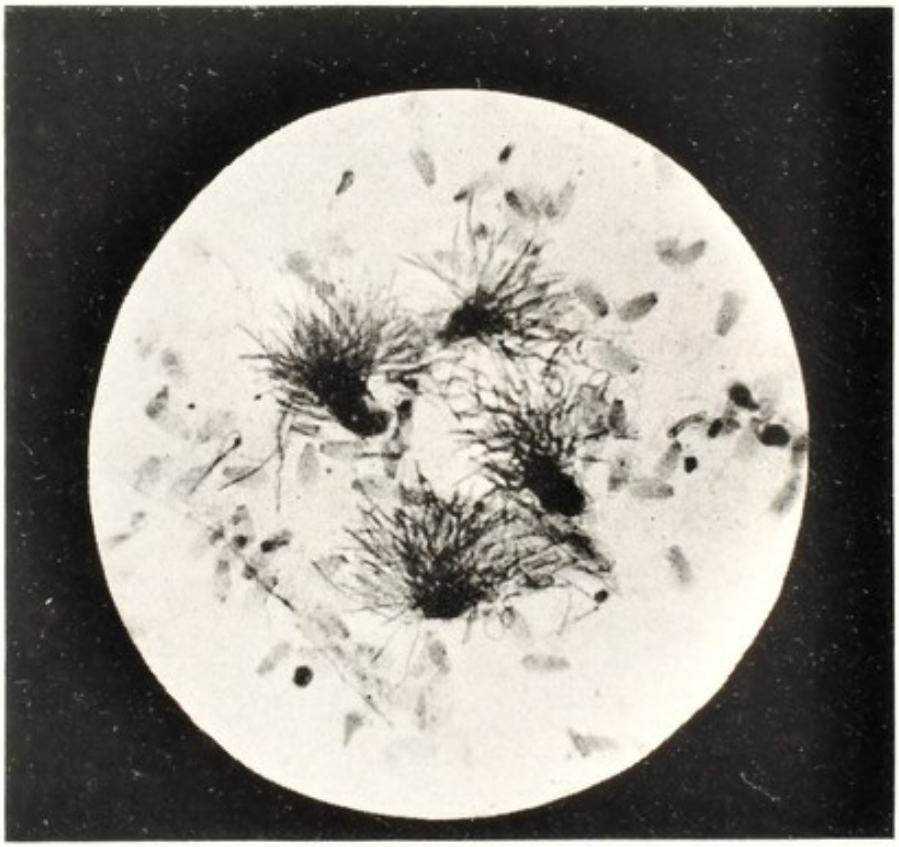


FIG. 129. Bovine Spermatozoa Tails Detached from Heads and Arranged in Sheaves or Bundles. (Webster)

usually separate from the tails. There is room for the suspicion that the division occurs in the living cell, as a pathological phenomenon. The detached tails are sometimes motile.

Sometimes the sperm heads are broken off because of undue force in making the film, but in such cases there are usually present mutilations of various parts, evidently resulting from mechanical violence.

The heads of defective spermatozoa show limitless variations in size, form and affinity for stains. Sometimes almost all heads are pathologically narrow, as indicated in Fig. 130. Numerous detached

heads are present, smaller than the heads of the complete cells. The middle piece is abnormally slender and the differentiation between it and the tail is not clear. Such cells may be fairly motile but incapable of fertilizing ova. Many types of giant and dwarfed heads are observed. In Fig. 28 no two heads or tails are alike in volume or form.

The significance of the various defects of the spermatozoa, so far as now known, depends upon the ratio of abnormals, rather than upon the character of the morphological deviation. The sperms which are visibly defective perhaps rarely fertilize ova. The conclusion seems



FIG. 130. Elongated, Narrow Spermatozoan Heads. (Webster)

inevitable that, in most sires of low fertility, the defects in many of the cells are not microscopically recognizable. The ratio of visible abnormals affords an authentic meter of the fertility and vigor of those cells which appear morphologically perfect. Hence, the higher the ratio of abnormals, the lower the fertility of the sire, the more insecure his pregnancies, the weaker his born young, and the lower their value as adults. Williams and Savage have concluded—and their conclusion has been in general accord with clinical observation—that for the bull reasonably satisfactory fertility cannot be expected when the total abnormals exceed 170 per thousand. For the stallion the total of abnormals, with fair fertility, may reach 200

per thousand. When the abnormals exceed the ratio designated, prudence dictates that the sire be discarded, or that his services be suspended pending possible recovery. When selecting a sire it is well to reject any male with a ratio of 50 abnormals per thousand. It should be constantly borne in mind that, the lower the ratio of abnormal sperms, the higher the value of the sire. A proven sire with a relatively high ratio of abnormals may be retained with the hope that by conservative use and handling his value may be restored.

EPIDIDYMITIS

The epididymis consists of a greatly coiled tubule, representing the cranial end of the Wolffian duct. Schmaltz[178] states that when the tubule is uncoiled by dissection it is 25 to 30 meters (82 to 90 ft.) long in the horse. The caudal end of the Wolffian duct, the vas deferens, consists of a straight tubule. In the fertile male both the epididymal tubule and the vas deferens are densely packed with spermatozoa. The tubules serve as a reservoir for the spermatozoa after their discharge from the testicles until they are ejaculated at coitus or have otherwise disappeared. There are sufficient numbers of spermatozoa stored in the vas deferens that a horse copulating immediately following castration may cause conception.

Epididymitis is not infrequent in stallions and bulls. It doubtless occurs in all species. As indicated in Figs. 118, 119, 121, epididymitis is not readily distinguished clinically from orchitis. When abscessation of the testicle occurs, as in Fig. 114, the epididymis usually disappears along with the glandular parenchyma. Generally the phenomenon is designated orchitis but it is not authentically known in which structure the primary invasion occurred. So as a rule orchitis and epididymitis are described as distinct diseases, although clinically they frequently, if not usually, coexist. If the one is primary, and the other secondary, their order is unknown. In such cases as Figs. 118, 119, and 121 there is room for the conjecture that the epididymitis, by closing the avenue of escape of the spermatozoa, brought about degeneration and atrophy of the glands.

The chief clinical symptom of epididymitis is the enlargement of the structure, usually most prominent at the tail. There also the comparative size of the two structures serves to mislead, and the only safe basis for a conclusion is the *absolute* enlargement. It would naturally be expected that in the early stages the structure would be painful upon palpation, but as a rule the disease is well advanced prior to the search.

When the testicle and epididymis have been removed and sec-

tioned, the organs present variable lesions. Frequently there is a marked exudate in the surrounding connective tissue and enlargement of the epididymal tubules. Sometimes the tubules are distended with pus or other material.

The cause of epididymitis is unknown. The purulent orchitis of bulls is generally ascribed to *Bacillus abortus*. It is not known that, in cases so diagnosed, the abortion bacillus was the primary invader. Epididymitis is not generally purulent in any species. There is no definite preventive measure known, except that inferentially the conservative handling would affect epididymitis as it does reproductive diseases generally. Once established, there is no known remedy.

The Diseases of the Accessory Sexual Glands of the Male

The accessory genital organs of the male vary widely in their volume as compared with the body weight of the animal, and their vulnerability to disease is apparently somewhat in harmony with their comparative size. The seminal vesicles are highly developed in herbivora and swine, and disease of them is most common in the stallion and bull. The prostate glands are not conspicuously diseased in herbivora. The prostate glands are more highly developed in carnivora, and in the dog, prostatic disease is not rare. The bulbo-urethral glands are small in all species and are not the seat of notable disease.

Diseases of the Vasa Defferentia and Seminal Vesicles

It is not known that the vasa defferentia often suffer primarily from disease in those portions of the canal stretching from the epididymis to the ampulla. Apparently disease does extend from the epididymis along the deferent canal as indicated in Fig. 116. The ampullae, in those animals in which they are markedly developed, as in solipeds and ruminants, are not rarely diseased, usually associated with similar pathological changes of the seminal vesicles. The ampullae and seminal vesicles emanate alike from the Wolffian ducts and the two structures are not known to differ in their physiological function, beyond the fact that the ampullae contain spermatozoa in transit, while the vesicles do not. Pathologically they frequently, if not generally suffer alike in those animals where both are present. The ampullae are absent in the boar and the seminal vesicles are not present in the dog. The seminal vesicles of the boar are relatively enormous in volume, which inferentially subjects them to disease, but the field has not yet been notably explored.

The literature upon the pathology of these structures is extreme-

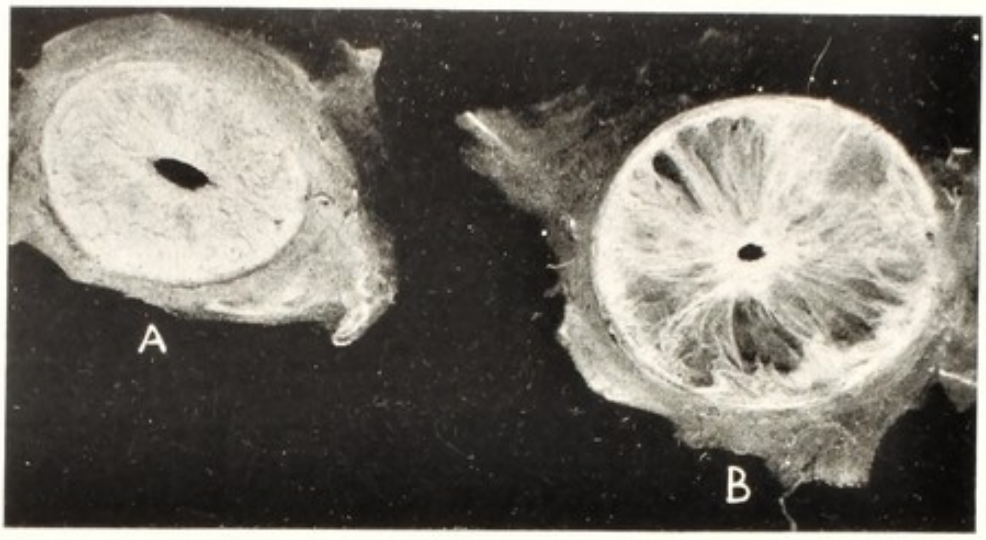


FIG. 131. Cross Section of Healthy and Diseased Ampullae of Vas Deferens of Stallion. A, Healthy ampulla; B, diseased ampulla from stallion illustrated in Figures 28 and 120.

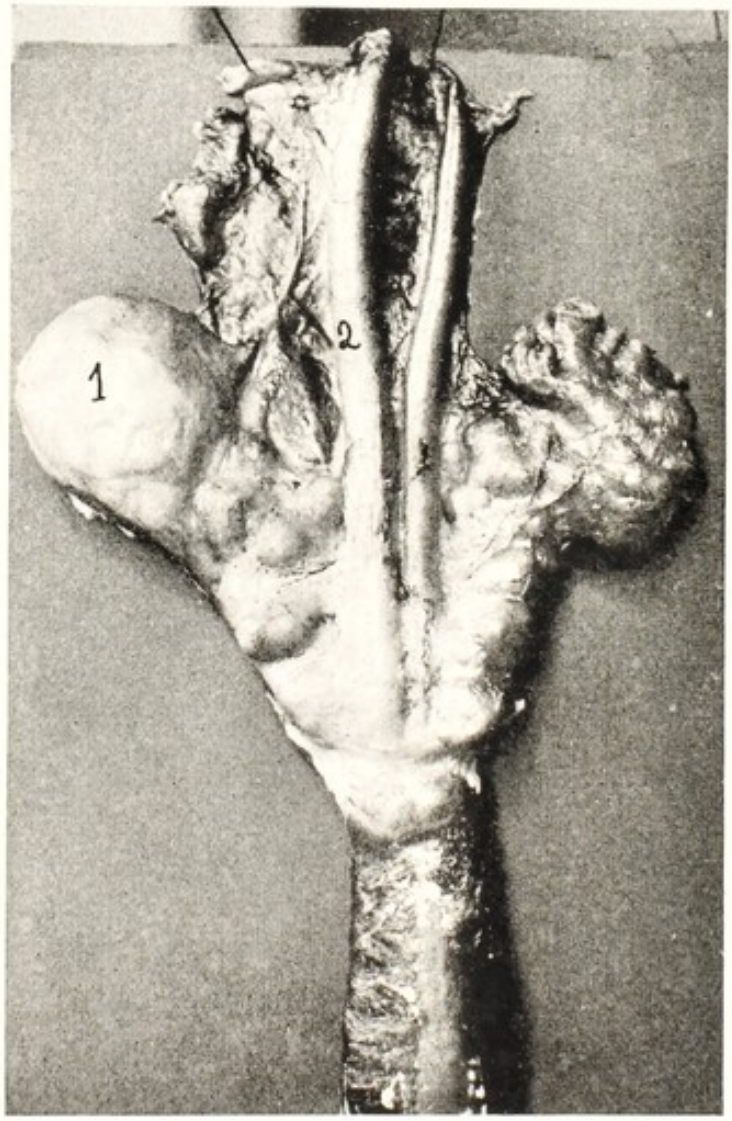


FIG. 132. Abscess of Seminal Vesicle and Ampulla of Vas Deferens Associated with *Bacillus Abortus* Infection.

1, Abscess in left seminal vesicle; 2, the infected left ampulla. (Lagerlöf)

ly scant. When animal husbandry methods begot sterility, abortion and the innumerable other interferences with reproduction, their control was chiefly sought in the handling of the female, and scant attention accorded the diseases of the testicles and their tubular outlets. Hence the literature upon the diseases of the vasa defferentia, their ampullae and the seminal vesicles is almost blank. A very few veterinarians have recorded superficial studies upon a small number of cases which serve to illustrate some of the clinical characteristics of the diseases of these structures. Limited as the contributions are, they do serve to show reliably that there is an intimate relationship between the diseases of these and of the testicles and epididymes, and that the entire group of derivatives from the Wolffian bodies and ducts—testicles, epididymes, vasa defferentia, ampullae and seminal vesicles—are vitally important in reproduction and that no one of them may suffer from recognizable disease without serious peril to breeding. The testicles must be healthy in order to produce physiological spermatozoa, and the tubular channel through which they must pass needs be sound lest the passage of the spermatozoa be barred or they be subjected to serious peril during transit to the exterior. A review of some of the most significant contributions may supply an imperfect account of the diseases of these structures.

A two-year-old, purebred Belgian stallion affected with well defined orchitis, ampullitis and semino-vesiculitis was incompletely studied in company with Professor Frost.[243] At the first attempt to breed mares, the colt showed intense sexual desire, mounted mares promptly, introduced the penis, and immediately dismounted, with the penis greatly erected. Upon rectal palpation, the ampullae and seminal vesicles were much enlarged, tense and highly sensitive. The testicles were enlarged and adherent within the scrotum. Several attempts to breed mares, uniformly failed—no pregnancies, no ejaculations. Pronounced hopeless, he was castrated and later sold. He died not long afterward and his death was not learned of until he had been buried. The cadaver was exhumed, but decomposition was too advanced for detailed autopsy. The seminal vesicles were adherent to the rectum and surrounding tissues. At date of clinical examination the ampullae were similar to that illustrated in Fig. 131.

It is not known at what date the disease became established, and the invader was not identified. The stud in which he was born, and an attempt made to use him as a sire, was not known to have suffered from any infection notably interfering with reproduction. The disease, definitely established before the first attempt at coitus, may have been congenital.

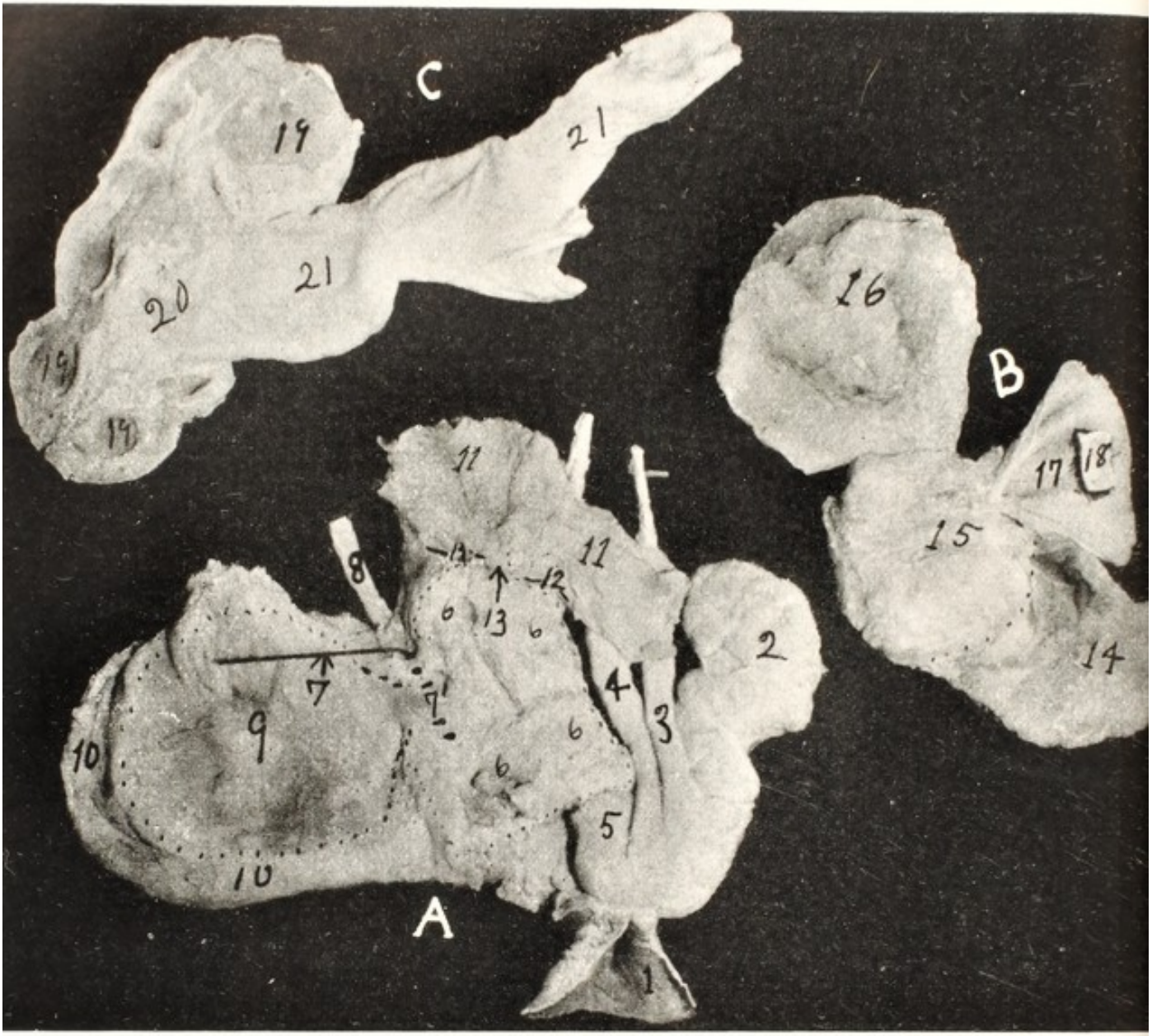


FIG. 133. Intra-pelvic portions of the urino-genital system of an adult bull, with extensive abscess of left seminal vesicle. In A the peritoneal covering has been removed from the seminal vesicles and they have been drawn apart anteriorly in order to expose to view a large portion of the ampullae of the vasa defferentia.

A. 1. Mid-dorsal section through the prostate gland and dorsal side of the pelvic urethra; 2, the apparently normal right seminal vesicle; 3, the ampulla of the right vas deferens; 4, the ampulla of the left vas deferens, enlarged; 5, the apparently normal base of the left seminal vesicle; 6, 6, 6, 6, unilocular abscess of the middle and anterior portions of the left seminal vesicle, adherent to the rectum and communicating with it through a fistula. The left wall of the abscess and left side of the rectum have been excised and are shown from the peritoneal side in B; 7, bent sound in the terminal section of the left ureter; 7′, approximate course of the left ureter through the abscess area; 8, right ureter; 9, secondary abscess from the left seminal vesicle extending along the left wall of the urinary bladder; 10, 10, a narrow strip of the urinary bladder not occupied by the abscess; 11, right side of the rectum; 12, line of adhesion between the seminal vesicle and rectum; 13, recto-vaginal fistula.

B. Left side of the rectum, left wall of the abscess of the seminal vesicle and outer wall of the abscess alongside the urinary bladder, viewed from the left, or peritoneal side.

14. Left side of rectum; 15, left side of abscess of seminal vesicle; 16, left wall of abscess alongside the urinary bladder; 17, the dilated left ureter laid open immediately

In the calf shown in Figs. 115-117 the conclusion is virtually inevitable that the infection (*Bacillus pyogenes*) was congenital, the dam had required repeated services for one and one-half years before recognizable pregnancy occurred, and the calf was weak and ill at birth. Some might term this heredity, in the sense that the foundation for the disease was acquired within the uterus of a critically diseased dam.

Lagerlöf[116] describes a bull in which the left ampulla and seminal vesicle were extensively diseased, the vesicle being occupied by an apparently torpid abscess, free from adhesions to adjacent organs, as indicated in Fig. 132. The abscess is attributed to *Bacillus abortus* (Bang). Inferentially, infection is always present in semino-vesiculitis, and according to bacteriological reports, Bang's bacillus is one of the most, if not the most common invader in abscesses of cattle and solipeds. The picture may be somewhat distorted by the eagerness to report the identification of this invader.

Except the outlets of the ampullae and vesicles become blocked by the formation of an abscess, or otherwise, any ejaculation of semen or of the products of the diseased glands becomes contaminated with bacteria, pus or any invader causing the disease. The presence of pus in the semen (Fig. 110), unless its origin can otherwise be authentically traced, calls for a careful search of the ampullae and seminal vesicles. Pus from suppurating testicles and epididymides is commonly associated with blocking of the vasa defferentia, thus barring its escape in an ejaculate.

Accordingly, the services of some of the very few bulls studied clinically because of disease of these glands, have been followed by severe cervicitis in the cows with which they have been mated. Such was the case in the bull illustrated in Figures 102, 123, and 137. The epididymal disease, Fig. 123, quite certainly blocked the vasa defferentia and limited ejaculation largely or wholly to the products of the ampullae and vesicles; the disease of the penis, Fig. 102, possibly contributed significantly to the infection. The limited available data quite justify the conclusion that if a male suffering from clinically recognizable disease of the ampullae or of the seminal vesicles, is used as a sire, his services are followed by important diseases of the genital system of the females with which he is mated, the lives of the intrauterine young are jeopardized, and the living young are

anterior to the area of its impingement by the abscess of the seminal vesicle; 18, cross section of the normal right ureter laid open and inserted for comparison with the dilated left tube.

C. Left kidney and ureter. 19, 19, small areas of quasi-normal kidney tissue; 20, dilated pelvis (calyces majores) of kidney; 21, 21, dilated ureter.

largely affected with the diseases to which newborn are commonly susceptible. The multiplicity of the organs involved may render it difficult in a given case to authentically determine which one is at fault; two or more of the organs may participate. Whatever the conclusion may be, no authentic record is available of a sire with a definitely satisfactory breeding history, in which clinical evidence was present of orchitis, epididymitis, ampullitis, or semino-vesiculitis. While the disease may primarily affect only a single organ of right or left half of the system, the relations between the individual organs are such that commonly two or all of the organs of the affected side eventually become involved.

Since the male reproductive organs, anterior to the urethra, are distinctly paired, it would seem that the organs of one side might become significantly diseased, without seriously involving the other half. This sometimes occurs, but when the affected side remains in communication with the urethra, any bacteria or other invaders, or any other products of disease from the affected side, tend to mingle with the ejaculate from the healthy half of the system and cause disease. In the bull with abscess of the left seminal vesicle, described by Williams and Bushnell,[264] with blocking of the left vas deferens at its caudal end, associated with atrophy of the left testicle, Figs. 133 and 134, there remained some fertility for presumably several years, the degree of which could not be authentically determined. This suggests that the abscess of the seminal vesicle, with discharge into the rectum, closed the outlet of the vas deferens into the urethra and rendered possible the ejaculation of fertile semen from the right side without serious pollution from the left. When severe orchitis occurs on one side, the disease seems usually to prevent physiological spermatogenesis in the other apparently healthy testicle.

The symptoms of ampullitis and semino-vesiculitis have not been extensively recorded. Such cases as that illustrated in Fig. 132 would probably not be suspected except for the sterility, and this might be due to any one of a multitude of causes, and may only be identified by rectal exploration or autopsy. In cases like Fig. 133 there probably occur significant symptoms at the time of the maturation and rupture of the abscess, but no record of these is found. There would probably be interferences with defecation and, if the abscess forms after the male is placed in service, there would probably be difficulty in copulation. Thus in the Belgian colt described above there was impossibility of copulation, with extreme sensitiveness of the ampullae and vesiculae upon palpation. Here the inability to copulate was presumably due to pain. Such is not

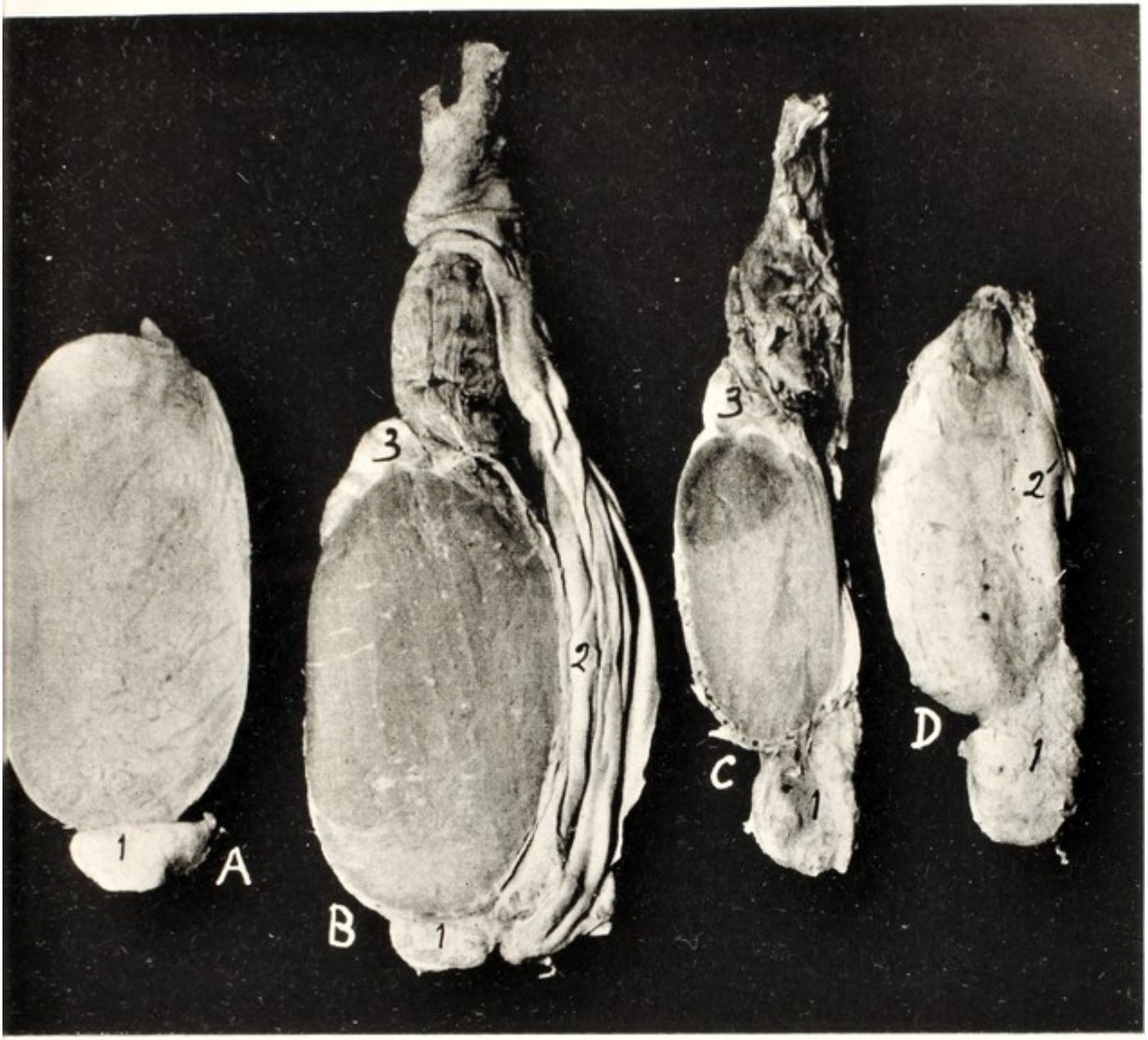


FIG. 134. Testicles of bull, some of whose organs are illustrated in Fig. 133.

A. Half of apparently normal right testicle shown from peritoneal side; B, the other half viewed from sectioned side; C, section through the left testicle seen from sectioned surface; D, peritoneal side of same.

1. Tail of the epididymis; 2, body of epididymis; 2′, enlarged body of epididymis of atrophied left testicle, covered by the adherent tunica reflexum of scrotum; 3, head of epididymis.

always true. A prize shorthorn bull, which I attended, had been unable to copulate for about a year. A semino-vesicular abscess had ruptured into the rectum at an unknown date and was quite free from recognizable sensitiveness of the parts upon rectal palpation. The cause of his inability to copulate could not be determined by clinical examination and, since the owner wished to exhibit him at leading cattle shows in the hope of winning further prizes as a sire, autopsy was not available.

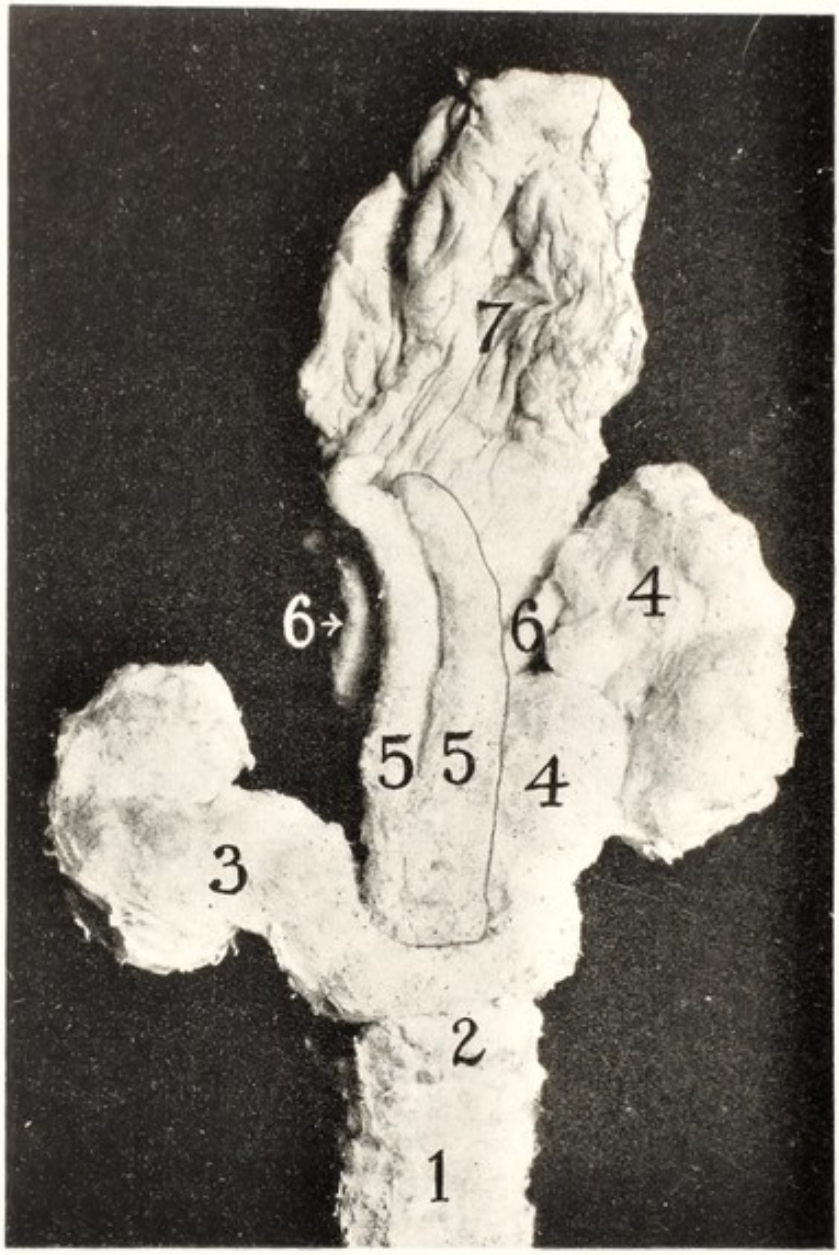


FIG. 135. Old-standing semino-vesiculitis in mature Jersey bull. 1, Wilson's muscle surrounding the pelvic urethra and the main portion of the prostate; 2, body of prostate; 3, the approximately normal left seminal vesicle, drawn aside; 4, right seminal vesicle, enlarged to thrice normal volume; 5, 5, ampullae of vasa defferentia; 6, 6, ureters; 7, urinary bladder.

Such extensive involvement of both of the ampullae and seminal vesicles, as shown in Fig. 137, is readily recognizable as an indistinct, adherent mass, ventral to the rectum and dorsal to the neck of the bladder, occupying the normal ampullo-semino-vesicular area. There is an absence of definite fluctuation, the ducts communicating with the urethra remaining open, permitting the pus and other fluids to escape freely. This permitted the bull to copulate freely, ejaculating large quantities of virulent pus, causing severe cervicitis in the cows

which he served, constituting, probably, the most dangerous type of disease of the male genital organs.

The bull from which Figs. 135 and 136 were obtained was a highly prized aged Jersey, designated *Case 36* by Williams *et al.*[243] Ampullo-vesiculitis was readily diagnosed by rectal palpation and the bull was continued in service for three years, despite our advice to the contrary. During that period he was credited with 37 preg-

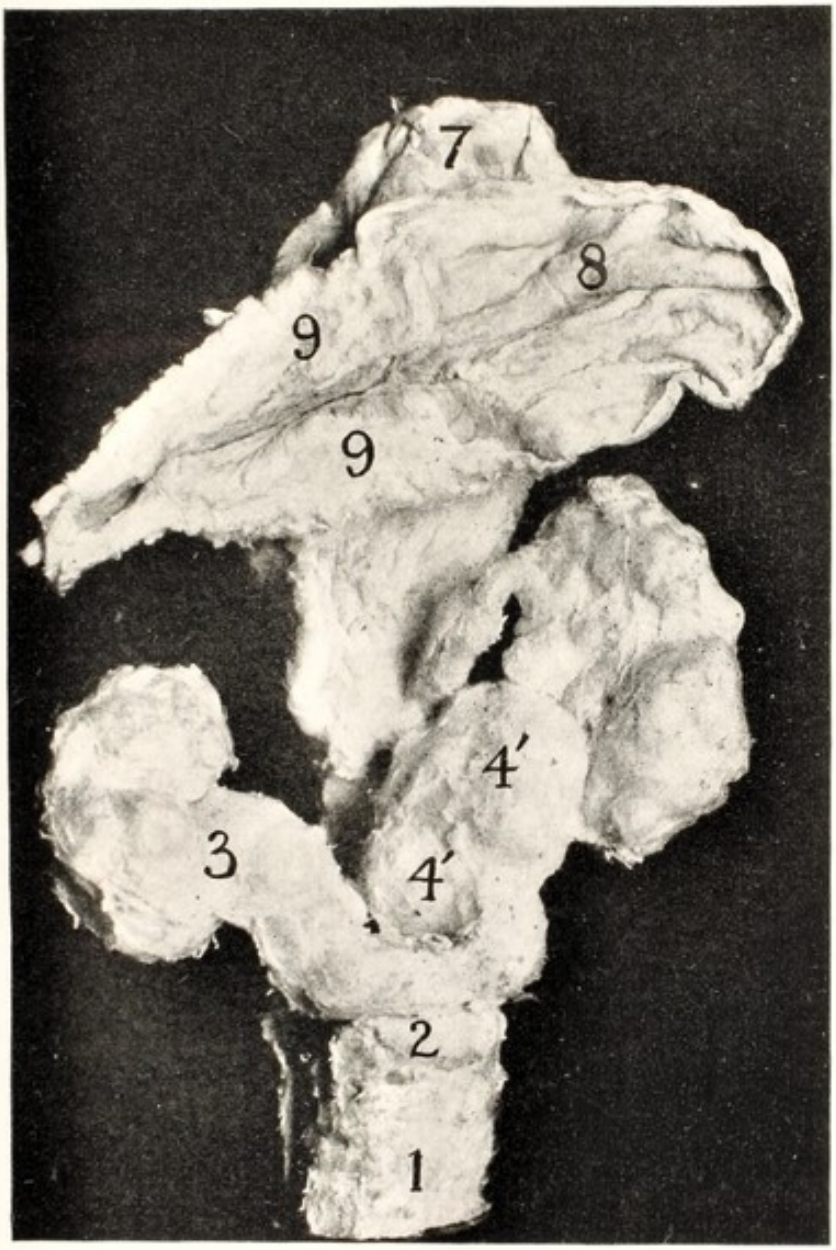


FIG. 136. Same as 135. The ampullae of both vasa defferentia have been removed, disclosing at 4′, 4′ a gross enlargement of the right seminal vesicle which has pushed across the neck of the urinary bladder, beneath the ampullae and displaced the left seminal vesicle to the left. The ventral half of the urinary bladder has been removed and laid transversely across the dorsal half and seen from the interior. The walls of the fundus and anterior half, 8, are approximately normal in thickness, while those of the posterior half, 9, 9, are enormously thickened, apparently due to the mechanical compression of the enlarged right seminal vesicle at 4′, 4′.



FIG. 137. Inflammation of the Vesiculae Seminales and Ampullae in a Bull.

(Figs. 102 and 123 made from same bull.) On the left normal urinary bladder, seminal vesicles and ampullae of vasa defferentia for comparison. The vesiculae seminales are contracted and somewhat deformed from hardening in formalin.

1, 1, Greatly enlarged, adherent vesiculae seminales; 2, 2, enlarged ampullae of vasa defferentia; 3, urinary bladder; 4, pelvic urethra covered by Wilson's muscle and prostate gland; 5, body of prostate.

nancies, with 4.4% abortions and 95.6% calves. Of the 37 cows, 30% had metritis, 24.3% retained placentae, and 40.5% milk fever. The mortality of the new-born calves was 59.5% (convulsions, diarrhea, etc.), 13.5% of weak calves survived and 21.6% of the calves appeared healthy. There were four other breeds in the herd, among which the diseases of cows and calves above mentioned were not notable. While it can not be undeniably shown that the ampullo-semino-vesiculitis caused the milk fever in the cows and the fatal convulsions in the calves, no other hypothesis presented itself.

Scanty studies in this field render it impossible generally to fix the date of origin of these diseases. The case illustrated in Figures 115-117 was quite certainly congenital, the heifer dam having been fruitlessly bred for about one and one-half years prior to her con-

ception which resulted in this diseased bull. Similarly, the bull, designated by Williams *et al*[243] as *Case 120*, was absolutely sterile from the first service onward. The bull was well matured, physically vigorous, copulated promptly and apparently naturally, but ejaculated no spermatozoa. His epididymes and vasa defferentia were blocked by pus, inferentially of antenatal origin. It has already been suggested that in the Belgian colt described above, the disease was probably congenital. Likewise the bull whose seminal vesicles are illustrated in Fig. 138, infected his cows from the first, as shown in Table II, and the conclusion seemed justified that the disease was congenital, although possibly the abscess formed later.

The lesions associated with ampullitis and semino-vesiculitis vary widely. The location of the ampullae and vesicles is such that disease of them may involve the rectum, the urinary organs and other adjacent tissues. Thus in the case described by Williams and Bushnell,[264] in which an abscess of the left seminal vesicle had ruptured into the rectum, as shown in Fig. 133, there existed such extensive adhesions to the urinary bladder as to suggest the possibility of rupture into that organ. As indicated in the figure, the abscess had so compressed the terminus of the left ureter that the urine had been retained to such degree as to cause great dilatation of the ureter and of the calyces majores or pelvis of the left kidney, leading to almost complete atrophy of the renal tissue, and logically resulting in corresponding hypertrophy of the right gland. The left ampulla was also compressed, as well as diseased, so that its canal was essentially closed, which signified little, since the left testicle was atrophic and created few, if any spermatozoa. Unlike the kidney, the testicle is not known to become distended and atrophied because of compression of the vasa defferentia at outlet. Hence epididymitis, as suggested in Figs. 117-119, effectively blocks the passage of spermatozoa or other product, and eventually leads to atrophy, with macroscopic appearance of fibrous degeneration. According to Richter[169] testicular atrophy in the goat may be due to embryological defects in the vasa efferentia, as a result of which they fail to become canalized, so that when spermatogenesis begins the sperms are stalled and spermatocele with testicular degeneration follows.

The vasa efferentia and the intricately coiled epididymal tubules apparently constitute a formidable barrier to the passage of bacterial invaders in either direction between the testicle and its excretory system of canals. Abscesses occur in the testicle, frequently

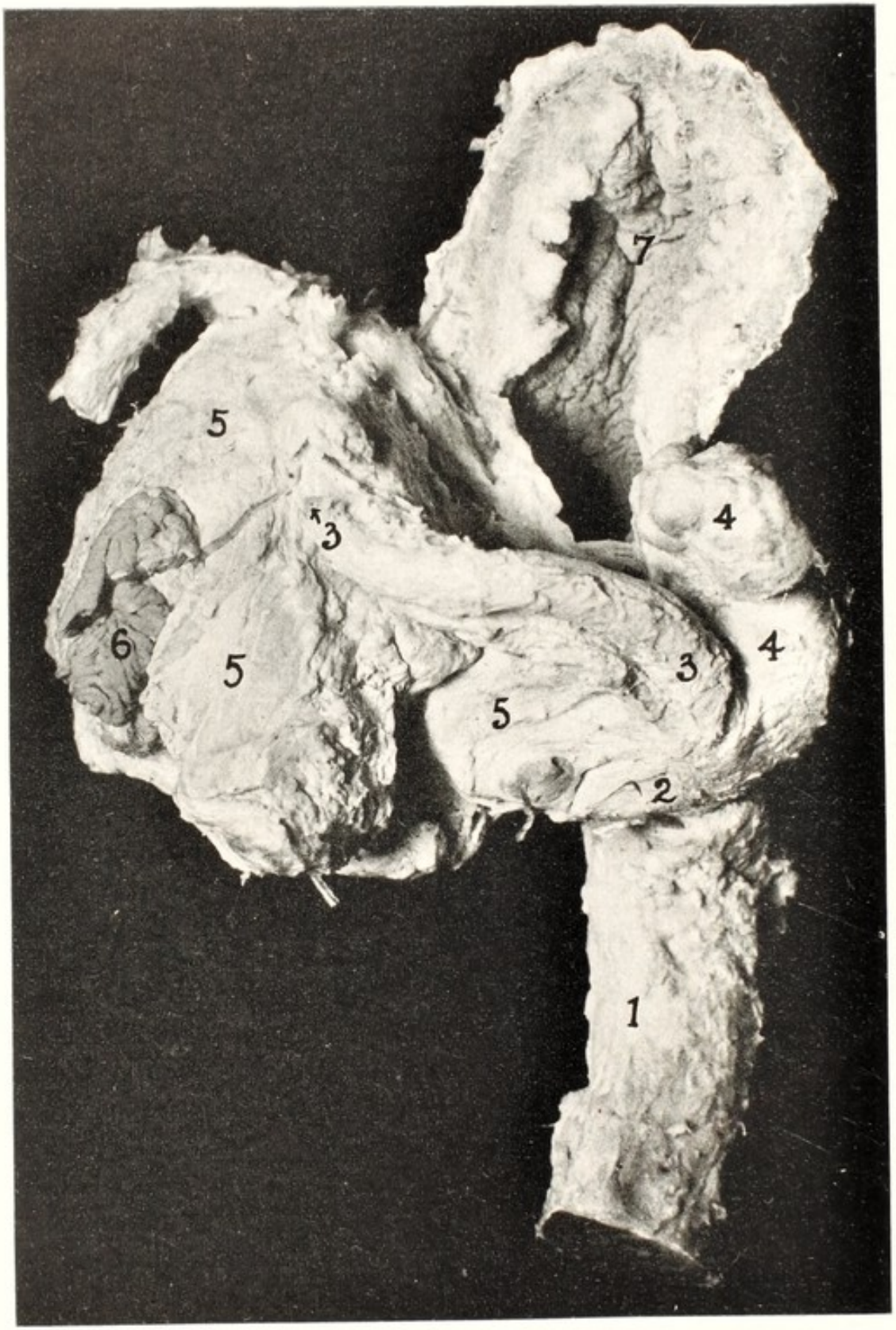


FIG. 138. Abscess of Seminal Vesicles of Bull 2 in Table II.

1, Pelvic urethra covered by Wilson's muscle and prostate; 2, body of prostate; 3, ampulla of the right vas displaced to the left by the abscess; 4, 4, right seminal vesicle nearly normal in volume; 5, 5, 5, large abscess of left seminal vesicle with indurated walls; 6, the rectal floor adherent to the abscess and showing a fistulous opening into the rectum through which the abscess contents had escaped; 7, ventral half of urinary bladder. *(Cornell Veterinarian)*

attributed to tubercle bacilli or to *Bacillus abortus,* but the invasion is not known to occur through the vas deferens.

The date of the onset of ampullitis and semino-vesiculitis is not authentically determinable. The Belgian colt described above apparently came to breeding age with the disease already existing. There was no observed disease of unusual character in the small stud where he was born and tried as a sire. There was nothing to justify the conclusion that he had become infected through his attempted service as a sire. The duration of his intrauterine life and his vigor at birth were not learned—he may have been carried far over time and born diseased. So with the other cases described, the date of the onset of the disease is commonly unknown.

Reliable means for early diagnosis are not known, but must await the development of definite lesions which are clinically recognizable by palpation. Although there is presumably always present a pathological invader, it is not at present practicable to recognize the infecting agent. In such cases as shown in Fig. 133 there is a strong probability that the patient may react to the test for brucellosis, but that does not show that the Bang bacillus was the primary invader—the original disease may have been due to other infection. The difficulty of dependable early diagnosis generally bars proper handling until the male has infected some or many females, except in those cases in which he is unable to copulate.

A study of the semen provides authentic information in some very important respects. The presence of pus in the ejaculate generally indicates suppuration of the vasa defferentia, their ampullae, or the vesiculae seminales. An absence of spermatozoa in the semen probably usually indicates the blocking of the vasa efferentia with spermatozoa—spermatocele—or it is due to epididymitis, which bars the passage of semen into the vasa defferentia, and limits the ejaculate to the secretions of the ampullae and vesiculae seminales. The presence of anatomically normal, non-motile sperms most probably indicates injury from some virulent invader contacted in the vasa defferentia or their ampullae, but disease of the tubuli seminiferi may be responsible. The presence of structurally defective spermatozoa, whether teratological or pathological, such as shown in Figures 127-129 and 28, may confidently be attributed to testicular disease.

Although it may be difficult or impossible to authentically assign given defects in spermatozoa, or genital diseases of the female, to a given male organ, a reliable clinical diagnosis may be made by

studying: (a) the fertility of the sire as measured by the ratio of recognizable pregnancies; (b) the rate of abortions or "born dead" among his pregnancies; (c) the duration of pregnancies resulting in the birth of living young; (d) the vigor of the new-born young, including the occurrence of such phenomena as retention of the meconium (equine), diarrhea (equine and bovine), and pneumonia (bovine), etc.; and of puerperal diseases of the female such as retained placenta, excessive weight of placenta, metritis, etc. Such clinical data, associated with adequate study of the stained spermatozoa, and the clinical examination of the male genital organs, constitute a dependable foundation for diagnosis and provide the clinician with trustworthy means for the control or eradication of the disease. But first of all, the clinician and the animal husbandman must fully realize that genital diseases are not a mere conglomeration of "female complaints" as taught by patent medicine vendors, but that reproduction is dependent equally upon the two sexes, and that successful animal husbandry depends primarily upon the mating of two healthy, mature animals.

There is no known remedy for established diseases of the testicles, epididymes, ampullae of the vasa defferentia and the seminal vesicles. Lagerlöf,[114] Phillips and McKenzie[156] and others have shown that, by elevating the temperature of the pendant scrotum of the bull for a brief interval, the testicles become disturbed and the spermatozoa degenerate structurally and physiologically, so that the animal becomes sterile; if the insulation is not too prolonged or severe, the testicles may regain their physiological function and elaborate potent spermatozoa. This suggests that, as in other diseases, those of the male genital organs may sometimes recover if timely rest is obtained. But in this group of diseases recognition is usually impossible until after the pathological changes have become permanently fixed. Such was the experience of W. L. Hindmarsh, Australia, who relates in a personal communication, that in a large herd, almost all cows bred to the old herd sires suffered much from metritis and retained afterbirth, while a number of females bred to a young bull calved physiologically. The herd was negative to the agglutination test for Bang's disease, and the infection was attributed to streptococci and colon organisms.

The bull whose reproductive system is illustrated in Fig. 139, with defects of the ampullae, seminal vesicles and testicles, also shows an abnormal prostate, which is uncommon in bulls. His pregnancies were much abbreviated, abortions were numerous and retained afterbirth highly prevalent.



FIG. 139. Diseased testicles, vasa defferentia and vesiculae seminales from a pure-bred bull in a herd officially accredited free of tuberculosis and *Bacillus abortus,* with the genital tract of a normal bull as check.

A, Healthy genital organs, lettering as in *B. B.* 1, 1, the vasa defferentia; 2, 2, the enlarged ampullae of the vasa defferentia which end bluntly anteriorly instead of tapering gently as in health; 3, the enlarged, edematous seminal vesicles, the right one having been congenitally abnormal, bent at right angles to the long axis of the pelvic urethra; 4, the body of the prostate, enlarged; 5, adhesions of the parietal, to the visceral peritoneum of the scrotum; 6, surface of the testicle roughened by general adhesions between the peritoneal layers; 7, prominent adhesions; 8, body of epididymis; 9, vas deferens.

No remedy is known for this group of diseases. No vaccine administered to bull, cow or calf could be of avail. The only known path of safety is the discard of the defective sire.

The bull whose reproductive system is displayed in Fig 139 with disease of the ampullae, seminal vesicles and testicles also shows prostatic disease, which is unusual. His pregnancies were much abbreviated, abortions were numerous and retained afterbirth highly prevalent.

PROSTATITIS

Prostatitis is rarely observed in domestic animals except in the dog, where it is not uncommon in aged animals. The chief clinical symptom is difficult urination. The diagnosis is somewhat readily made by digital palpation of the pelvic urethra per rectum. Compared with that of a healthy dog, the gland is enlarged, hard, or sometimes fluctuant from the formation of an abscess.

Prostatitis is technically operable but is rarely, if ever, advisable from the standpoint of the restoration of valuable reproductive power. The dog is usually old; the function of the gland is not restorable and therefore the dog cannot logically be made a desirable sire. Sentimentally, the life of the dog may be prolonged by a surgical operation upon the gland itself, or according to some writers, the prostatitis may be ameliorated by castration.

Diseases of the Glans, Prepuce and Sheath

BALANITIS

The glans penis, prepuce, and sheath of solipeds are comparatively well protected by a hairless membrane somewhat intermediate between skin and mucous membrane. It is well supplied with sebaceous glands which excrete a smegma having a characteristic odor. The parts are not strikingly vulnerable to invasion by pathogenic forces. The outstanding exception is the virus already described as *vesicular venereal disease*. The urethral opening and the ventral surface of the preputial ring or fornix are also invaded, as already described, by the infection designated bursatte.

Collections of sebum or smegma sometimes accumulate upon the glans and within the sheath and prepuce and, undergoing desiccation, become hardened masses which irritate the parts and may lead to suppuration. This is observed chiefly in geldings. If the colt is castrated early, the penis, prepuce, and sheath are arrested in development and the animal fails in some cases to protrude the penis during urination. Masses of smegma sometimes accumulate in the *fossa navicularis* or the urethral sinus about the urethral opening and may so compress the canal that urination becomes difficult. These accumulations of smegma occur mostly in geldings, apparently owing to the inhibition of castration upon sexual functions.

Phimosis and paraphimosis are sometimes observed in the stallion, usually following traumatic injury. The phimosis is usually caused by a narrowing of the preputial orifice due to inflammation of the

parts. Paraphimosis in the stallion is most frequently the result of traumatic injury, followed by swelling, with the increased weight, and paralysis of the retractor muscles of the organ. Paraphimosis is also a prominent phenomenon in dourine of the stallion, owing apparently to the advent of lesions in the spinal cord somewhat resembling those of syphilis in man. Paraphimosis also occurs in both stallions and geldings in conjunction with spinal diseases.

The method of handling is dictated by the character of the individual case: it may call for the mechanical suspension of the penis in order to ameliorate the edema due to gravity in the weakened organ; it may demand amputation of the penis, as already described.

The glans, prepuce, and penis of ruminants and swine are covered with extremely delicate, highly sensitive mucosa which is vulnerable to numerous pathogenic invaders. Accordingly primary penial tuberculosis of the bull, as already described, constitutes a highly important peril to the herd sire when caused to copulate with a cow suffering from genital tuberculosis. Ruminant and swine males commonly show the nodules of *infectious vaginitis* in the mucosa of the glans and prepuce, usually in a mild, unimportant type. Dairy bulls, however, not infrequently show important lesions like those of Fig. 102, which may be so virulent as to bar coitus owing to the severe balanitis. Its handling has already been discussed.

Owing to coital rupture of the prepuce, as indicated in Fig. 68, or because of other injuries or disease the prepuce may become adherent to the glans and prevent coitus. So far as known the lesion is beyond remedy. The enthusiastic advocates of artificial insemination might claim that the potential loss of a valued sire might be evaded through the procuring of semen from him by masturbation, but such course presents important objections.

The prepuce of the ruminant, as indicated in Fig. 66, is completely fused with the mucosa of the glans at the time of birth, and the two lamellae of mucosa separate as the young male nears puberty, after which he is able to protrude the penis. If the calf is castrated very early, the preputial sac does not develop and the steer unavoidably urinates within his sheath throughout life. So it occurs that steers sometimes suffer severely or even mortally from infection within the sheath. The pathology of the disease has not been determined. I have observed it in a herd of steers where a majority of individuals were importantly diseased, requiring extensive incisions in order to permit the escape of highly fetid pus and large masses of necrotic connective tissue: some of the animals succumbed.

POST-COITAL URETHRAL HEMORRHAGE

Injuries to the penis, prepuce, or sheath naturally lead to hemorrhage in some cases. The injuries may result from coitus, already considered, and I have known it to occur when obtaining bull semen for artificial insemination by the use of an artificial vagina. When adult bulls are caused to serve immature heifers, and especially when genital hypoplasia is present, laceration of the prepuce with consequent hemorrhage is invited.

I have observed profuse hemorrhage from the urethra after each coitus by a thoroughbred stallion. After the stallion dismounted, there was a gush of blood amounting to about one pint. The source of origin of the hemorrhage was not determined. It probably came from the seminal vesicles. I failed to learn of the fertility of the animal.

Chapter XXVII

DISEASES OF THE FEMALE GENITAL ORGANS

CONSIDERATION has already been given to hereditary and congenital defects and to hypoplastic development of the genital organs of the new-born. Tumors of the organs of reproduction, and infections believed to have a special affinity for the genital organs have also been described. There remains an endless variety of diseases involving one or more organs, largely ascribed to infections, errors in diet (want of vitamins or minerals), defective functioning of the hormone system, etc.

Atretic Graffian Follicles

Schmaltz[178] cites Käppeli as having enumerated in the ovaries of heifer calves aged three months an average of 75,000 Graffian follicles; at three years the number has dropped to 21,000, and after ten years to 2500. Dairy heifers commence to ovulate as early as six months post-natal and ideally ovulate at intervals of about 21 days. The common practice is to breed a dairy heifer at 15 months, at which date she should hypothetically have ovulated 13 times. After the termination of her first pregnancy she probably ovulates first at 30 days post-partum, and if rebred at 80 days so that she may again calve within one year she will be served at her third ovulation. Hypothetically then when a cow has reached three years she will have discharged 16 ova, while the number of ova within her ovaries will have been reduced from 75,000 to 21,000, or 54,000 ova will have disappeared, all of which, except 16, will have vanished unseen.

The many thousands of vanishing ova are said to have disappeared owing to atresia or atrophy. In general the atresia may be regarded as normal, but in numerous cases there are definite evidences of pathological processes. Thus in Fig. 41 are shown ovaries of a fetus and of a heifer calf with hemorrhages into follicles of gigantic ovaries, closely simulating the hemorrhages into the ruptured sacs after ovulation. Writers now and then relate finding ripe ovisacs in a fetus; rarely a new-born heifer develops nymphomania and numerous clinical evidences of disease of the ovisacs are noted. When heifers reach breeding age there are frequently observed ovaries with countless cysts up to 4 mm. in diameter, which presumably contain no ova. Careful study of these has not been made but limited observation indicates that such animals are of low fertility and that the numerous

defective follicles signify defective ovarian function. My impression is that the phenomenon is chiefly the result of genital hypoplasia owing to erroneous handling of the young calf, or it is present at birth as a congenital defect.

Some writers record laboratory studies of such ovaries but provide no clear explanation of their etiology. The most popular hypotheses are that the atresia is due to deficient vitamins in the food or to disturbances in endocrine secretions. What appears most informative is the enormous normal atresia of the follicles during the early years of life. The conclusion cannot well be avoided that the atresia is not uniform in degree and that in some cases the defective ovum may be sufficiently developed that a ripe ovisac forms and ruptures followed by fertilization and an imperfect new individual—amorphous, acardiac, asexual, and endless arrests in development referable to the defective Graffian follicle. It has been asserted that in some cases a cow or other female does not ovulate at a given estrum. It is not readily proven or disproven. The evidence is strong that the atrophy or arrest in a given follicle may be of such a character that the sac will not rupture, although sufficiently developed to induce heat; or that it may rupture and discharge an ovum not capable of fertilization; or that it is capable of fertilization but incapable of independent existence although viable if it makes vascular anastomosis with a viable twin or litter mate, as in acardiac monsters; or the ovum may have reached such maturity that it may develop independently into some type of monster.

Clinical observation teaches that the development of the follicles is under the control of physiological and psychological forces. If a cow suckles her young she does not come in estrum so early as the dairy cow from which the calf is separated at birth, although the food (vitamins, minerals), the endocrine secretions, the housing and handling remain the same in each case. The female rabbit and cat are said to not ovulate except coitus occurs. The coitus may be performed by a vasectomised male, after which pseudo-pregnancy occurs. It is not known that coitus does not stimulate physiological ovulation in all species of mammals. The psychological forces apparently exert a profound influence upon ovulation. Thus in Table XI, and in all other observations available for study, the dairy cows, deprived immediately of their calves, show highly erratic heat cycles following calving: most services go awry, the pregnancies induced are unsafe, the duration of gestation abbreviated, and most twins, defectives, and monsters occur among the early conceptions—the proper development of the ovisac and its contained ovum have been upset.

OVARITIS

Uncomplicated ovaritis in domestic animals is rare. There is observed in the cow an adhesive inflammatory process between the pavilion, the ovarian pocket and the ovary (Fig. 140) which may justify the inference that it is fundamentally salpingitis or pavilionitis, rather than ovaritis. This leads in some instances to tubo-ovarian abscess, causing complete destruction of the ovary.

The diagnosis of such peri-ovaritis can usually be authentically made in the cow by rectal palpation. In some cases adhesive strands are palpated which loosely bind the ovary to adjacent structures. Usually the ovary is completely encapsulated and can be recognized only as a spheroidal node in the ovarian area, rather than as the naked, directly palpable gland. If but one gland is involved, it may be removed in well selected cases, and the cow permitted to breed upon the other side. Usually there is no occasion for the removal.

CYSTIC DEGENERATION OF THE OVISACS
NYMPHOMANIA

Nymphomania occurs in all domestic mammals. The nymphomania of cows constitutes the most interesting and economically important type of mental disease known in veterinary medicine. It has not been sufficiently studied histologically or endocrinologically to justify any designation based upon its pathology. The term "nymphomania" i.e. sexual insanity, based upon its clinical phenomena, furnishes its best definition.

Bovine nymphomania is most common in cows of dairy breeds and attacks with greatest frequency those which are very high milkers. I have observed typical cases of nymphomania in dairy heifers not known to have been pregnant. Hence it would appear that, while heavy milking may largely prepare the way, it is not essential to the development of the disease. Furthermore I have observed typical nymphomania in Hereford heifers, a pure beef breed. W. W. Williams has observed nymphomania in dairy heifer calves only six to eight months old, arresting the growth of the calf and producing early senility, rendering the animal worthless. Nymphomania is unknown in pregnant, and in castrated females.

The advent of the disease is so erratic that it is of little value as a basis for determining the cause. Its appearance in young, unbred heifers or in those which have been bred without recognized conception has supplied no clue to the cause of the affection. It is observed most frequently in young cows which have calved or aborted two or

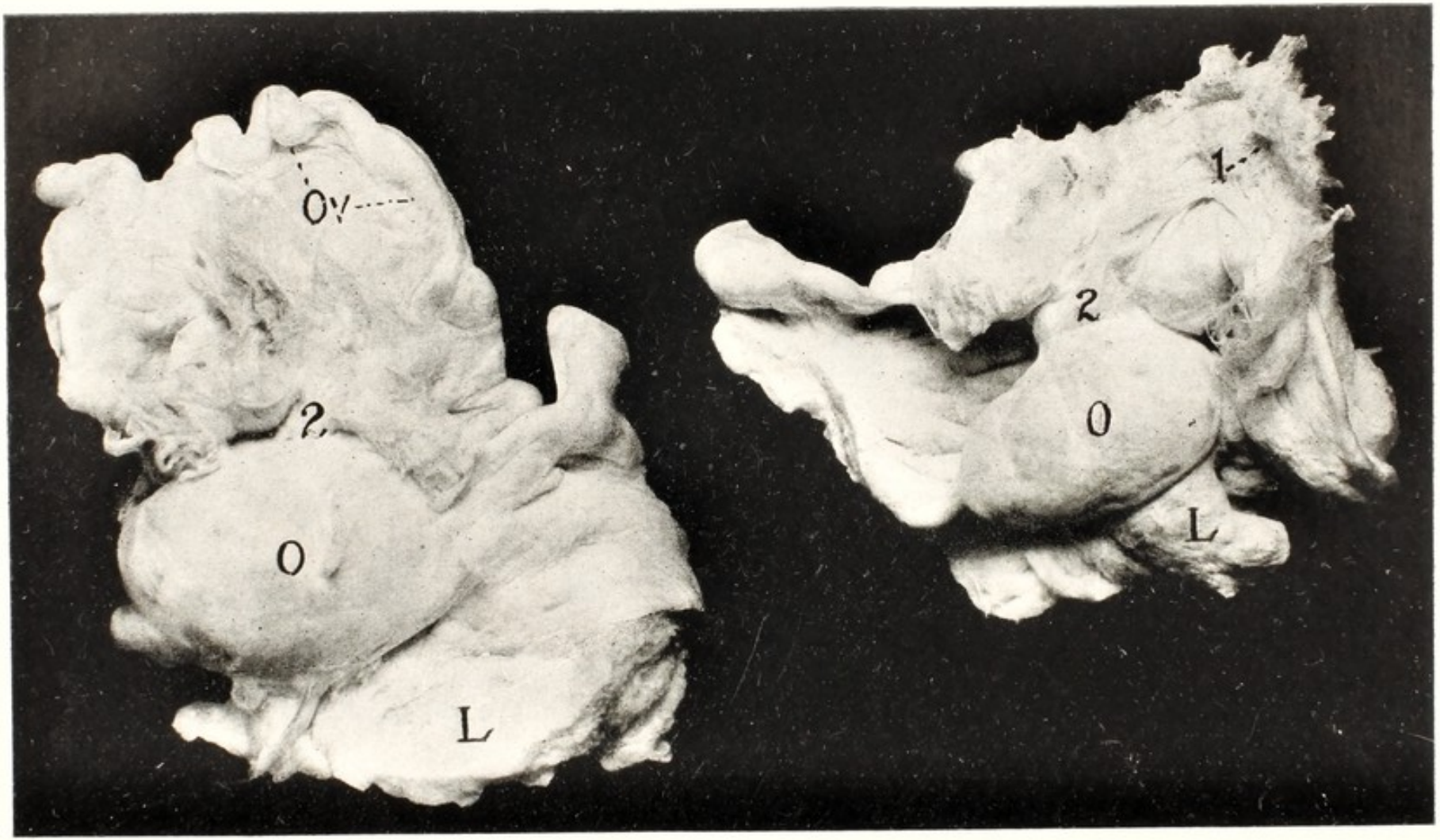


FIG. 140. Pair of Adherent Bovine Ovaries.

L, Ligament of ovary; O, ovary; Ov, oviduct; 1, ostium abdominale of the oviduct; 2, 2, bands of adhesions between the ovary, mesosalpinx and oviduct.

three times. Such animals are far more numerous than those which have calved four or more times, which are the healthiest cows, and generally most resistant to disease. Nymphomania is spectacularly linked with heavy milking. Fig. 141 illustrates a cow famous as the first in the United States to exceed forty pounds of butter in a week. Somewhat similar occurrences are frequent. High milking is associated with a rich diet and limited physical exercise, but the appearance of the disease in beef heifers, living entirely upon grass in the open field, staggers the conclusion that the basic cause is high feeding and close confinement.

In that great majority of cases which occur in pluripara, the disease appears at almost any period after parturition. Numerous cases become obvious within ten to thirty days post-partum, and according to my observations these cases are especially severe. Thus in the cow illustrated in Fig. 141, her record for butterfat production was spectacular, and her lactation was wrecked within a few days. While discussing the post-partum interval before rebreeding it was held that post-partum estrum at earlier than 60 days borders upon the pathological. Only a minority of cases appear early post-partum. Sometimes the disease develops after repeated unsuccessful services, and in cows which have been held out of breeding for prolonged periods.

The behavior of the cow at the close of the prior pregnancy has thus far afforded no definite clue to the fundamental cause. Frequently the cow has aborted, or has suffered from retained afterbirth or metritis, while in numerous cases the parturition and the puerperium have been considered as physiological. In some cases vaginal prolapse may have been observed during pregnancy, or the prolapse may develop during the nymphomania; sometimes there are present various diseases of Gartner's ducts and Bartholin's glands. The constant pathological heat and the persistent relaxation lower the general resistance to infection. The nymphomaniac cow usually has a mucopurulent vaginal discharge, which generally ceases immediately when the pathological estrum disappears, unless the accessory glands are involved.

The onset of nymphomania is usually, but not always, abrupt. In some cases, estrum seems infrequent for a time, or the opposite phenomenon of an abbreviated heat cycle and prolonged estrum prevails. The cow may be in estrum every 7 to 10 days and the heat be marked for 2 to 4 days. On the whole, when the onset is not abrupt, the estrual cycle is unstable and vacillating.

The cows which suffer most frequently are those which are largely

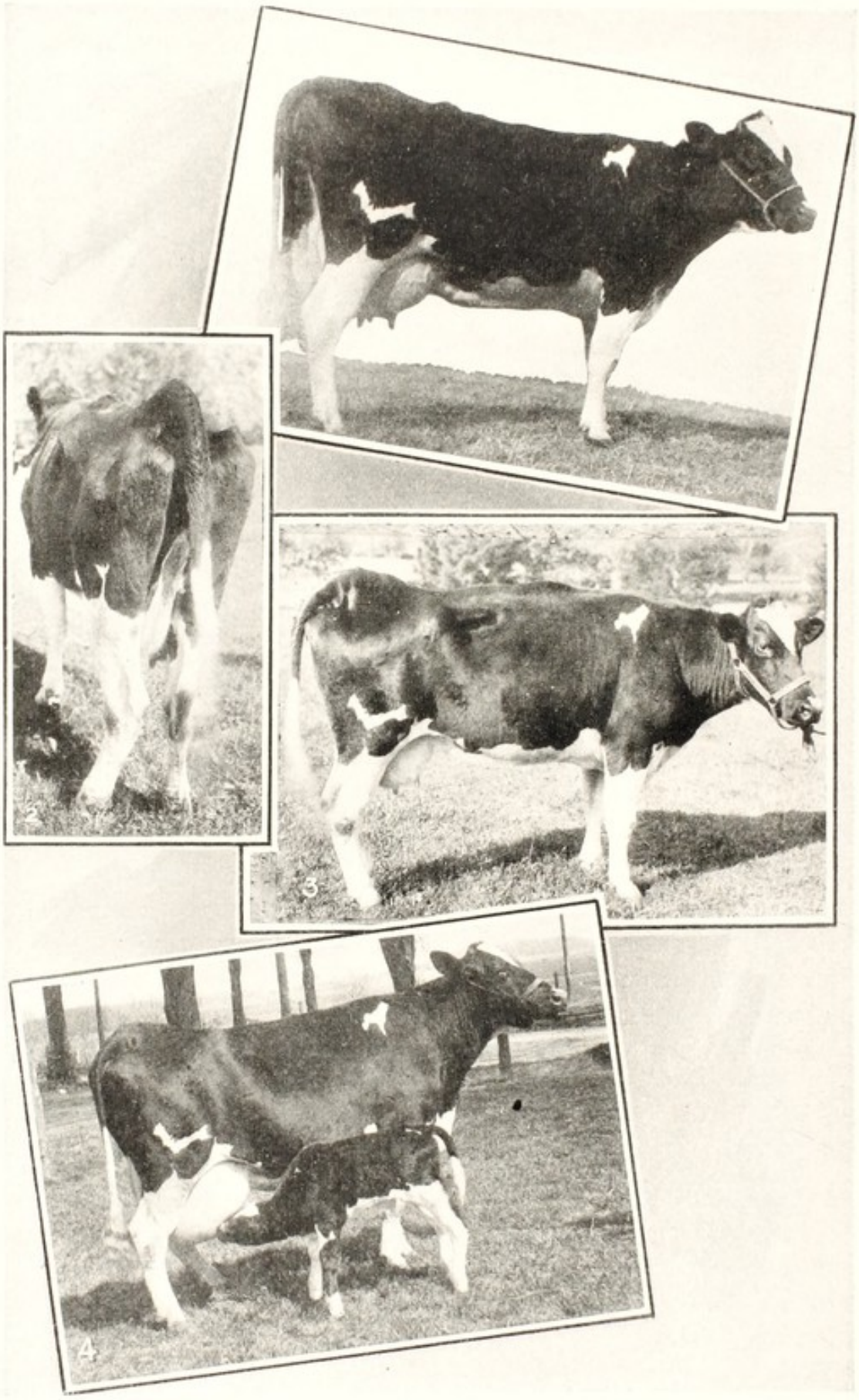


FIG. 141. Nymphomania of Cow.

1, The healthy animal; 2, rear, and 3, side view after the onset of nymphomania; 4, the recovered animal, the pelvic ligaments remaining sunken to some degree.

stanchioned, and are not free to exhibit fully their erratic behavior. Numerous cases are first revealed by unnatural bellowing. The bellowing is of increased frequency and the voice is of definitely lower pitch. The lowering of the pitch increases with the intensity of the disease, until the bellowing is readily mistaken for that of a bull: the cow becomes a *buller*.

When brought into the presence of other cows, the affected animal mounts her associates or permits them to mount her. Generally, though not universally, she will accept the bull repeatedly. In the more severe cases the nymphomaniac will mount, or attempt to mount, other species of animals, or she may mount an attendant not on his guard. In rare cases the cow will mount a low stall partition or other inanimate object, and sometimes attack a man as would an infuriated bull.

Along with the bellowing and coital mimicry, there appears a highly significant, so-called relaxation of the pelvic ligaments with spectacular changes in the morphology of the buttocks. This phenomenon is well shown in Fig. 141 in which the famous cow, K. P. Pontiac Lass, is shown before, during and after nymphomania. The phenomenon has been likened to an exaggeration of the relaxation of the pelvic ligaments immediately prior to calving or aborting. Such relaxation, though assumed to occur in all species of females just prior to parturition, is by far most evident in the cow. The preparturient relaxation is very inconspicuous when compared with that of nymphomania. The phenomenon is generally ascribed to a relaxation of the sacrosciatic ligaments. The hypothesis is difficult to follow.

A study of Fig. 142 will show that the bovine pelvis is quite evenly balanced upon the head of the femur. When the animal is in the standing position, the weight of the vast abdominal viscera is chiefly sustained as in a hammock by the abdominal muscles and the powerful linea alba. These, acting upon the pubic brim and sternum, serve to prevent the pelvis from tilting forward upon the head of the femur. When the prepubian tendon ruptures, as occurs in the pregnant mare (Fig. 167), the pelvis becomes tilted with depression of the cephalic end of the ilium and elevation of the ischial tuberosities. So in nymphomania, as illustrated in Figs. 141-42, the pelvis tilts forward upon the head of the femur, the ilial tuberosities drop, and the ischial tuberosities move dorsalward. The sacrum largely retains its relation with the ossa innominata, the lumbo-sacral articulation bends ventralwards, and the sacro-coccygeal articulation becomes elevated. The abdominal floor sags and the abdominal viscera tend to drop

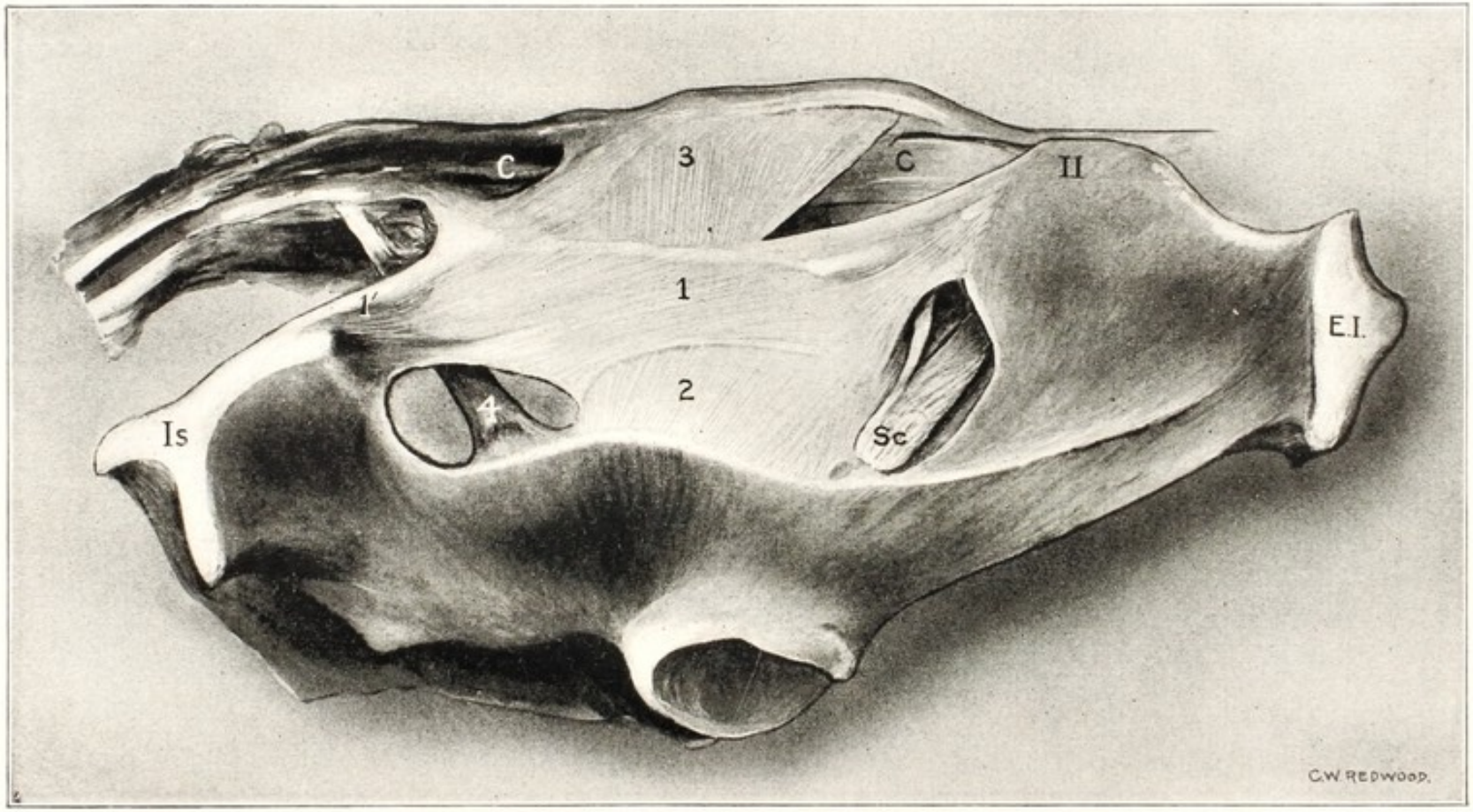


FIG. 142. The Pelvic Ligaments of the Cow.

1, Central portion of sacro-sciatic ligament; 1′, perineal border of ligament; 2, ischial portion of sacro-sciatic ligament; 3, sacral portion; 4, depressor coccygeus muscle; *I*, *I*, internal ilial tuberosity; *C*, *C*, coccygeal muscles; *EI*, external ilial tuberosity; *Is*, ischial tuberosity; *Sc*, sciatic nerve.

away from the pelvis and flank. This movement of the viscera away from the pelvic cavity creates a partial vacuum, and the sacrosciatic ligaments, with the muscles covering them, drop inward.

The relaxation of the pelvic structures disturbs the locomotor equilibrium of the posterior parts, so that the gait is unsteady and erratic. This predisposes the patient to fractures of the pelvis and

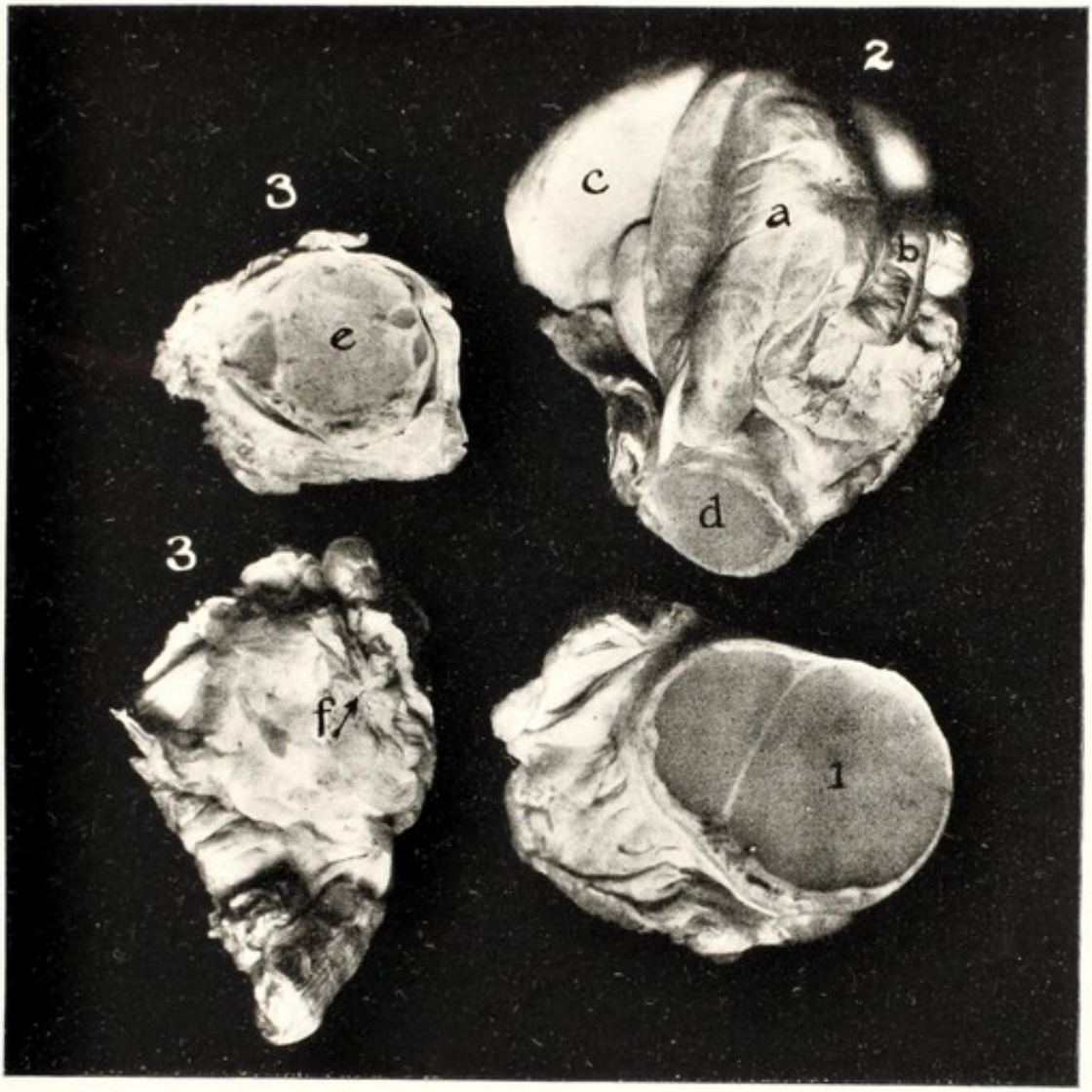


FIG. 143. Nymphomaniac Cysts and Cystic Corpora Lutea of Bovine Ovaries.

1, Nymphomaniac cysts; 2, cystic oviduct and corpus luteum; *a*, cystic pavilion; *b*, cystic oviduct; *c*, cornu; *d*, cystic corpus luteum; 3, *e*, encapsuled corpus luteum; *f*, ostium abdominale of oviduct.

posterior limbs, when mounting, or being mounted by other cattle. It seems also that the bones become unduly fragile, as contended by Küst.[112]

The nymphomaniac cow discharges varying quantities of thin mucus from the vagina similar to that observed in heat. As the disease progresses, the uterus, vagina, and vulva become atonic and the

genital discharge may become muco-purulent. The patient becomes emaciated, and the hair rough and lustreless.

Rectal palpation reveals one or more cysts in the ovaries, usually varying between 1 and 2.5 inches in diameter. In volume they occupy a middle ground between the small, multiple congenital cysts of Fig. 41 and the voluminous, solitary cysts of Figs. 56 and 60. Rarely only a single cyst is found. Usually two or more cysts are present and both ovaries are commonly involved.

The walls of the cysts are thin and tense. They may usually be ruptured by manual compression per vaginam, but in some cases are too firm to permit this. They differ markedly in this respect from the physiological ovisac, which is distinctly smaller, rarely more than 0.5 inch in diameter and, with its thin walls, must be palpated gently lest rupture occur.

So far as may be determined, the cysts of nymphomania are exclusively the result of pathological changes in the ovisacs which destroy the primitive ovum and cause an excessive secretion of follicular liquid, imprisoned by tough follicular walls. They have not been adequately studied histologically or chemically. No ovum and no lutein tissue have been found in them. Cystic corpora lutea, to be described later, may be of similar form, size, and consistency to those of nymphomania, and may cause confusion in diagnosis. So far as known, cystic degeneration of the corpus luteum does not cause nymphomania, but instead bars heat over a long period of time.

Nymphomania has not been produced experimentally. Instances occur suggestive of heredity: a nymphomaniac cow, and one or more nymphomaniac daughters and granddaughters, are sometimes simultaneously discarded. Table VIII shows a remarkably high ratio of nymphomania in the daughters, granddaughters, and great granddaughters of Sire X.

The fundamental cause of nymphomania—the cause of the follicular cystic degeneration—is unknown. Hypothetically it is attributed to an endocrine disturbance, but it is not known which of the endocrine glands is at fault. Ovariectomy terminates the disease.

The prognosis is unfavorable when prolonged history of reproduction is used as a meter. Generally, as in other genital diseases, an abbreviated history is applied as a standard for measurement and the prognosis may be made to appear fair. Thus Fig. 141 may indicate to the casual reader that the cow made a brilliant recovery. The illustration fails to show that the cow was sterile for about four years, with the inevitable expense of keep and veterinary service, that the calf was non-viable and perished when only a few days old, or that

the cow reacted to tuberculin and was destroyed. Or if we turn to the bovine monster illustrated in Fig. 26 as an example, it is found that the cow had been handled for nymphomania prior to conception. In Table VIII with a high prevalence of nymphomania, seven cows, handled nine times for nymphomania, produced after treatment a total of five calves. One of these (No. 12), was handled twice for nymphomania. She produced one daughter which was bred (No. 12′) which also had nymphomania. Williams and Williams[241] report 34 cases, seven of which had calved, ten were pregnant, ten had apparently recovered but were not yet pregnant and seven had been destroyed as hopeless. An equal number had calved once (21 per cent) and had then been destroyed as non-breeders. One had calved and was again pregnant. The meter applied covered too brief a period to reveal the ultimate economic result. When discussing twins in Chapter III, it was noted that there was an apparent linkage between nymphomania and twins. Clapp[31] was cited as having shown that twinning is highly probable if the cow is bred early during the convalescence from nymphomania. The cow illustrated in Fig. 141 had aborted twins in her second prior gestation. The twin ratio is excessive in Table VIII, along with the high incidence of nymphomania. The ultimate prognosis of nymphomania, according to the meter advocated in this treatise, contemplates the ratio of affected cows which so far recover that they again produce calves, the number of calves produced by each and their quality. The data for such prognosis do not justify a final conclusion. The outlook appears discouraging.

No satisfactory method of handling has yet been devised. The fact that the removal of the ovaries terminates the malady suggests that the immediate cause of the symptoms is the cystic degeneration of the ovisacs, and that consequently the prompt rupture of the cysts should prove beneficial. Clinical experience partly justifies this belief. The records are incomplete because as a rule they fail to indicate the promptness with which each recurring cyst is ruptured. The ruptures have been chiefly carried out by the veterinarian in person, and in a large ratio of cases he is unable to give constant attention. Clapp[31] submits data upon twenty-two cows handled because of cystic ovaries, of which five suffered a second time after recovery from the first attack and having been pregnant. Twenty-six pregnancies followed the twenty-seven cases. Apparently the milder cases were not classed as nymphomaniacs and, since the cysts were ruptured early, the extreme symptoms were not developed. It seems clear that Clapp, as manager of the herd, in constant attendance, and cooperating with a highly capable veterinarian, had excellent results. The final breed-

ing efficiency of the cows and the quality of the progeny are not revealed. The inevitable conclusion is that it is highly advisable that the cow be subjected to constant handling and that each cyst be promptly ruptured. As a rule the veterinarian is not in a position to do this personally. Some would criticize the plan of Clapp, in which a layman ruptures the cysts under the general supervision of the herd veterinarian. In most cases it is the only plan within economic bounds by which the prompt destruction of the cysts may be accomplished, and is deserving of thoughtful consideration.

The administration of various endocrine substances has been tried, but has not yet achieved definite results. Williams and Williams[241] tested whole ovarian extract, extract of the corpus luteum, adrenalin and pituitrin upon numerous cases, but attained no outstanding success with any.

Spaying eliminates the disease, and should be invoked in those cases where the outlook for economic recovery is bad. If applied early during good lactation, the value of the cow as a dairy animal may be prolonged. Generally it may be relied upon as an important aid in restoring the physical health, and will materially enhance the value of the animal for slaughter.

During the course of the malady the cow should be carefully and liberally fed. It is vain to expect that, in severe cases like Fig. 141, the general state of nutrition may be maintained. The fact that nymphomania rarely occurs in virgin heifers of the beef breeds kept wholly at pasture, and that most cases involve closely stabled cows suggests that the patient be given abundant freedom, preferably at pasture. She should be isolated to prevent her from mounting or being mounted by other animals.

When a nymphomaniac cow has apparently become convalescent, care should be taken that she shall not be bred too early. The data of Clapp are highly instructive, showing that a very discouraging ratio of cows bred at less than 35 days after the rupture of the last cyst suffered from twin pregnancy, while among those cows held out of breeding for 35 or more days, twinning was not notably common. Much the same object may be attained by deferring breeding until the second apparently normal heat. Palpation of the ovaries, at the first heat after apparent recovery, frequently reveals the presence of two ripe ovisacs—the logical antecedent of twins, free-martins and monsters. When the ripe ovisacs rupture, their place is taken by two or more corpora lutea. The multiple corpora lutea indicate multiple ovulation and that the cow is not yet fit for service, whereas the presence of a single corpus luteum which follows apparently normal

heat indicates that recovery has probably occurred and that service may be allowed with comparative safety.

CENTRAL CYST NYMPHOMANIA IN THE COW

Rarely there occurs in cows an obscure, persistent type of nymphomania associated with a small cyst located centrally in the ovary. I have observed two cases, one of which seen with Udall[183] has been recorded.

The symptoms have been those of comparatively mild nymphomania. The cow mounts her associates, but may not allow them to mount her. There is a discharge of clear, stringy mucus from the vulva.

Upon rectal palpation no large peripheral cysts like those of common nymphomania are observed. Unless the examiner is alert, the ovary is probably designated as normal and diagnosis fails. If a critical examination be made, it is found that one ovary is slightly enlarged and spheroidal. Further careful palpation indicates that the spheroidal ovary is tensely fluctuant, compared with the normal gland on the other side. The symptoms, combined with the finding of the spheroidal fluctuant gland, justify the diagnosis of small central cyst.

The cause of such cysts is unknown. The cyst cannot be ruptured by digital compression. The affected gland should be picked up per rectum, drawn back over the vagina, grasped per vagina, and the cyst punctured with the ovarian scalpel (Fig. 43) through the vaginal wall.

EQUINE NYMPHOMANIA

Nymphomania is less frequent in mares than in cows and presents important differences in its clinical phenomena. There are two types. The more frequent type occurs chiefly in closely housed, irregularly worked mares not generally used for breeding, and therefore it is not usually known if they are fertile. The inference follows that many, if not most cases are not attributable to cystic degeneration of the ovisac with death of the ovum as in the cow. The embryology of the ovaries differs in important particulars in the two species. The ovaries of fertile mares present gross variations in structure which do not at present admit of authentic explanation. I had occasion to spay a group of twenty high-grade Percheron mares, three to four years old. They had been grown on the open range at a high altitude in the Rocky Mountains and, so far as could be judged by appearances, were in ideal physical health. The fertility of these plains mares is phenomenal. The group had not been bred and most of them had not

been worked. Each of this group of highly fertile mares had ovaries 3.5 to 4.5 by 2.0 to 2.5 inches in diameter. The chief volume of these was due to a large number of cysts as great as 1 to 1.5 inches in diameter. So far as is known, these cysts were in no sense pathological. Ovulation occurs at the hilus of the gland. The general surface is dense, compressing the cysts and giving to the gland an even contour. Bengisch[13] reports that cysts of medium size are highly common in mares without interfering with the heat cycle or with fertility. This prevalence of non-pathological cysts is, according to my observations, the rule in draft mares. On the contrary, the ovaries of the thoroughbred mares and of the common mares largely used for anatomical studies are frequently, if not generally very much smaller and largely or wholly devoid of cysts. It is difficult to draw a boundary line between healthy and pathological cystic ovaries.

The usual type of nymphomania is seen chiefly in young or middle-aged mares. I saw one case in a mare mule, which was so vicious that spaying was necessary.

The symptoms of the ordinary type of nymphomania usually appear shortly before or during estrum, and subside at the close of heat. Usually the first symptoms are those of abnormal excitability and irritability, commonly pertaining to estrum. She attempts to bite or kick other horses. When hitched with another animal to a wagon, she will crowd against the pole and may kick over it. The ears are laid back, the tail vigorously switched, and she urinates frequently. The urine is ejected with unusual force. In numerous cases she gives vent to loud cries of anger. One mare in our clinic exhibited the mania almost exclusively when alone in her box stall, at which times she would stamp and kick violently, frequently kicking one of her own legs. If the owner or other person entered the stall, she became calm, began eating or fondled the person with her nose. The duration of the maniacal periods had gradually increased until at the time of observation they lasted 7 to 10 days. The mania ceased completely after ovariectomy.

The vulva is often somewhat swollen and the vulvar mucosa injected with an increased secretion of mucus. The clitoris is erected. The ano-vulvar region seems especially sensitive and the animal violently resents the handling of these parts. She resists the raising of the tail for the placing of the crupper or for other manipulation. While being driven, should she accidentally or intentionally get her tail over the rein, she grasps it firmly and kicks violently. The mania is usually periodic at first, but tends to increase in intensity and to become more or less constant, until the animal finally becomes an outlaw, highly dangerous to her attendant.

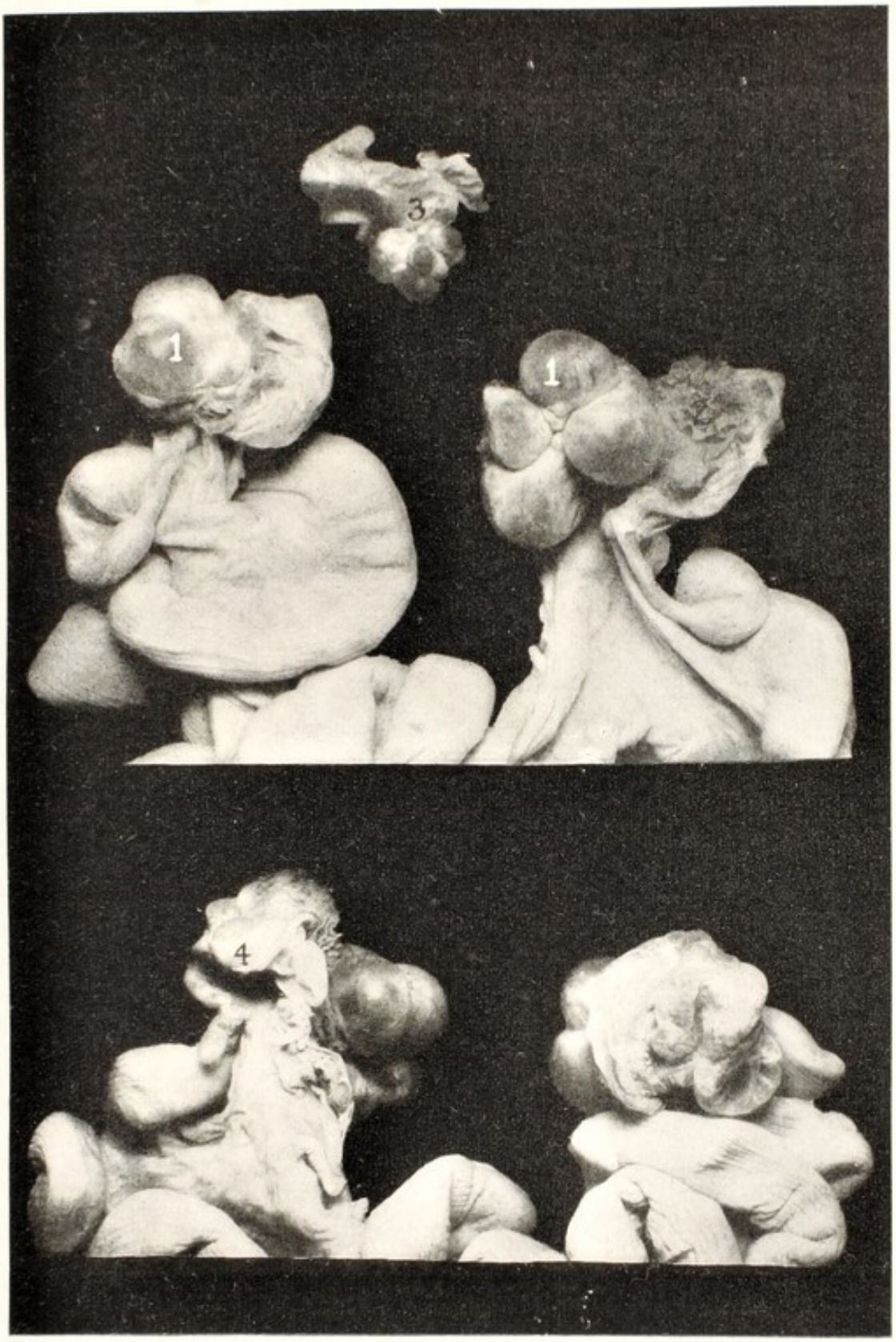


FIG. 144. Cystic Degeneration of Swine Ovaries.

1, 1, Cystic ovisacs; 3, a normal swine ovary with ripe ovisacs; 4, a small parovarian cyst.

Since nymphomania occurs chiefly in mares not used for breeding, the principal end usually sought is the elimination of the mania. This is best attained by ovariectomy. The operation sometimes fails, es-

pecially if too long deferred. Should the gripping of the reins by the tail constitute the chief vice, or persist after castration, the best remedy is caudal myectomy, by which eight or ten inches of the depressor coccygeus muscles are removed, leaving the animal with an apparently natural tail, with due control of lateral and dorsal movements, but without power to grip the reins dangerously.

Several decades ago, clitoridectomy was frequently applied for the relief of nymphomania, and many successes were reported. Whether

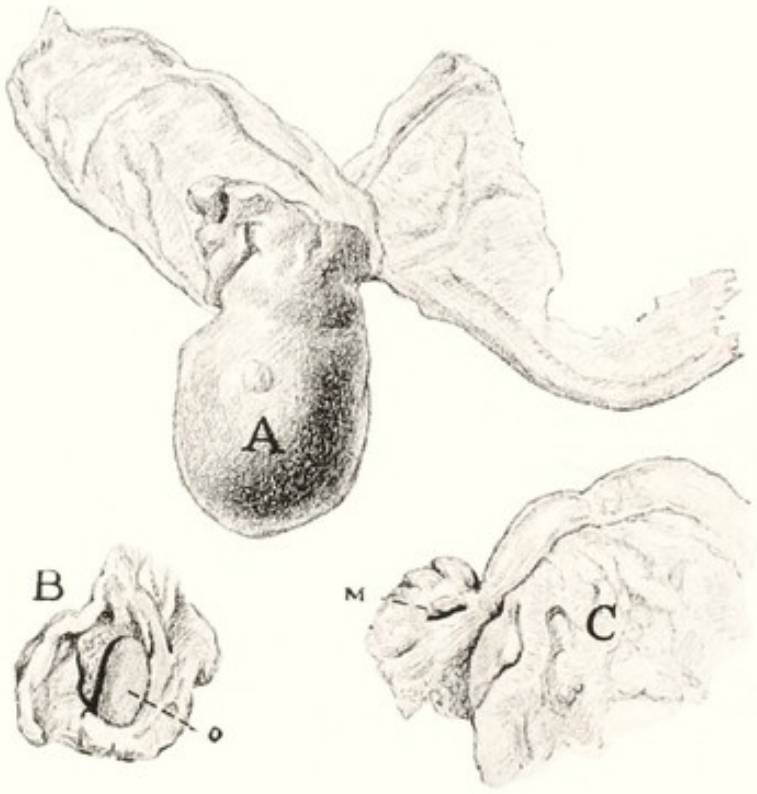


FIG. 145. Cystic and Normal Ovaries of Bitch.

A, Cystic ovary; *B*, normal ovary with the pavilion of the oviduct turned back in order to expose the ovary; *C*, normal ovary, normally covered by the pavilion of the tube; *M*, the fenestrum in the ovarian sac, communicating with the peritoneal cavity.

the benefit was owing to the elimination of the organ or to the discipline attending the operation, is not quite clear. The operation is simple and safe.

How frequently nymphomania produces sterility in valuable brood mares is not known. I observed one case of nymphomania in a thoroughbred mare which had long been hopelessly sterile. The mania developed suddenly during the breeding season and continued for some weeks. The mare exhibited great sexual excitement, moved about almost constantly in her paddock, sometimes at a rapid walk, at other periods at a trot. She whinnied frequently and loudly and at times was bathed in a foam of sweat. Handling was not attempted.

NYMPHOMANIA IN SMALL ANIMALS

Nymphomania is occasionally seen in the sow. The clinical phenomena and the cystic degeneration of the follicles (Fig. 144) so far as known, are parallel with those observed in the cow. Similarly, cystic degeneration of the ovisacs occurs in the bitch, as indicated in Fig. 145. Nymphomania due to cystic follicles is of minor interest from the standpoint of reproduction. It is controllable by spaying.

INTRA-FOLLICULAR HEMORRHAGE

I have in my collection the genital tract from an abattoir heifer, illustrated in Fig. 146, in which the right ovary contains a number of large cysts which have increased its diameter to four inches and its

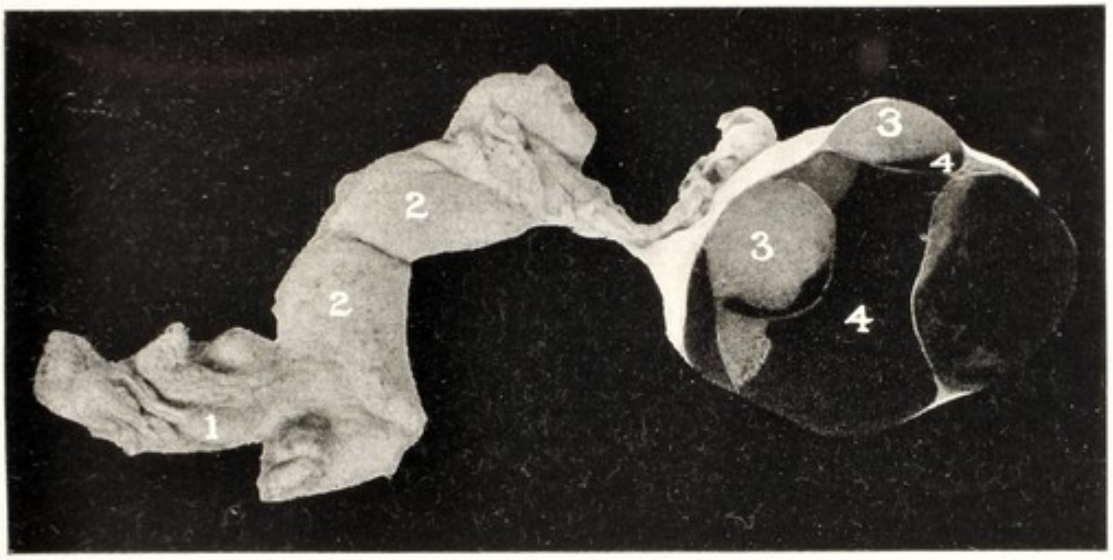


FIG. 146. Intra-Follicular Hemorrhage in the Ovary of a Heifer.

1, Right ovary and pavilion; 2, 2, the uterine cornua, the line midway between the numerals indicating the cervical area; 3, normal follicular fluid; 4, bloody coagula within the cysts. *The defect was presumably congenital.*

weight to about two pounds. Some of the cysts contain a clear lymph. Others contain blood and lymph mixed in varying proportions, some of them being filled almost entirely with blood. No lutein tissue is recognizable.

The genital organs illustrated were those of a heifer which had not been pregnant. The case suggests caution upon the part of the practitioner. It is difficult, if not impossible, to make a safe clinical diagnosis. Ovarian cysts, or cystic tumors of large size, should on no account be ruptured and the contents emptied into the peritoneal cavity. If an operation appears indicated, it should consist of the removal of the gland after laparotomy.

POST-OVULATION HEMORRHAGE INTO THE FOLLICULAR CRATER

Immediately after ovulation there is physiologically a slight hemorrhage into the crater of the ruptured ovisac in the cow, to form a very small hematoma which occupies for a brief time the center of the corpus luteum and then disappears.

Pathologically there occurs at times an abnormal amount of hemorrhage into the crater of the ruptured follicle. Clinically I have recognized extensive blood clots hanging from the crater, which are readily detachable. In one instance the hematoma was more than 3 inches in diameter. Presumably considerable blood had escaped into the peritoneal cavity in addition to that which remained attached to the ovary. Now and then a cow appears quite unwell at the close of her estrual period. It is not improbable that hemorrhage from the follicular crater may at times be responsible for the symptoms. The clot, which is fairly firm, breaks up under digital compression with a characteristic grating.

The cause of such hemorrhage is unknown. Inferentially it may be fundamentally like the severe, and sometimes fatal hemorrhage following the dislodgment of the corpus luteum. So far as known, the prognosis is favorable. It is not known that any interference is necessary or advisable.

Diseases and Abnormalities of the Corpus Luteum

PERSISTENT, RETAINED, OR EMBEDDED CORPUS LUTEUM

There is no universally accepted definition of retained, or persistent corpus luteum. The body is the physiologic successor to ovulation, and its color and duration are dependent upon the fertilization and viability of the ovum. When pregnancy ensues the corpus luteum physiologically persists in the mare to about midterm; in the cow it remains throughout pregnancy, and recognizable heat ordinarily recurs at about 60 days post-partum.

The actual physiology or function of the corpus luteum is none too clear. It has been noted in Chapter XII that artificial abortion may be induced by removal of the corpus luteum. Nevertheless the corpus luteum of the mare physiologically disappears in midpregnancy without leading to abortion. In both mare and cow estrum and coitus sometimes occur during pregnancy without being followed by abortion. Presumably the estrum and coitus are due to the disappearance of the corpus luteum of pregnancy. The relationship between the corpus luteum and the state of the uterus is somewhat

confusing. It is stated that the presence of the corpus luteum in the bovine ovary inhibits the expulsive contractions of the uterus and the emptying of its contents. Nevertheless it persists, without recognizable atrophy or other changes, to the end of gestation and for several weeks thereafter. The presence of the corpus luteum in the bovine ovary does not prevent abortion or calving. If, however, the corpus luteum is expressed from the ovary of the pregnant cow, she promptly aborts. When the typical hematic bovine mummy is present in the uterus, the corpus luteum retains its volume, sinks into the ovary and both corpus luteum and mummy persist indefinitely. The corpus luteum persists in trichomonad pyometra. If the pus be siphoned out, the corpus luteum atrophies, ovulation occurs, and the uterine contents are expelled. Or, if the corpus luteum be surgically removed, estrum and ovulation occur and the pus is expelled.

Two types of retained corpus luteum may be said to exist, that of pregnancy and of estrum. When the corpus luteum is retained, because of the presence of a mummy (bovine), a macerating fetus (trichomonad pyometra), puerperal pyometra or other forms of uterine disease, the lutein mass tends to sink toward the center of the ovary: it becomes an embedded corpus luteum, encapsulated within the ovarian tunic. Upon rectal palpation the corpus luteum does not stand out with the normal prominence above the general surface of the ovary.

The removal of the embedded bovine corpus luteum constitutes a valuable method for handling the hemorrhagic mummy, to be more fully discussed later. Trichomonad pyometra may be relieved and the macerating fetus expelled by siphoning out the pus, thus mechanically unloading the uterus. This failing, the dislodgment of the corpus luteum is greatly facilitated by having removed the pus, after which the dislodgment of the body may beneficially stimulate utero-ovarian function. When the corpus luteum is retained and embedded following retained placenta, metritis, and pyometra, the dislodgment may be of distinct remedial value. Usually it may be dislodged by digital compression, but occasionally it is essential to puncture the ovarian capsule with the ovarian scalpel (Fig. 43). It may prove desirable or necessary in some cases to resort to laparotomy in the right flank and thus directly reach the ovary. The operation is readily performed on the standing cow.

Hess[89] and his followers emphasized the persistence of the corpus luteum of estrum, as an important cause of bovine sterility. Not a few veterinarians adhere to these teachings, although more recent studies have modified the conclusions of three decades ago.

It is difficult to determine the persistence of a corpus luteum of heat as a distinct pathological entity. It has been related in Chapter XI that a group of heifers under experiment, fed on a low diet, failed to come in heat. A change to a reasonably nutritious diet was promptly followed by heat and conception. Presumably the corpora lutea of estrum had remained in their ovaries for months. It would be misleading to designate them as retained. Had they been dislodged during the starvation, the heifers would probably have come in heat. Had they been bred during the artificially induced estrum, they would probably have conceived and the pregnancies would almost certainly have ended in disaster. The prolonged duration of the corpus luteum of heat constitutes a valuable safeguard against conception of a female not physically competent to withstand pregnancy.

Embryonic abortion may be cited as a second example of confusion regarding a corpus palpated in the ovary. Such abortions pass unobserved in the vast majority of cases, and the condition is designated sterility. If a cow aborts unseen at 30 days and, examined 10 days later, is found non-pregnant, if her ovary contains a corpus luteum, a persistent yellow body of estrum will probably be diagnosed. In reality it is the corpus luteum of pregnancy, which constitutes a valuable safeguard against conception until the reproductive system shall have recovered its vigor. Causing service to follow the dislodgment of such a corpus luteum, is courting disaster.

Some instances of so-called retained yellow bodies, without recognizable manifestations of heat, are the consequence of imperfect follicular development. If such animals are repeatedly examined, corpora lutea in various stages of subnormal development may be found. They do not usually tend to become embedded; neither are they markedly protuberant. If such cases be closely followed, the presence of a ripe follicle with slight uterine engorgement, but without definite signs of heat, may be found.

The dislodgment of the persistent corpus luteum in prudently selected cases is highly commendable. Some current writers advocate the wholesale removal of corpora lutea as a panacea for sterility, regardless of the basic cause. The decision to remove surgically the yellow body should be based upon conclusive evidence that its presence is interfering materially with the physiological sex cycle. If an apparently healthy cow has gone over 90 days post-partum without having shown recognizable signs of heat, and it is desirable that she be bred, it may well be concluded that the corpus luteum is at fault and its removal considered. Even then the dislodgment of the yellow body should be regarded as experimental and as an aid in authentic

diagnosis. If heat appears three to five days later, that does not constitute proof that she is fit to be bred. If the cow is again in apparently normal heat after an interval of about twenty-one days and seems in all respects healthy, breeding may be advisable. In this way the artificially induced heat is employed as a stimulant to the restoration of the physiological sex cycle.

The technic for the dislodgment of the corpus luteum is described in Chapter XII.

The principal dangers associated with the removal of the corpus luteum are hemorrhage and the lighting up of latent infection. The dislodgment of deeply embedded corpora lutea from markedly fibrotic ovaries may possibly involve the severing of important arterioles. Hemorrhage of dangerous character may ensue without important vascular wounds. Thus in Fig. 42 the fatal hemorrhage could not be ascribed to injuries to the vessels about the yellow body, but was apparently caused by the profound upset in the sexual vascular system by the abrupt removal of the body. Similarly I have observed clinical symptoms of critical hemorrhage following the ready expulsion of hypertrophic corpora lutea of estrum. The gigantic size of the yellow body is in itself proof of a pathological condition. A colleague reports a case in which a cow died a few hours after dislodgment of the corpus luteum. No important hemorrhage occurred from the crater which the yellow body had occupied. The uterus was tremendously engorged and there was a general oozing of blood from it.

When tubal disease is present, any attempt to remove the corpus luteum, whether evidently pathological or apparently normal, is highly perilous, owing to the great danger of increasing the virulence of the tubal infection. W. W. Williams personally relates his observation in a herd where the wholesale removal of corpora lutea was followed by widespread, virulent salpingitis, with adhesions, which necessitated the discard of almost the entire herd. Dislodgment of the corpus luteum is not uniformly safe or beneficial. Like other operations it may be highly useful in prudently selected cases, and it may lead to disaster when wrongly applied.

CYSTIC DEGENERATION OF THE CORPUS LUTEUM

When the Graffian follicle ruptures, limited hemorrhage occurs within the crater. The lutein tissue is elaborated from the follicular walls. For a time the crater remains open at the point of follicular rupture and a central cavity exists. Later the ovulation-rupture heals and the enveloping membrane of the ovary resumes its continuity. Physiologically the corpus luteum of estrum and of pregnancy dis-

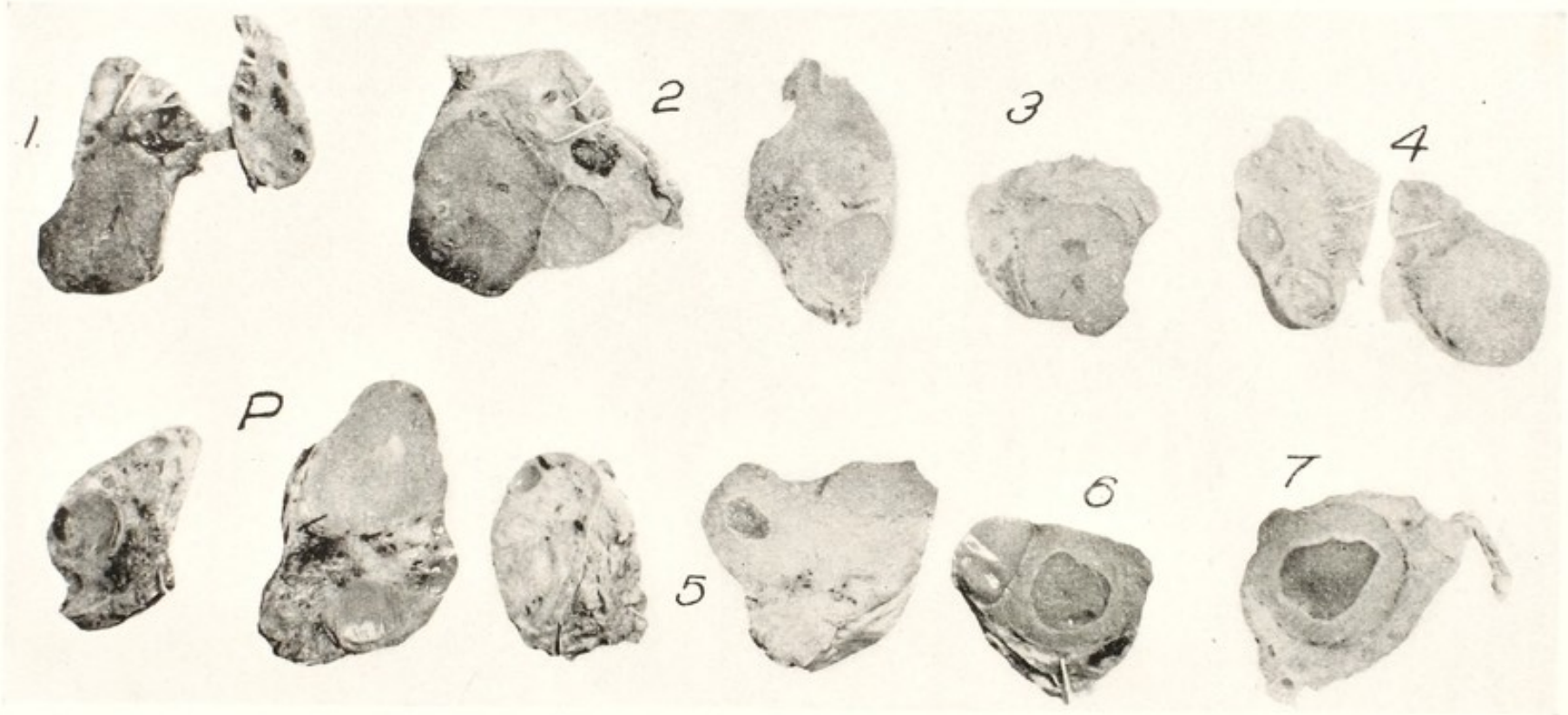


FIG. 147. Cystic Corpora Lutea of Cow.

1-7, *Ovaries with cystic corpora lutea of varying degrees, shown with their mates; P, a normal corpus luteum of pregnancy.*

appear by general resorption, and the lutein mass gradually decreases in size but retains its solidarity.

Pathologically the yellow body undergoes cystic degeneration, one of the most frequent types of ovarian disease. The cysts vary greatly in size, as indicated in Figs. 147 and 148. In Fig. 147 the volume of the corpus luteum and of the ovary is not notably affected; in Fig. 148 there is gross enlargement of both corpus luteum and ovary. The cysts in the latter case are as large as those in nymphomania, but so far as known do not cause that malady. The chief clinical history is that of delayed estrum. Observed chiefly in cows designated as *sterile*, it may be the successor to embryonic abortion. There is no reason to assume that it causes the abortion, or vice versa. A more probable hypothesis seems to be that the cystic degeneration and abortion (called sterility) are both due to a common cause.

The diagnosis in the live animal is not uniformly possible. Rectal palpation may reveal slight fluctuation in such ovaries as 6 and 7 in Fig. 147. If pressure is applied, the cyst palpably ruptures and the lutein mass collapses. If the pressure is continued, the collapsed mass is forced out and readily identified. Diagnosis in Fig. 148 is more difficult. The cyst is readily ruptured, but the practitioner needs be keenly alert to palpate the lutein vestige at *X*. Finally the insignificant volume of lutein tissue at *X* in Fig. 148 may wholly disappear and there remain simply a cyst, usually somewhat less tense, but otherwise not differing palpably from that of nymphomania. The clinician has the highly valuable clinical fact of the absence of nymphomania to aid him in arriving at a diagnosis. Cystic degeneration of the corpus luteum in sterile cows is often associated with tubal disease. In many cases, like Figs. 149 and 153, the ovary is completely encapsulated within the pavilion of the oviduct and the adjacent tissues. The ovary is not clearly palpable, but appears as a tumor at the normal location of the gland, firmly adherent within the surrounding tissues. Almost invariably such adherent ovaries contain a cystic corpus luteum. Rarely tubo-ovarian abscess (Fig. 156) is present instead.

The corpus luteum of pregnancy may undergo cystic degeneration during gestation. This may lead to estrum and coitus. The coitus is not known to cause abortion. The cystic degeneration of the corpus luteum of pregnancy may be present in an advanced state within two to three weeks after calving.

The cause of the cystic degeneration of the corpus luteum has not been determined, nor has any material study been devoted to the

subject. When combined with salpingitis, as in Figs. 149 and 150, a common origin is logically suggested. If the tube contains an infection at the time of ovulation, the invasion of the crater of the follicle from the tube becomes highly probable. Studies of these cysts in the abattoir indicate that in many instances the foundation is laid at the time of ovulation. As in the gonorrheal salpingitis of woman, the tubal infection may be conveyed by the male at the coitus of fertilization,

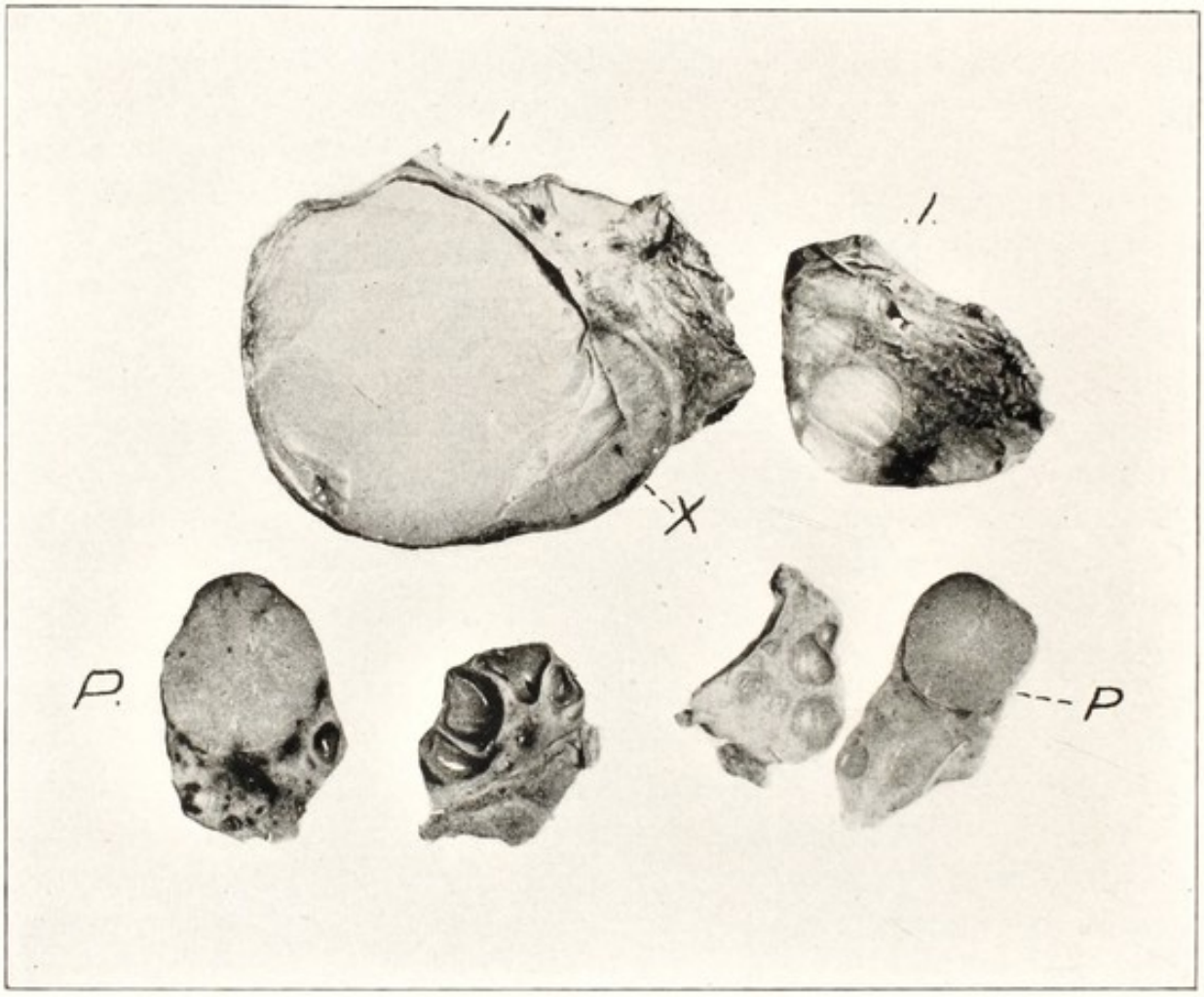


FIG. 148. Large Cyst of Corpus Luteum of Cow.

1, 1, Pair of ovaries, that on the left having a very large cyst with remnants of lutein tissue at *X*; *P*, *P*, paired ovaries from pregnant cows.

as indicated in Fig. 151. If pathogenic organisms are present in the cysts, they are not usually capable of causing suppuration.

The handling of cystic degeneration of the corpus luteum, when associated with tubal disease, is hopeless in so far as the restoration of fertility upon the affected side is concerned. If the ovary is only slightly adherent to the pavilion of the tube, the expression of the yellow body, by maiming the ovarian tissues, virtually assures perilous extension of the infection resident in the oviduct. Statistics

are not available for a highly dependable prognosis in such non-adherent cases as shown in Fig. 149. Apparently the prognosis is fair or good, if one can exclude the presence of tubal infection. If chronic metritis is present, tubal infection may well be suspected. The involved animals are usually at best of low fertility. There is no definite evidence that the dislodgment of the yellow body removes the basic disease; it may be merely an expression of a more deeply entrenched cause. In cases like Figs. 150 and 151, which are beyond

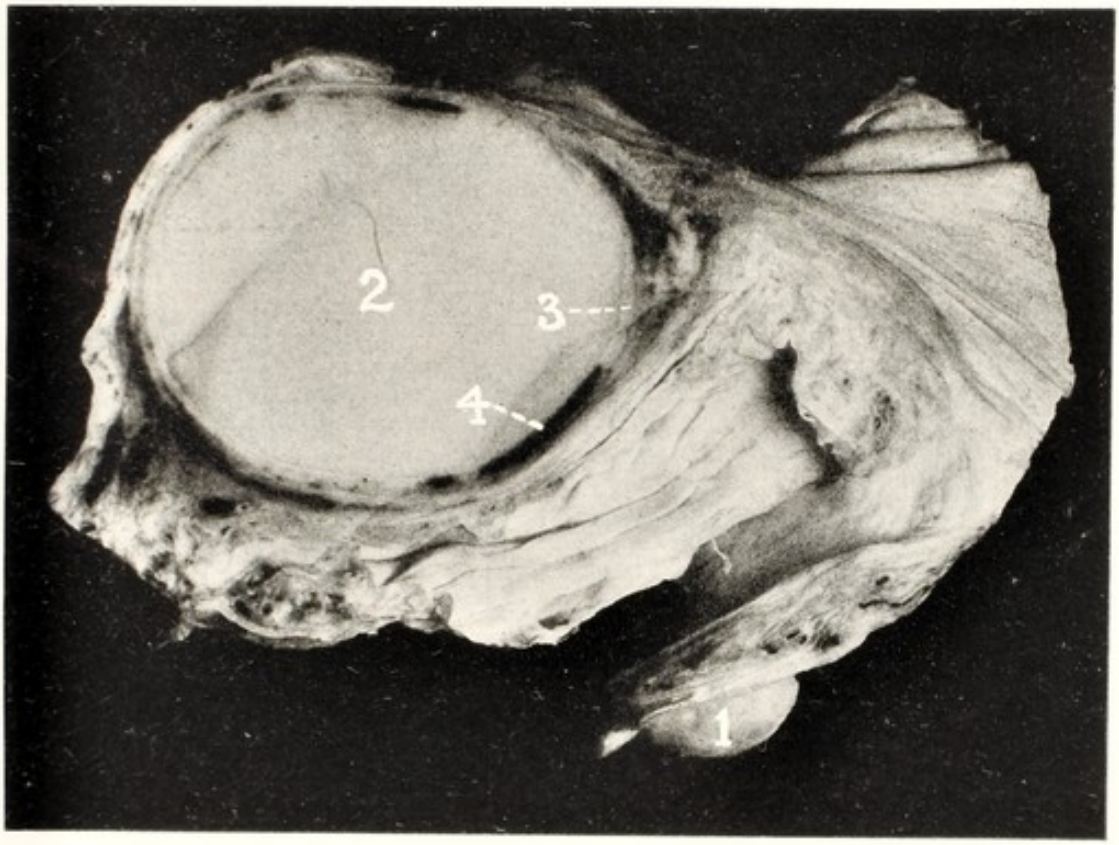


FIG. 149. Cystic Corpus Luteum with Adhesion of the Ovary in the Pavilion.

1, Apex of cornu; 2, cystic fluid (gelatinized by formalin); 3, thin layer of lutein tissue; 4, old blood coagulum.

repair, the disease is generally bilateral. If unilateral the ovary with the oviduct on the affected side may be successfully removed by laparotomy.

Such non-adherent cases as Fig. 147 are best handled, so far as at present known, by the rupture of the cyst and the expression of the collapsed yellow body. If there is clinical evidence of recent infection of the genital tract, any attempt at handling should be deferred for some weeks, until the tubo-uterine disease has subsided. The removal of the yellow body will probably be followed promptly by heat. It would seem prudent not to breed the animal in such artificially induced heat, but to await the recurrence of estrum at about 21 days, in an apparently natural form.

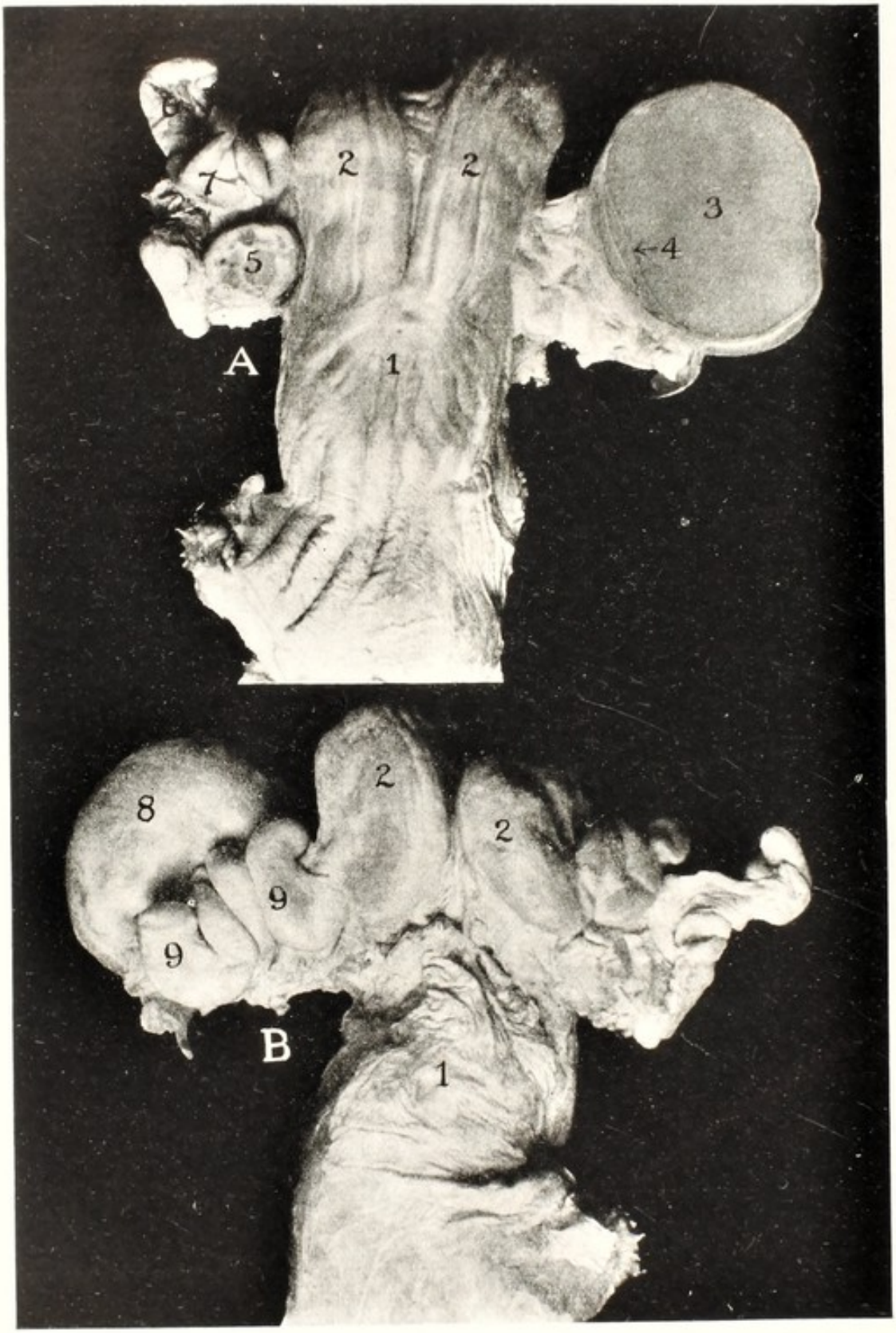


FIG. 150. Cystic Corpus Luteum With Adherent Cystic Oviducts in Cow.

A, Dorsal, and *B*, ventral view. 1, The so-called "body" of the uterus; 2, 2, the cornua; 3, the cystic corpus luteum in section; 4, remnant of lutein tissue; 5, sectioned left ovary; 6, cystic left oviduct; 7, ostium abdominale of oviduct; 8, ventral surface of right ovary; 9, 9, cystic right oviduct.



FIG. 151. Salpingitis (Coital?) in Pregnant Sow.

1, 1, The two uterine cornua, each containing an apparently normal fetus; 2, abscess at the ovarian pole of the cornu, probably embryonic debris; 3, 3, ovaries in section, showing small cysts; 4, cystic distension of the adherent pavilion of the tube; 5, 5, cystic oviducts.

HYPERTROPHY OF THE CORPUS LUTEUM

Hypertrophy of the corpus luteum has been described by some writers as a frequent phenomenon in cattle. It is not clearly defined and has perhaps been confused at times with the enlargement due to cystic degeneration, in which there is a decrease, instead of an increase of lutein tissue. Among more than 3000 cows studied in one series in abattoirs, I found but one definite instance of abnormal volume of lutein tissue. This was a case of corpora nigra in a pregnant cow, Fig. 152.

I have clinically observed abnormally large corpora lutea only rarely. A heifer, which had aborted at about six months, had not been discovered in heat five or six months post-abortum. I palpated and dislodged a corpus luteum fully 2 inches in diameter. Threatened collapse ensued, presumably from ovarian hemorrhage. Fifteen days later I dislodged another corpus luteum of similar volume and again symptoms of hemorrhage followed. She came in estrum, was bred and conceived. The inference seems justifiable that giant corpora lutea follow abnormal hemorrhage into the follicle and that when the gigantic yellow body is removed, the hemorrhagic tendency re-

mains. It is not known how long such corpora lutea persist. It is well not to be hasty in removing them. When it is decided that such a yellow body should be removed, it would appear advisable, according to my experience, to guard against hemorrhage by administering a generous dose of adrenalin prior to the operation.

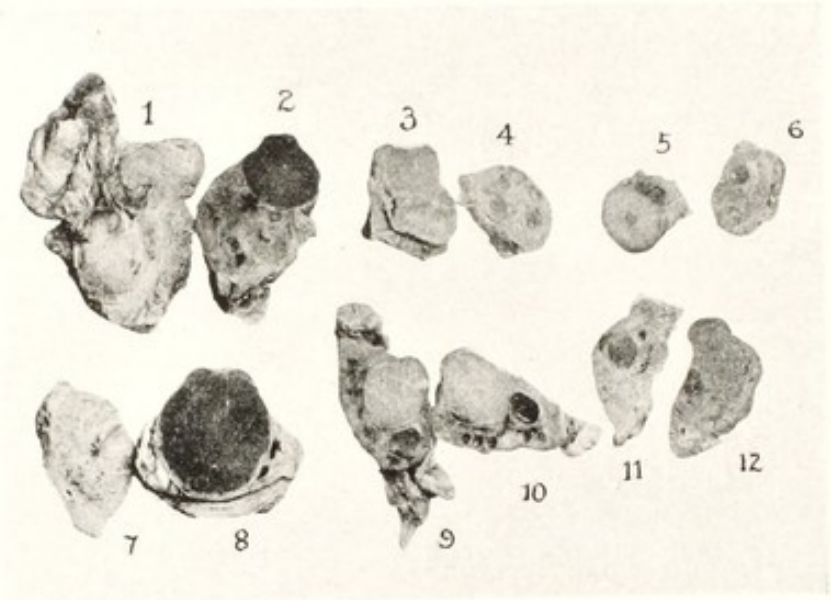


FIG. 152. Corpus Luteum, Variety Nigrum, in Ovary of Pregnant Cow.

1, Adherent ovary; 2, corpus luteum (nigrum) of pregnancy; 3, corpus luteum of pregnancy; 4, mate to 3; 5, 6, corpora lutea of pregnancy; 8, gigantic black corpus of pregnancy; 9, 10, twin corpora lutea of pregnancy; 12, fresh corpus luteum.

CORPORA NIGRA

I have seen two instances of black instead of yellow bodies in the ovaries of pregnant cows. One was of normal size, form, and consistency; the other was greatly enlarged, as shown in Fig. 152. The bodies were a dark blue-black. Aside from the excessive size of one and the color of both, they appeared perfectly healthy. No signs of disease were observed in any part of the genital system or elsewhere. Neither the cause nor the significance of the condition was apparent. It is probably a mere "sport" without relation to sexual health or efficiency.

Diseases of the Oviducts

Tuberculosis of the oviducts has already been mentioned. Various bacteria invade the tubes and cause important lesions. Logically, as in other types of genital disease, those of the oviducts of dairy cows have the greatest economic importance. W. W. Williams[267] reports that, among dairy cows having definite genital lesions, there was present 15.3 per cent of identifiable tubal disease.

Tubal diseases in domestic mammals are not known to be caused solely or chiefly by any one invader. They are commonly classed as belonging to the pyogenic group of bacteria. In contrast, tubal disease in woman is largely ascribed to the one infection of gonorrhea, which plays a highly important role in gynecology, leading frequently to incurable sterility, to tubal abscess and tubal pregnancy, associated with great peril to life. Tubal pregnancy very rarely, if ever, occurs in the cow or other domestic animal and the tubal abscess of a type to imperil life is virtually unknown. The importance of tubal disease in animals is virtually confined to the financial losses due to sterility. Isolated cases are observed in which pregnancy and closed tubes are simultaneously present, as in Fig. 151, suggesting that the tubal infection resulted from the fecundating coitus. More frequently there is an absence of uterine caruncles at the ovarian pole of the cornu, indicating prior placentitis. When the tubal affection is unilateral, and pregnancy occurs upon the healthy side, the infection tends to emerge from the tube, invade the non-gravid cornu and cause necrosis of the non-gravid chorion as in Colored Plate II.

As a rule, tubal diseases are secondary and follow infections of the uterus and cervix. Some cases are observed in heifers which are not known to have been pregnant, especially in that type designated *hydrosalpinx* as illustrated in Fig. 153. The majority of cases appear after retained afterbirth, metritis and cervicitis. When pyometra occurs, especially after retained afterbirth, the virulent pus falls by gravity to the ovarian pole of the diseased cornu, as may readily be understood by a study of Fig. 14. The pus lies against the tubal opening into the cornu. Studies in this field supply much evidence tending to show that the tubes constitute a highly important rendezvous for chronic bacterial infections, from which they emerge into the gravid uterus where the hurriedly-formed fetal membranes and placentae offer a highly favorable ground for multiplication. Thus one finds occasionally, in the uterus of a cow destroyed because of sterility, a minute fertilized ovum with large quantities of pus upon the ovarian side, while upon the cervical side, the uterus appears healthy. This is well shown in later gestation in Fig. 37. Bacteriologists usually do not report *Bacillus abortus* in the diseased tubes.

The lesions are imperfectly divisible into three types: salpingitis, including pavilionitis; hydrosalpinx; and pyosalpinx.

SALPINGITIS. PAVILIONITIS

Inflammation of the oviduct without pronounced distension of the tube with pus or lymph is the most common type of tubal disease. It

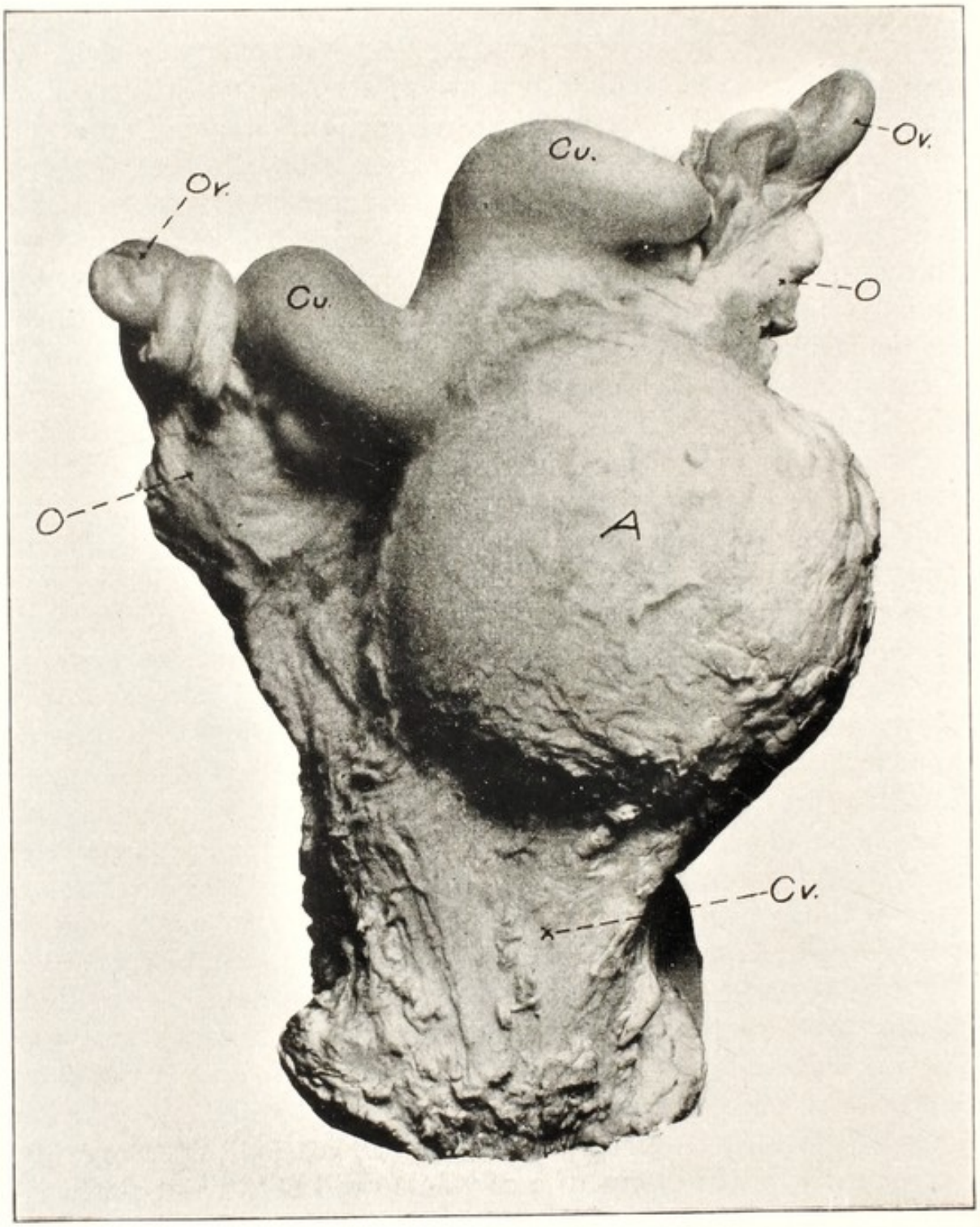


FIG. 153. Bovine Hydrosalpinx and Traumatic Uterine Abscess in a Heifer.

Cv, Cervix; *A*, abscess in the ventral wall of the uterus; *O*, *O*, approximate location of ovaries; *Ov*, *Ov*, cystic oviducts (hydrosalpinx) causing incurable sterility; *Cu*, *Cu*, free portions of uterine cornua.

is without clinical symptoms beyond the inevitable sterility. The disease is largely bilateral and when well advanced, terminates the reproductive life.

Rectal palpation provides almost the sole resource for an accurate diagnosis. The operator traces each cornu in turn to its apex and grasps the oviduct between the thumb and the index and second fingers. In palpating the left oviduct with the right hand the thumb passes down behind the tube into the ovarian pocket, and the tube may be thence followed and accurately palpated until near the ovary. There, owing to the curvature of the tube, it is desirable to reverse the direction and grasp the pavilion upon the lateral side of the ovary and thence follow the tube toward the cornu, until that point is reached where the palpation from the other direction ended.

With moderate experience and care most tubal lesions may thus be detected. Prominent among these are adhesions varying in extent. In many cases there are few or numerous fibrous strands passing from the tube to the mesosalpinx or to the ovary. These are sometimes readily broken down. Often the pavilion is firmly adherent to the ovary, as shown in Fig. 140. Sometimes the ovary is adherent and completely encapsulated within the ovarian pocket. Large cysts may surround the ovary. In numerous instances there is a large cystic corpus luteum within the encapsulated ovary.

The oviducts frequently reveal, upon palpation, enlargements with increased hardness. The enlargement may involve the entire length of the tube or may be somewhat restricted in its area, usually most frequent at or near the distal end. Naturally cases of salpingitis occur which are not palpable. Its presence may then be suspected owing to sterility unaccompanied by identifiable lesions in the ovaries, uterus or cervix. Mild salpingitis is the most difficult of detection among lesions of the female genital organs.

W. W. Williams[268] has used the Rubin plan of insufflation of the uterus with carbon dioxide in order to determine the patency of the tubes when salpingitis is suspected. This can only determine if both tubes are obstructed. If one tube is patent and the other closed the gas escapes into the peritoneal cavity through the open tube.

Salpingitis is incurable in domestic animals. Any efforts at handling by massage, dislodgment of the corpus luteum, uterine douching or other popular remedies are imprudent or dangerous. If one tube is apparently healthy and the other evidently and extensively diseased, it may be well to consider, in potentially valuable cows, the surgical removal of the tube and the corresponding ovary. The operation should preferably be by laparotomy and the apex of the horn amputated with the tube.

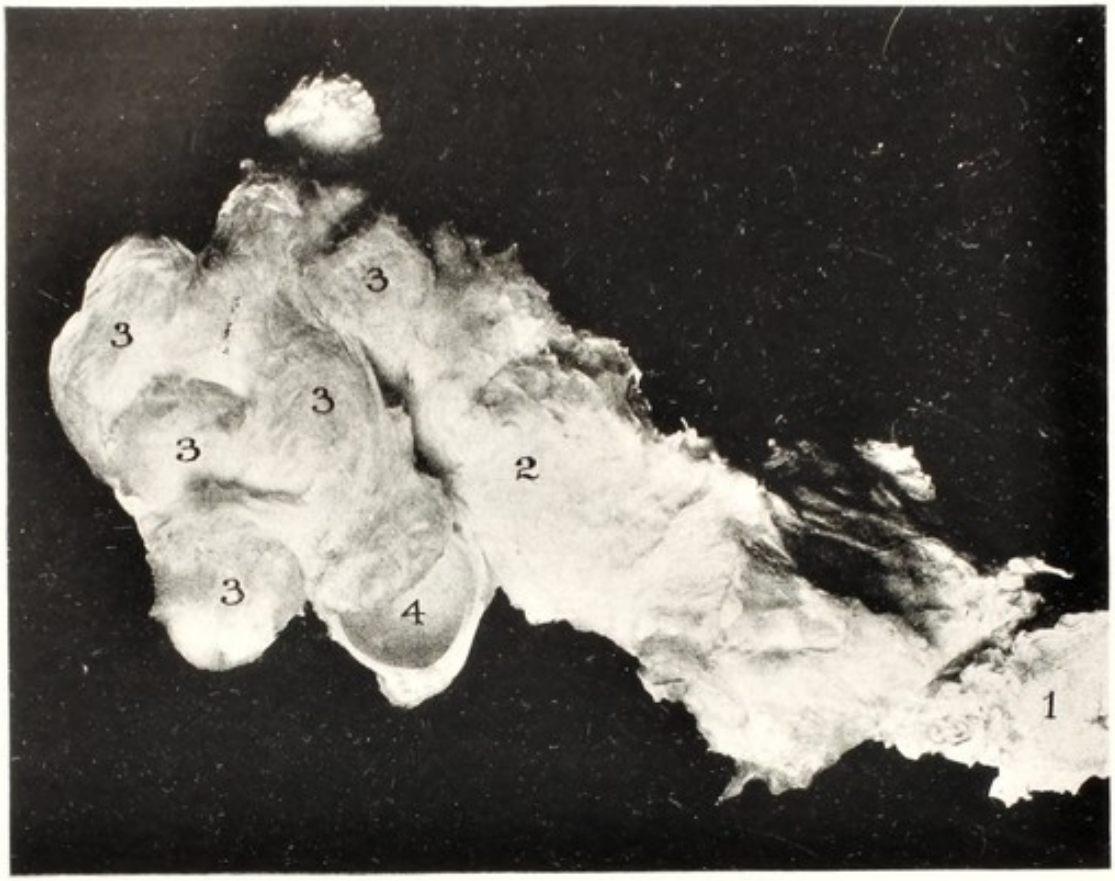


FIG. 154. Complete Pelvic Adhesions of Internal Generative System of Heifer, with Cystic Oviducts and Corpus Luteum.

1, Cervix; 2, cornua; 3, 3, cystic oviducts; 4, left ovary with cystic corpus luteum.

HYDROSALPINX

Superficially it would appear that hydrosalpinx is regularly a consequence of salpingitis. This is strongly suggested by the general coexistence of adhesions of the pavilion of the oviduct to the ovary with, in many cases, cystic degeneration of the corpus luteum in the adherent gland, as shown in Fig. 143. This is not always true. Rarely the ovary is free and the ridges of the mucosa of the pavilion radiate out from the ostium abdominale.

Hydrosalpinx is occasionally observed in heifers not known to have been pregnant. Such cases are illustrated in Figs. 153 and 154. It is not known at what period the tubes became cystic. Evidently the heifers had been bred unsuccessfully until the owner called a veterinarian.

In some cases the pavilion of the tube does not uniformly adhere to the ovary. Instead, only the periphery may adhere and the central cavity, with patent ostium abdominale, become greatly distended as in Fig. 155. This results in a large, fluctuating sphere, with the ovary possibly palpable at some point on the caudal side of the fluctuant sac. Continuous with it is the more slender distended oviduct.

Hydrosalpinx is to be differentiated from ordinary salpingitis by the distension of the tube, its thin walls and its fluid content. It is difficult clinically to differentiate it from pyosalpinx. In the latter, the content is less fluid and the walls are usually thicker. It is not generally highly essential that the two be positively differentiated,

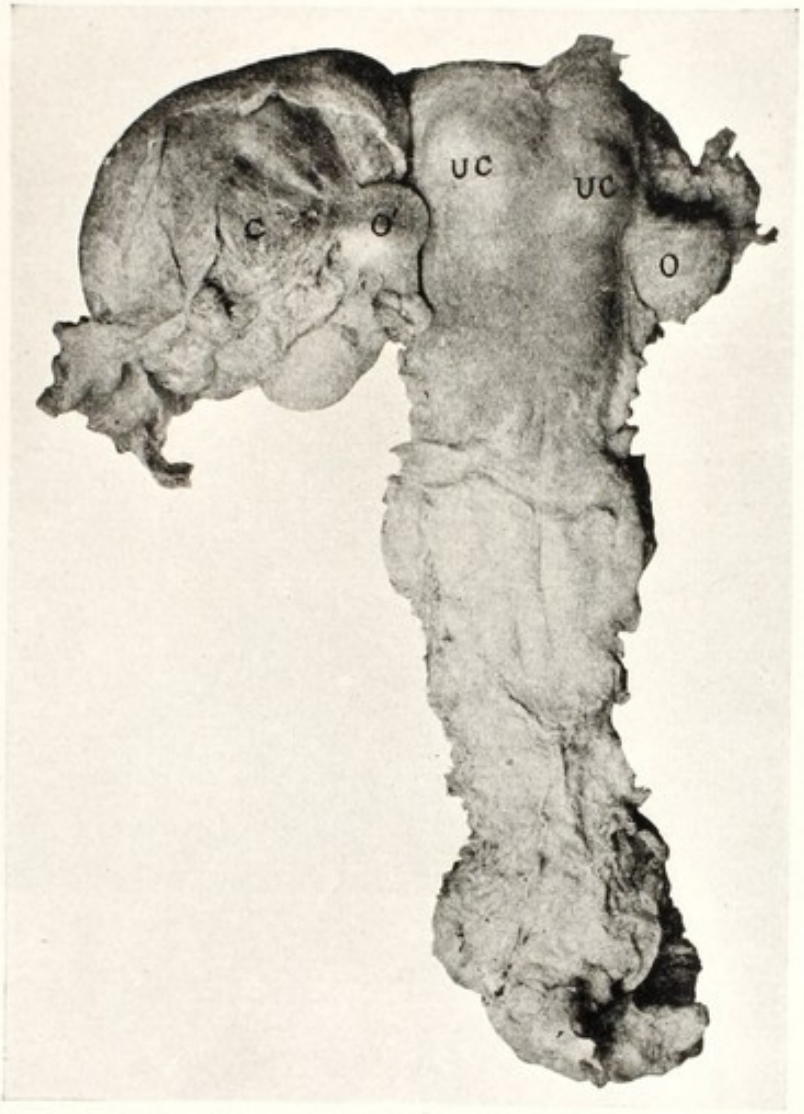


FIG. 155. Cystic Pavilion of the Oviduct, With Hydrosalpinx in Cow.

O, Right ovary; *O'*, left ovary, mostly hidden in adhesions; *UC*, uterine cornua; *C*, cystic pavilion adherent to the ovary.

since each closes permanently the reproductive life of the animal upon the affected side.

PYOSALPINX

Pyosalpinx is not common in any species of domestic animal. When present, it is frequently complicated by involvement of the ovary, to constitute tubo-ovarian abscess as shown in Fig. 156. In other cases the ovaries may not be involved, but extensive disease of the uterus and vagina may be present as in Fig. 161.

The assumption is that pyosalpinx generally arises owing to the migration of infection from the uterus. Most cases probably are the consequence of post-partum metritis. Rarely the infection is coital. At times the infection may accompany fertilization, as indicated in Fig. 151.

Pyosalpinx is not known to compromise the life or general health

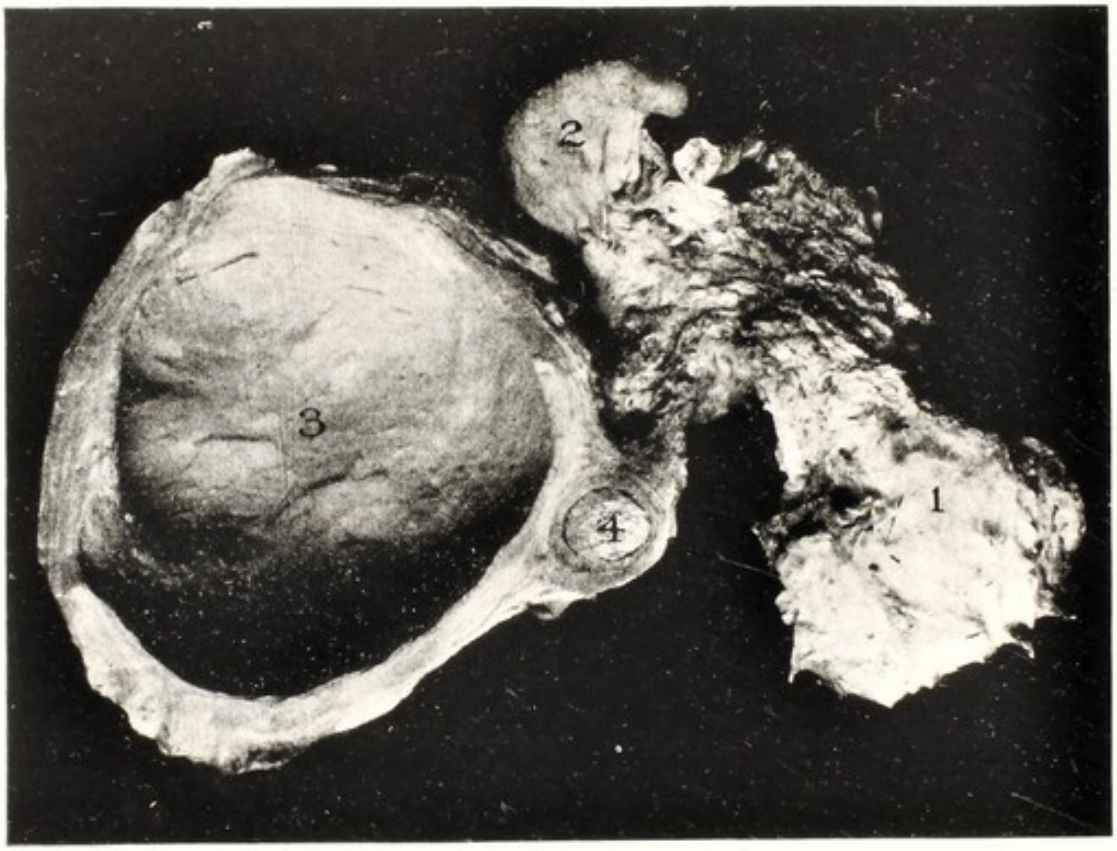


FIG. 156. Tubo-Ovarian Abscess in Cow.

1, Vagina and cervix seen from ventral side; 2, left uterine cornu; 3, tubo-ovarian abscess; 4, cross section of the oviduct filled with pus.

of the patient. It is an irremediable barrier to pregnancy upon the involved side. It is frequently bilateral. When unilateral, and pregnancy occurs upon the healthy side, the infection from the diseased oviduct may enter the non-pregnant cornu and cause progressive necrosis of the fetal membranes, as indicated in Colored Plate II. Surgical interference is justified in prudently selected cases of unilateral disease in unusually valuable cows. Such cases call for the removal of the ovary, oviduct and a liberal portion of the cornu. Laparotomy is to be preferred to vaginal incision.

Chapter XXVIII

THE CHRONIC DISEASES OF THE UTERUS, CERVIX AND VAGINA OF THE NON-PREGNANT FEMALE WHICH MAY INTERFERE WITH REPRODUCTION

Chronic Metritis

PATHOGENIC organisms presumably may invade the organs of reproduction at any period in the life of the female subsequent to the formation of the ducts of Mueller. It is known that bacteria invade the alimentary tract during fetal life and cause diarrhea. In order to do this the bacteria must invade the fetal blood and thus may reach the reproductive system. The fetal genitalia are without known function and are not known to suffer importantly from bacterial invasion.

Soon after birth bacteria invade the system of the young. It has not been determined to what extent the infection becomes domiciled in the genital system. *Bacillus abortus*, which has been most studied among organisms interfering with reproduction, enters the fetal blood. Soon after birth, it may enter per oram in infected milk and become domiciled permanently within the body, but it is not known to acquire a permanent habitat in the reproductive system. In what organs it persists has not been determined. I have reported[236] observations upon a group of heifer calves which largely became positive to the agglutination test for *Bacillus abortus* when a few days old and many of them continued positive up to the date of abortion in first pregnancy. Presumably the *Bacillus abortus* was present in the uterine contents at the date of abortion; but, according to the teachings of some bacteriologists, it was probably not present in the uterus until after pregnancy had become established.

Somewhat similarly the large group of heifers in Table X, affected with genital hypoplasia, arrived at breeding age in a state of low fertility. Apparently this was in part referable to ovarian hypoplasia. The uterus participated in the hypoplasia, the estrual cycle was erratic and abortion, abbreviated gestation, retained afterbirth and other abnormalities prevailed. Whether some disease of the uterus existed when the heifer was first bred, or whether the uterine hypoplasia rendered the organ more vulnerable to invasion from other organs or from sources outside the body, the final result was the same.



FIG. 157. Pyometra in Bitch.

A normal uterus and ovaries have been placed on the diseased organ as check.
A, Diseased uterus; B, healthy uterus.

As a rule, the virgin female genital tract is not critically studied in dairy heifers or in other females designed primarily for reproduction. Instead they are first bred, and if they fail to become visibly pregnant after a series of matings, it is sought to discover the barrier to conception. It is then too late to readily determine whether any of the lesions recognized existed prior to the first coitus or were the result of coital infection. Occasionally metritis or other genital disease ascribed to infection is observed in adult mares and bitches which are not known to have been bred. The observed cases are usually those of pyometra. Thus in Fig. 157 is shown an enormously distended uterus of a bitch not known to have bred. The relative enlargement when compared with the normal organ introduced for comparison is well nigh unbelievable. An adult, unbred mare in our clinic had extensive pyometra with black pus, which the bacteriologist ascribed to a mold. In some cases of imperfect uterus unicornis, one cornu is atretic at its base and open at its ovarian pole. This latter becomes greatly distended by the accumulation of endometrial excretions. The cow may breed on the normal side for some time and it is later found that the excretions in the atretic horn have become infected, to constitute a large, atonic abscess. In such case the suppuration can not be ascribed to either coitus or pregnancy. Thus there is much evidence that the virgin uterus may harbor infection which under favorable environment may cause important lesions.

There is much evidence that coitus causes disease of various portions of the genital tube. Although the dairy heifer regularly has the nodules of *infectious vaginitis* from early calfhood, coitus sets it aflame, as already noted in Chapter XXV. Its significance is in controversy. Dourine of horses is undeniably transmitted by coitus. The same is true of the trichomonad disease of cattle. The trichomonad disease becomes spectacularly more virulent if the infecting coitus produces pregnancy; the ovum supplies a favorable soil in which the protozoon may multiply. Such is the rule for most, if not all invaders having a special affinity for the genital tract. In Table II it is evident that coitus by Bull 2 resulted in destructive, chronic intrauterine infection. There is room for controversy regarding the details of the bacterial invasion. Some may hold that the infection which caused abortion, abbreviated gestation, and metritis was directly coital; others may contend that the spermatozoa were defective and caused a weak fertilization which invited invasion by pathogenic forces not contained in the semen. Clinically the controversy is nonsignificant, the end result being the same in either instance, and the disease demands the same measures for prevention. In numerous

herds many cows bred to a given sire show severe cervicitis and salpingitis, under such conditions that it is unavoidably ascribed to coitus.

The evidence points definitely to coital infection as highly significant. Its importance has been repudiated by some, because they have failed to trace *Bacillus abortus* to coital infection. While unquestionably many cases of metritis and cervicitis have their origin in coitus, the evidence is historical rather than clinical. In a large ratio of utero-cervical infections, definite lesions are largely excluded until pregnancy becomes established, and the newly-formed maternal placentae and the ovum provide favorable soil for multiplication. The diseases of the ovum and of the pregnant uterus do not generally reveal their presence until pregnancy is terminated by abortion or birth, at which period they reveal themselves in a multitude of forms. A complex group of phenomena appear, such as parturient paresis (milk fever), parturient eclampsia in various species, and numerous other illy defined types of disease which suddenly appear just prior to, during, or early after the expulsion of the fetus. They are variously ascribed to toxaemia of pregnancy and to deficiencies of glycogen, calcium, etc., in the blood. Or the existing genital diseases reveal themselves in dystocia. These two groups of phenomena have already been described in the companion volume, *Veterinary Obstetrics*.

Following the termination of pregnancy, the lesions of gestation in both the mother and young may be learned from the fetal membranes. These constitute an authentic replica of the endometrium. The fetus may supply important information of what has occurred during intra-uterine life.

Pregnancy constitutes the supreme burden of reproduction. The rapidly developing fetus and its membranes, the sudden immense expansion of the uterus, the demands of fetal nutrition upon the pregnant female, and the excretion of the waste products of fetal development, invite pathogenic invaders which may multiply rapidly and perilously. Finally, when parturition occurs, except the general and genital health of the female is ideal, the exhausted reproductive system is further depressed by labor. Any infection which has existed during gestation has an opportunity to acquire great virulence. Invasion from the exterior is invited. The genital tube as a whole is highly vulnerable to disease. Hence parturition and the puerperal period supply the highest mortality rate in the breeding female. The diseases of pregnancy and of the puerperium will be considered later.

The female which survives parturition and the puerperal diseases immediately following, is frequently left with persistent chronic dis-

ease, lesion or defect which may inhibit, or render difficult future conception. Such diseases and defects, combined with those of the oviducts and ovaries, already considered, constitute the general basis for the phenomena which are collectively designated as sterility.

It is not easy to measure the relative significance of ovarian, tubal and utero-cervical diseases as the primary causes of sterility. Early in the history of the war against bovine sterility, the two most prominent leaders in the field held radically conflicting opinions. Hess[89] firmly believed that *infectious vaginitis* was the chief offender and that it caused ovarian, as well as uterine disease; Albrechtsen[4] was equally certain that *Bacillus abortus,* by causing metritis, was the basic cause of sterility. The ovarian diseases, Albrechtsen held, were secondary to the metritis and promptly disappeared after the uterine disease was brought under control. This, he claimed, he succeeded in accomplishing in 90 per cent of the cows. At present there is scant room for doubt that the diseases of the various reproductive organs are intimately linked together: if the ovaries are seriously diseased, the uterus loses its physiological function; if the uterus is extensively diseased (mummified fetus, trichomonad pyometra) the functions of the ovary are suspended, and the corpus luteum becomes pathologically embedded and persistent. A diagnosis of cystic degeneration of the ovaries or of salpingitis, however accurate it may be, does not exclude metritis or cervicitis. The reproductive system largely behaves as a unit, and serious disturbance of any one of the organs tends to cause interference with the proper functions of all the others.

When attempting to salvage the reproductive power of a wrecked cow by handling the uterus it becomes essential that due heed be given to the status of other organs. Thus an attempt was made to retrieve the breeding power of the heifer whose genital tract is shown in Fig. 153, by douching the uterus. It was possibly diseased, but the hydrosalpinx present rendered all efforts at treatment useless and imprudent.

Clinical experience teaches that various infections may persist in the uterus indefinitely and serve to prevent conception, or when pregnancy occurs, make it unsafe and imperil the health of both the mother and young. Many cases of metritis properly handled may be caused to recover to such degree that the animals may prove financially satisfactory as breeders or as milk cows. The recovery of 90 per cent of such cows, as reported by Albrechtsen, is not in harmony with general observation in most lands. The wisdom of undertaking to salvage the breeding life of a female, sterile because of chronic metritis, is dependent upon the accuracy of diagnosis con-

cerning the extent and significance of the lesions present, the potential value of the animal as measured by pedigree and individuality, the prospective expense involved and the probabilities of eventual restoration of breeding power.

The accurate diagnosis of chronic metritis demands an authentic history of the animal, and a detailed clinical study, involving in mares

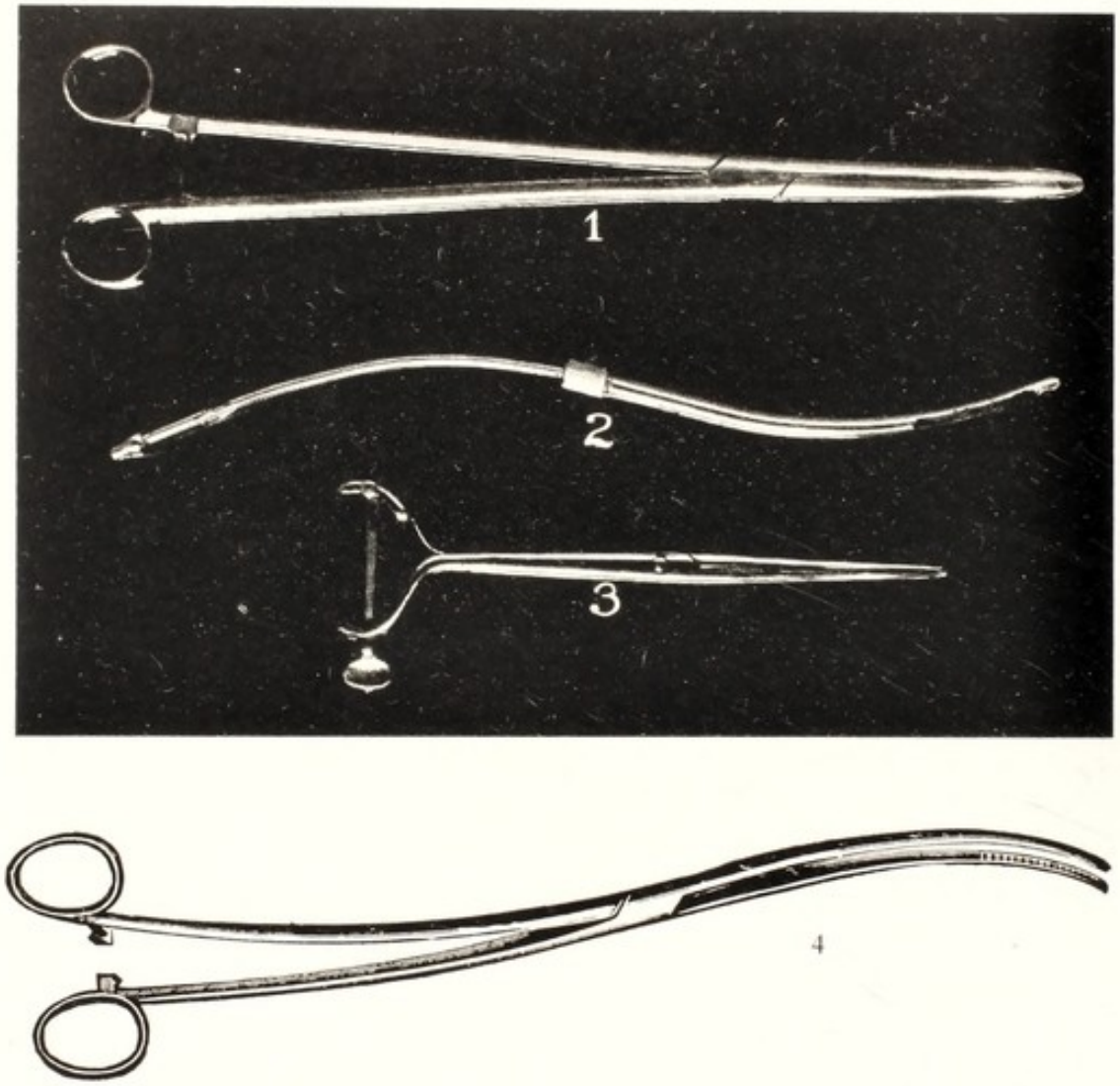


FIG. 158. Instruments for Douching the Uterus of the Cow.

1, Albrechtsen's uterine forceps; 2, Albrechtsen's small uterine catheter; 3, Palmer's cervical dilator; 4, Bozeman's uterine dressing forceps.

and cows, vaginal examination, palpation of the reproductive system per rectum, and exploratory douching of the uterus. Unfortunately an accurate breeding history is rarely available. If a cow has suffered from retained afterbirth, she may have lost, by necrosis, all caruncles from one cornu, rendering her virtually sterile on that side; or she may have lost all caruncles from both cornua, in which case there is possibly a chance of 1:100 that she may again produce a live calf.

These damages are not usually fully recognized during the period of puerperal disease. When the time comes for breeding, the presence or absence of the caruncles cannot be authentically determined by clinical examination. Thus where history fails, clinical examination may be rendered futile.

If, for example, a cow has a purulent genital discharge following the termination of pregnancy, and there is repeatedly observed a mass of pus lying in the gutter behind her, the phenomenon is not recorded. It occurs so frequently that it is quickly forgotten and the cow ap-

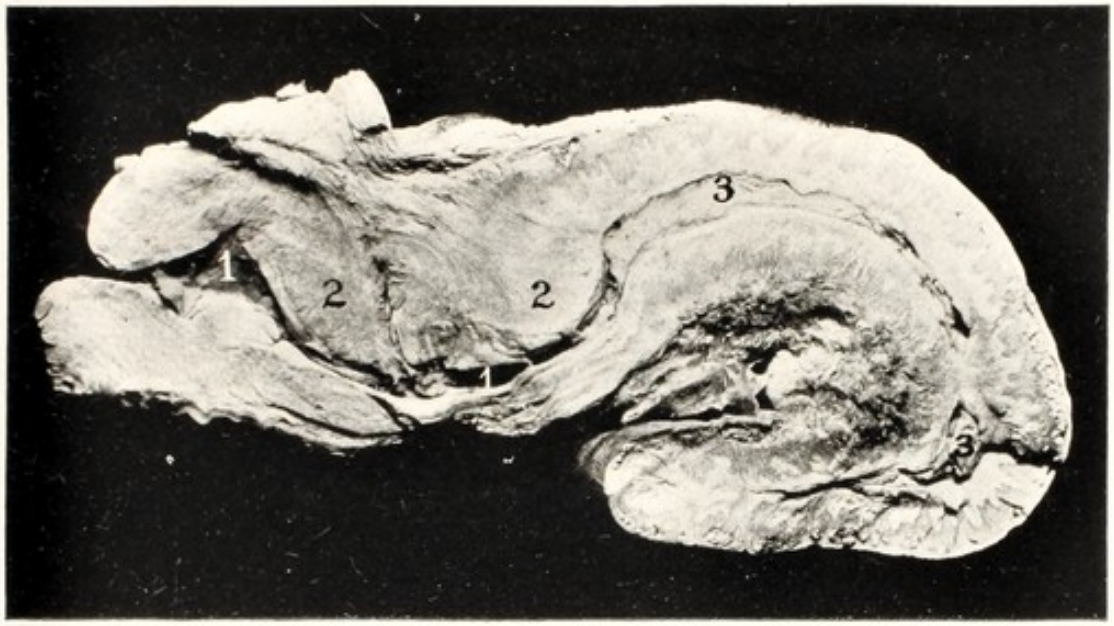


FIG. 159. Sclerosis of Cervix and Uterus.

1, 1, Cervical canal; 2, 2, sclerotic enlargement of annular folds of cervix, displacing the cervical canal ventralwards; 3, 3, cornual cavity filled with pus. *The placental tissues have been destroyed and the uterine cavity consists of a purulent fistula.*

pears well. If a careful examination of the afterbirth was made at the close of the last pregnancy and accurately recorded, the facts are invaluable in arriving at a reliable diagnosis and prognosis, should difficulty in conception arise.

The vagina may contain pus, which usually emanates from the uterine cavity or cervical canal. The vagina of aged cows with pendulous abdomen and atonic reproductive system sometimes contains several ounces of urine which have gravitated to the fundus of the organ. An inspection of the vaginal mucosa reveals the extent of *infectious vaginitis* present. Chronic cervicitis at its vaginal end is visually recognizable with the aid of the vaginal speculum. It is also identifiable by digital palpation. In most cows, two pairs of uterine forceps (1, Fig. 158) may be applied to the cervical lips and the

cervix drawn back into the vulva, where it may be carefully inspected. Such gross disease as is shown in Colored Plate III is readily recognizable by sight or touch. It is to be borne in mind that the cervix usually changes with the age, the number of pregnancies experienced, and the health of the cow. In the cervix of the healthy heifer, 1 and 2,

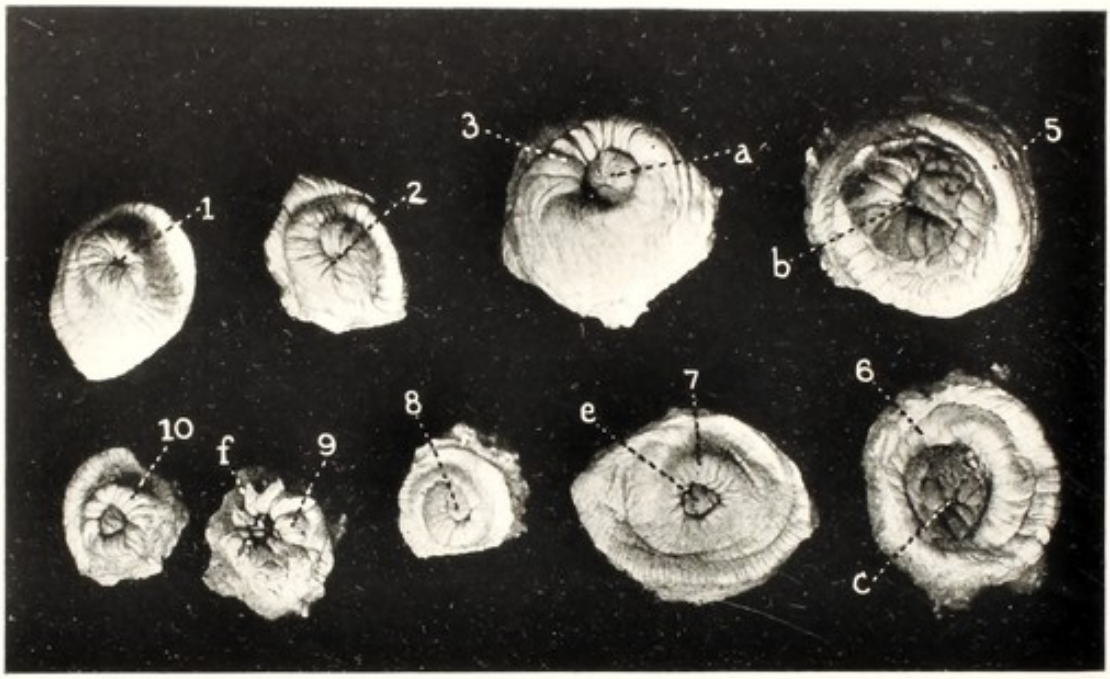


FIG. 160. Vaginal Ends of Bovine Cervices.

The numerals indicate the lips of the original os uteri externum. In 1, 2, and 8 the second annular fold does not protrude, as is typical in healthy heifers which have not calved. After having calved, the second annular ring of the cervix (compare Fig. 161) tends to protrude beyond the original cervical lips as indicated by letters.

Fig. 160, the margins of the encircling vaginal mucosa are in contact and the circular folds of the cervix (Fig. 15) are completely hidden. When the first pregnancy has terminated and the cervical canal has been sufficiently dilated to permit the passage of the fetus, its mucosa may be more or less injured and infection of varying degrees ensue. As a consequence the first or caudal circular fold of the cervix may protrude as indicated at 5 and 6 of Fig. 160. The abnormality grows with the number of parturitions and virulence of infection until it reaches and exceeds that illustrated in Colored Plate III. The vaginal end of the cervix becomes a great, cauliflower-like tumor. It is intensely injected, and in some cases bleeds readily. The surface consists of *cervical,* not vaginal mucosa. It may excrete pus in considerable quantity, or there may form on its surface a thick layer of highly viscid exudate—a pathological uterine seal, in a sterile animal.

By rectal palpation, enlargements or other abnormalities of the cervix, like that in Fig. 159, may be identified. Any adhesions of the uterus to other pelvic organs or structures are usually palpable without difficulty. Their presence clearly indicates the prior occurrence of severe metritis. There may be present definite thickening and sclerosis of the uterine walls as in Fig. 159. The uterus may be of approximately the same size and similar in the degree of hardness to that of estrum. If a corpus luteum is present, it is imbedded. The presence of a ripe ovisac is virtually excluded. The cow does not usually exhibit signs of estrum. The vagina contains pus, not estrual

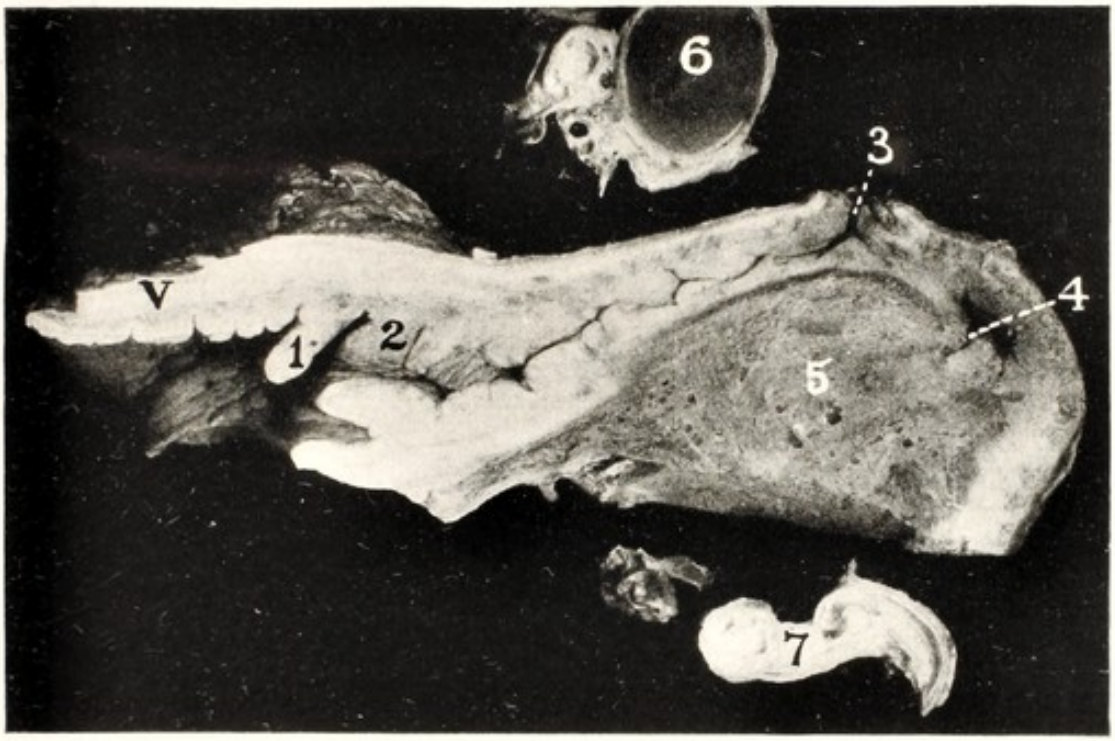


FIG. 161. Rupture of Uterus With Catheter (See also Fig. 146).

V, Vagina; 1, lip of os uteri externum; 2, first annular fold of cervix; 3, 4, ruptures of uterine walls with catheter; 5, enormously thickened broad ligament of uterus; 6, cystic corpus luteum with cystic oviduct adherent to ovary.

mucus. The uterus is frequently extremely atonic and flaccid. The variations in the character and extent of adhesions to surrounding parts, the thickness and sclerosis of the uterus and cervix are illimitable.

Highly important evidence regarding chronic metritis and cervicitis is available by exploratory uterine douching. This may be accomplished in various ways. The Albrechtsen method usually answers the purpose well. The uterine forceps, 1, Fig. 158, are applied to the lips of the cervix, or to the vaginal wall at either side of that organ. Traction is then applied and the vaginal end drawn back near to, or

into the vulva. Usually the cervix of the dairy cow, in regions where they are largely stabled, may be readily drawn back into the vulva. The genital organs of dairy cows kept constantly out of doors, as in the tropics, and of beef cows are more firmly attached and cannot be drawn so far backward. By drawing the two pairs of uterine forceps apart the cervix is brought clearly into view. Illumination of the parts with a flash light is at times advisable or necessary. The cervical canal of the virgin heifer (1, 2, Fig. 160) is extremely narrow. During estrum the canal is dilated and filled with lubricant mucus.

The cervical canal may be examined by means of the dilator, 3, or catheter, 2, Fig. 158, or other suitable instrument. As may be seen by a study of Fig. 15, the free margins of the annular folds of the cervical mucosa are directed toward the vagina and obliquely inward toward the central axis of the canal. The sound or catheter may lodge behind one of these folds and any force will tend to wound the cervix or to push the mucous fold into the lumen of the canal. Patience needs be exercised and an attempt made to follow the tortuous canal by alternately advancing and retreating, and varying the angle at which it is sought to advance the instrument. When difficulty arises, the method of operation may be modified and, instead of retracting the cervix, it may be grasped per rectum with one hand, and the instrument, held in the other, guided into the os uteri by the hand within the rectum. The hand may then encircle the entire cervix and at all times palpate the instrument. The difficulty remaining unconquered, the operator may await estrum, when the cervical canal becomes dilated and the cervical walls relaxed. Should convenience so dictate, the corpus luteum may be dislodged and the douching performed during the subsequent heat.

The examiner should eliminate from his thought the ancient fable of atresia of the cervical canal. True enough, atresia still occurs, but in those rare cases the uterus promptly becomes distended with menstrual debris, pus or other liquid. Very rarely, congenital defects may cause retention of uterine secretions as in Fig. 8, but in such cases the cervical canal is patent and the fluid may be mechanically drawn off, only to promptly refill. Atresia of the canal, as popularly taught a few decades ago, is now unknown. When chronic cervical disease is present, as in Fig. 159, the annular folds may become so enlarged and sclerotic at one or more points, that the canal is so bent that the passage of an instrument through it becomes difficult and dangerous. Any imprudent force employed may rupture the walls and lead to an abscess as shown in Fig. 153. Or the catheter or other instrument

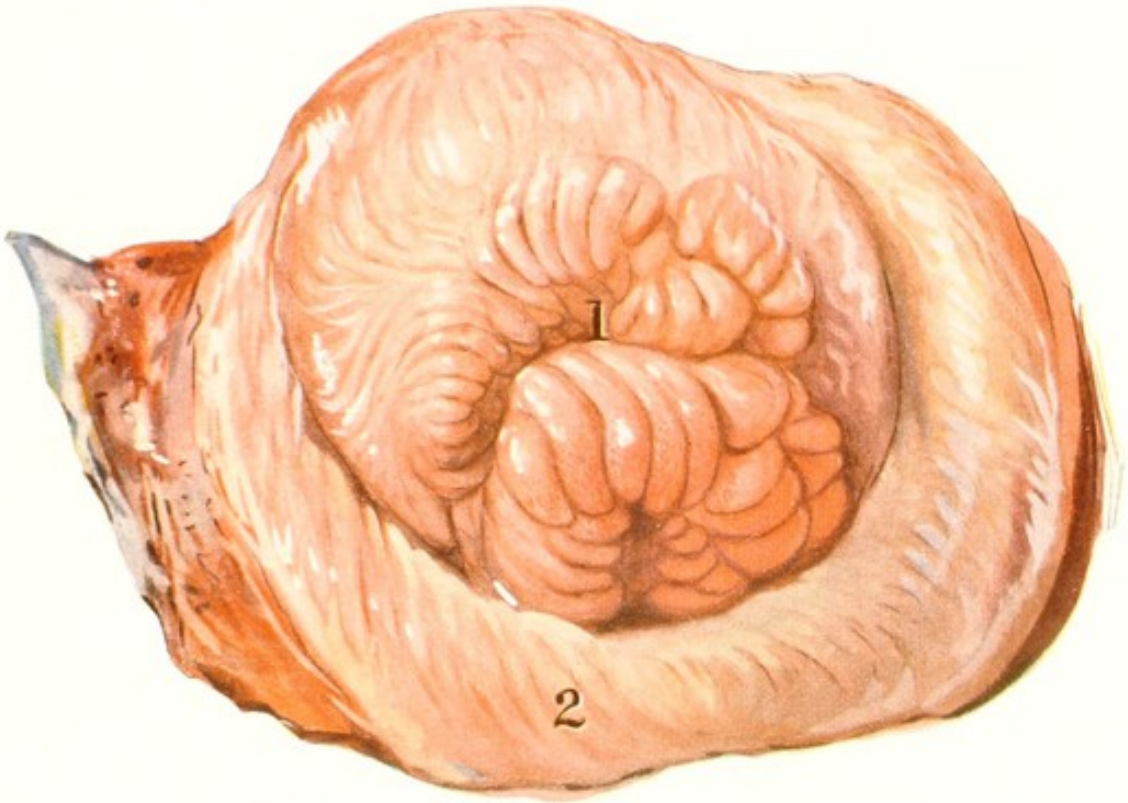


PLATE III

CERVICITIS. COW

1. Greatly swollen, inflamed and prolapsed first annular, mucous fold. 2. Lip of os uteri externum pushed aside by the protruding, first mucous fold.

may penetrate the peritoneal cavity, causing fatal peritonitis, as in Fig. 161. In each case the attempt to douche the uterus was wholly unjustified, the basic and irremediable lesion being in the oviducts and diagnosable by careful rectal palpation.

The cervical canal having been sufficiently dilated, the uterine catheter, 2, Fig. 158, is introduced, and the uterus douched with warm physiological salt solution. If any important degree of metritis or cervicitis exists, pus will be present in the return flow. If the amount of pus be very small, it may have come from the cervical canal. Sometimes the amount of pus clearly indicates extensive endometrial disease. Thus in Fig. 159 there is an important volume of pus in the uterus, the endometrium has been totally destroyed, the cornual cavity has become a purulent fistula and the breeding life of the cow is definitely closed. The history of the case, the return of pus in the douche, the absence of purulent distension of the uterus, and the presence of sclerosis of the walls, indicate with reasonable assurance, the incurability of the disease. In some such cases, if not in all, there is a total absence of estrum. Sometimes when a sterile cow is repeatedly in estrum at irregular, pathological intervals, the douche may reveal one or several cc. of pus and very rarely, a tiny ovum is caught in the uterine catheter: the cow has repeatedly conceived, the very small ovum has died, and a small amount of pus, varying in volume from time to time, is present in the uterine cavity.

Cervicitis and metritis are intimately related and almost uniformly co-exist. If metritis is present, the infection almost inevitably moves caudally into the cervix; if the cervix is importantly diseased, the uterus almost always participates. There are rare exceptions. I once found the caudal half of the cervical canal open and suppurating freely. Without further search I passed a uterine catheter through an adhesive uterine seal at the cephalic end of the canal and aborted the cow.

Having satisfactorily determined that chronic metritis or cervicitis is present, that the ovaries and oviducts are healthy, and that no irremovable obstacle to fertility is present, the handling of the disease may be considered. Usually if the female is of moderate potential value, and the disease is not markedly extensive, the prognosis is good.

Some cases of cervicitis are so extensive and virulent, like that shown in Colored Plate III, that remedial efforts are imprudent, since they must end in failure. Occasionally the cervix is enormously enlarged, 3 or 4 inches in transverse diameter, and elongated to 5 or 6 inches, the mucous folds protruding into the vagina, covered, in some

instances, by a heavy layer of tough, adhesive exudate, and the cervical canal filled with the same substance. It is a pathological uterine seal of enormous proportions, in a non-pregnant cow. So far as I have seen there is no justification for attempting to cure such cases. In most animals the endometrium is also hopelessly diseased.

The treatment for chronic endometritis and cervicitis has made scant progress since the days of Albrechtsen and Hess. The repeated douching of the uterus with 2 or 3 per cent of Lugol's solution of iodine (iodine 5 parts, pot. iodide 10 parts, water 85 parts) as recommended by Albrechtsen is probably as beneficial as any other treatment. Possibly in some cases the introduction into the uterus of bismuth subnitrate, iodoform and mineral oil may be preferable, because the bismuth and iodoform dissolve tardily and remain in the uterus for some days, whereas the Lugol's solution and other soluble disinfectants are promptly expelled or absorbed. Some practitioners favor the use of stronger Lugol's solution, even up to full strength.

The prevention of cervicitis is largely available by eliminating as far as practicable the known causes. Heifers and other females, developed in a healthy uterus and prudently handled during early life, arrive at breeding age with healthy cervices. If mated with healthy sires, they do not become venereally infected, and if the ovum and uterus are healthy, there is no notable injury to the organ during parturition. Careless handling of the cervix, such as the introduction into the canal of dirty instruments or fingers, invites infection and the danger is greatly intensified if abrasions or wounds are caused by rough handling. Once infection has become established and lesions caused, the condition is intensified at each subsequent termination of pregnancy, as the dilating of the canal sufficiently to permit the passage of the fetus, causes traumatic injuries of varying degrees. Parturient lesions of the cervix are further exposed to irritant uterine discharges passing over them during the puerperium. Yet more perilous is the contact of putrefying retained membranes with the diseased tissues.

The cervix is regarded as an important barrier against bacterial invasion of the uterus from the vagina. Yet it frequently becomes diseased and acts as an infectious focus, especially in the cow, where the cervix is most elaborate.

In the handling of the most common type of cervicitis in the cow, it is advisable usually to resort to powerful antiseptics. The diseased mucosa may be treated by the application of pure Lugol's solution or of 10 per cent silver nitrate. This is to be accomplished by means

of a pledget of absorbent cotton saturated with the disinfectant and introduced through the canal with the long dressing forceps (4, Fig. 158) or other suitable instrument.

If the recovery from the cervicitis be incomplete, and be interrupted at that stage where conception becomes possible, the pregnancy may be very insecure. If the infection persists after conception, the cervical infection constantly threatens the invasion of the uterine cavity and the ovum. An infected cervix constitutes an important starting point for infections which induce abortion. Less frequently, especially in heifers, the cervicitis causes extreme sclerosis and interferes with the expulsion of the fetus, necessitating surgical dilation of the cervical canal, or the application of severe traction. In one heifer where I thought traction justifiable, the sclerotic cervix ruptured longitudinally, along the mid-dorsal line.

Cervicitis is most frequent and severe in highly bred dairy cows. There sometimes occur deep longitudinal fissures in the cervical walls, during the expulsion of the fetus. Unless promptly handled, these do not properly heal but leave behind a deforming cicatrix or a sclerotic enlargement in the cervical wall similar to that shown in Fig. 159. In order to overcome these, various operations have been advocated.

Logically they can only be successful when the cervicitis or cervical deformity is the sole existing obstacle to reproduction. Generally, the cow must be of exceptional potential value in order to justify the more complex operations, and the outlook needs be fair that the operation will not only render the cow capable of conception but also provide security for any pregnancy attained.

Chronic Vaginitis and Vulvitis

The chronic diseases of the vagina and vulva interfering with conception are not numerous if the *contagious vaginitis* of cows already described be omitted. The most frequent obstacles encountered consist of lesions due to parturient injuries or to the use of highly irritant vaginal injections. Heifers suffering from genital hypoplasia, like those mentioned in the herd represented in Table X, inevitably suffer from vaginal abrasions during their first parturition. Following calving or abortion, retained afterbirth and metritis prevail and the vaginal mucosa is constantly irritated from decaying membranes and purulent secretions. This may result in lesions which interfere with conception. I observed a mare which had apparently suffered severe vaginal injury at the close of a prior pregnancy. She was bred and

immediately began to strain violently, everting her vagina. The straining was controlled by complete chloral anaesthesia. The obstacle to breeding was insurmountable.

I studied several purebred Hereford cows which had aborted. As a remedy, an attempt had been made by laymen to follow an official bulletin recommending vaginal injections of Lugol's solution. Apparently because the solution was too strong, or owing to some other error, severe vaginitis ensued, followed by complete atresia of the vagina—which barred the escape of utero-cervical excretions. The anterior portion of the vagina, the cervical canal and the uterine cornua became enormously distended with transparent, thin lymph or mucus, the volume reaching one gallon or more. Treatment was not attempted.

A client discovered a mare in dystocia, the fetus in anterior presentation with the posterior limbs projecting beneath the abdomen and chest. The fetal chest had advanced to the vulva. Labor had come on during the night and the dystocia had probably existed for ten hours. Embryotomy was performed. The vulva underwent extensive necrosis with severe constriction so that coitus was thereafter barred. As a rule there is no remedy for chronic lesions and constrictions of the vagina and the vulva. Most of them may be prevented by timely precautions.

Chapter XXIX

DISPLACEMENTS OF THE GRAVID UTERUS AND VAGINA

HUMAN obstetrists describe displacements of the gravid uterus of woman known as anteflexion and retroflexion, and anteversion and retroversion. Some veterinary obstetrists describe under the same names conditions in pregnant domestic animals which are difficult of identification with the displacements in woman.

It needs be borne in mind, when attempting to compare the uterine displacements in woman and domestic animals, that the posture of the former differs radically from that of the latter. When a woman walks, stands, or sits, the gravid uterus rests within the pelvis as in a basin (as the anatomical name implies), and the pelvis supports the weight. There is no tension upon the cervix and the vagina, but these parts are compressed instead. These conditions are reversed in quadrupeds, in which the pregnant uterus moves by gravity toward the diaphragm and the weight of the organ is borne by the diaphragm and the ventral wall of the abdomen, and in part suspended through the vagina and vulva, from the perineum. The suspension from the perineum is greatly exaggerated in the cow, ewe, and goat when arising from the recumbent position. When arising from sternal decubitis, the cow first extends her posterior limbs, while the sternum still rests upon the floor. This places her body at an angle of about 45 degrees, so that the pregnant uterus, except as supported by the diaphragm, is virtually suspended from the vagina and vulva, with such aid as the broad ligaments of the uterus contribute. There is little change in relations in decubitis. This is chiefly sternal. Rarely the general intra-abdominal pressure favors vaginal prolapse in the recumbent female in either sternal or lateral decubitis. Dorsal decubitis, so usual in woman, is incompatible with life especially in solipeds and ruminants. Hence the cervix and vagina of a pregnant woman are usually in a state of compression from the superposed gravid uterus and except in accident are never in a state of tension: the cervix and vagina of the cow are constantly in a state of tension, with the possible exception of very rare accident. In advanced pregnancy the length of the bovine fetus may exceed the distance from the pelvic border to the diaphragm, and the head and anterior feet extend into the pelvic cavity as indicated in Colored Plate I, to rest above the cervix and cephalic end of the vagina. This

exerts no compression upon the vagina. The weight of the gravid uterus (150 to 300 lbs.) is still borne by the diaphragm and linea alba. The ventral walls of the uterus, cervix, and vagina remain tense. The phenomenon superficially resembles the retroversion of woman, but it is purely physiological. It is merely an increase of the slight extension of the uterus above the cervix, notably present at the fortieth to sixtieth day of pregnancy, which serves as a mark of differentiation between pregnancy and pyometra. Hence the displacements of the pregnant uterus of woman are the consequence of compression; those of domestic animals are due to tension.

The sustenance of the enormous weight of the digestive viscera of solipeds and ruminants, to which is added in advanced pregnancy in the mare and cow approximately 20 per cent of the total body weight, causes the abdominal floor to sag markedly. The linea alba, extending from the pubic brim to the sternum, stays the abdominal floor. It is assisted by the notably developed abdominal muscles, and by the powerful panniculous carnosus and vast elastic addominal tunic, which are far more developed than in human anatomy.

Physiologically the longitudinal axis of the vagina and gravid uterus extends obliquely forward and downward from the vulva, and the weight of the pregnant uterus keeps its cervical pole tensely stretched. When the abdomen becomes unusually pendant, the obliquity of the central utero-vaginal axis inevitably increases, and it may be gently curved. This does not imperil pregnancy: when labor sets in the impediment of gravity in the expulsion of the fetus may be overcome by the female lying down. It appears erroneous to class this as anteflexion or anteversion. Some cases described as latero-version in the mare are probably bicornual pregnancy.

Uterine Torsion

Uterine torsion is commonly defined as a revolution of the uterus upon its long axis. A study of Fig. 14 indicates that in the cow, in which by far most uterine torsions occur, each cornu constitutes the arc of a circle and that torsion of the organ involves the revolution of a U-shaped loop, much like intestinal volvulus.

The point of incarceration of the genital tube of ruminants is usually in the vaginal area. Normally the uterine cornua are too firmly distended with fetal fluids to permit the revolution upon the cranial side of the cervix. The rigid cervix revolves with the cornua. The caudal end of the vagina is firmly fixed within the pelvis to the perineum, and torsion in that area is excluded. The cervical pole of the vagina, normally empty and with pliable walls, offers the most

favorable area for the torsion: it serves as the fixed and constricted mouth of the bag upon which the distended uterine sac revolves. When the torsion becomes extreme, and rupture occurs in ruminants, it is commonly immediately caudal to the cervix.

Rarely the uterus may be so flaccid because of disease that the fetal fluids gravitate toward the ovarian poles of the cornua and permit the torsion to occur immediately in front of the cervix at the juncture of the two cornua. Harms[80] states that in twin pregnancy, and in multipara, only one section of the uterus is involved. Apparently he had in mind only multipara. A study of Fig. 14 indicates that in bicornual twin pregnancy in the cow, as in the normal uniparous pregnancy, the two uterine cornua, firmly bound together by the intercornual ligament, must inevitably revolve together.

It is understandable that in unicornual twins, especially when independent, that portion of the uterus containing the fetus at the ovarian pole of the gravid cornu may undergo torsion without involving the cervical pole. It is to be remembered that in advanced gestation of independent unicornual twins, the cornu reaches a length of eight feet or more, and that the length of each fetus exceeds the distance from the pelvis to the diaphragm. The cornu necessarily becomes acutely bent in its middle. The two segments of the cornu, each containing a fetus, thus come to lie side-by-side, and the ovarian segment may, theoretically, revolve and cause a torsion between the two ampullae. This is far less probable if chorionic fusion, which regularly occurs, is present, and the cornu is equally distended at all points. If, however, the cervical segment revolves, the non-gravid cornu as well as the ovarian pole of the gravid cornu must accompany it in the torsion.

The non-pregnant uterus of the mare (Fig. 13) is cruciform and suspended hammock-like in the broad ligaments. With the advent of normal pregnancy, the gravid horn and the body elongate almost equally and at the juncture of these the non-gravid cornu is given off somewhat like the thumb of a mitten, as indicated in Fig. 50. The non-gravid cornu with its broad ligament acts as a barrier to torsion. It is probably in part for this reason that uterine torsion is relatively rare in the mare, according to the prevailing methods of diagnosis. I have identified but one case. The mare, in advanced pregnancy, had repeatedly climbed a straw stack, probably owing to pain, and lain down in such a position that she could not get up. The owner rolled her over backwards. Some weeks later she appeared unwell without marked pathognomonic symptoms. I found, upon rectal palpation, severe uterine torsion with gangrene.



FIG 162. The Allantochorion of the Mare.

The non-vascular endodermic layer has been removed in order to fully expose the vascular network.

1, Arterial trunk; 2, venous trunk; 3, 3, ultimate arterial and venous trunks, communicating with each other by means of sinusoidal or cavernous vessels of equal size; 4, secondary venous trunk; 5, a sinusoidal vessel apparently passing directly from artery to vein; 6, a very fine, non-branching anastomotic arterial vessel. *(Cornell Veterinarian)*

The conditions are materially different in multipara, which for all practical purposes have twin uteri, devoid of inter-cornual ligaments, as may readily be seen in Figs. 16 and 17. Each cornu is free to revolve independently, and only very rarely are both cornua simultaneously in torsion. Benesch[12] reports a series of cases in the bitch, all unicornual. The torsion of one horn would inevitably cause some constriction at the base of the other, as may well be understood by studying Fig. 163. When several or many fetuses are present, the uterus becomes greatly elongated and folded like the small intestines, and torsion of a short section of the cornu, containing one or more fetuses, may occur, like intestinal volvulus. This possibility is best illustrated in the sow, as shown in Fig. 18, the uterus of which may contain 12 to 20 fetuses and acquire a length of 15 to 20 or more feet, compared with some 80 feet of intestines.



FIG. 163. Pregnant Uterus of Bitch in Situ.

U, Direct cornua; *U′*, retrograde cornua. *In the presence of numerous fetuses in advanced pregnancy, the uterus becomes thrown into loops like the small intestines.* (See also Fig. 17.)

Numerous hypotheses have been advanced concerning the causes of uterine torsion. Rolling the mare or cow in an effort to undo torsion is a common method. But uterine torsion is very rare in the mare—I have seen but one case—and extremely common in the cow. The former habitually rolls, while the latter does not attempt it. The arciform disposition of the bovine uterus, and the attachments of its broad ligaments far back in the pelvis, render torsion more probable. Her method of lying down and getting up, by which the gravid uterus is virtually suspended for a brief period, apparently favors the dislocation. But in other ruminants having these anatomical characters torsion is rare. Others attribute the torsion to the movements of the fetus. According to my observations, the most abundant and violent fetal movements are seen in the mare, where torsion is uncommon.

Stoss[189] cites Tapken as having observed a ratio of 2.7 per cent of torsions among dystocias of cows at pasture, compared with 8.6 per cent in stabled cows. In the tropics I have not seen uterine torsion in beef cows, nor in dairy cows where constantly at pasture. My cases have all been in dairy cows, much stabled and of low vigor. The incidence has advanced and retreated somewhat parallel to that of sterility and abortion.

The conclusion that the basic cause of uterine torsion is to be sought in a want of vigor in the uterus itself is supported by the rare occurrence of torsion in the non-gravid animal. Harms[80] cites Hammerschmidt as having observed uterine torsion in a cow, 4 days post-partum, with the uterus distended with pus. Detorsion was effected by rolling her over 20 times. Harms also cites König as having observed post-partum uterine torsion in a goat. Benesch[12] observed torsion of the uterus in a bitch which had neither been pregnant nor bred, with the involved cornu filled with pus.

The diagnosis of uterine torsion is not difficult in severe cases: in the milder forms the line of demarcation between normal and abnormal has not been clearly defined.

A study of Fig. 14 shows that the ruminant uterus is disposed in the form of an arc or loop, with the ovarian pole ventral to the cervical end. The ideally healthy pregnant uterus retains the outlines of the non-gravid organ, with the median line of its convex dorsal border extending almost directly forward in a line with the long axis of the vagina. When the uterus is atonic, as is highly prevalent in dairy cows, the mid-dorsal line of the gravid horn sags to the right or left. The slight displacement attracts no attention, and causes no recognizable dystocia. Veterinary obstetrists are familiar

with dystocia due to dorso-ilial or dorso-pubic position. The fetus is said to have revolved upon its long axis 90 or 180 degrees. It is not generally stated that the uterus and the fetus have revolved together. The pregnant uniparous uterus with its contained fetus lies *en arc* with the convex dorsal surface of the fetus parallel with the convex dorsal wall of the uterus. It is logical to assume that, when a fetus lies in the dorso-ilial position, the uterus also has revolved so that its convex surface is directed toward the ilium. The dystocia is overcome by revolving the fetus upon its long axis. The question arises whether at the same time the obstetrist does not also revolve the uterus. The correction of the dorso-ilial or dorso-pubic position of the fetus is performed in the same manner as detorsion of the uterus when it has revolved through 180 degrees.

The confusion is intensified because some influential writers adhere to the tradition that during gestation the equine and bovine fetuses rest in the dorso-ilial or dorso-pubic position. The origin of this tradition is unknown. In clinical examination, I have constantly found the healthy fetus in the dorso-sacral position as indicated in Colored Plate I. Colleagues verify my observations.

The supporters of the tradition that the healthy equine and bovine fetuses lie in dorso-ilial or dorso-pubic position during pregnancy, fail to state at what date and by what means the fetal position is changed to dorso-sacral, the physiological position at birth. Clinical experience teaches that, in overcoming dystocia due to dorso-pubic position, the obstetrist may fail at his first effort to rotate the fetus upon its long axis, but may readily succeed in a second effort when rotation is attempted in the opposite direction.

It appears improbable that a healthy fetus would come to rest in an arciform uterus, with its ventral, concave surface directed toward the dorsal convex face of the uterus. In my experience, most fetuses in the dorso-sacral position are dead and the uterus atonic from disease. It seems highly probable that in most cases of dystocia due to dorso-ilial and dorso-pubic positions, the basic cause is to be sought in a diseased uterus and fetus, both of which have revolved through 90 to 180 degrees. If these be regarded as uterine torsion, the incidence of the displacement becomes multiplied.

Uterine torsion is defined as right and left, or clockwise and counter-clockwise. In the former it is meant that the dorsal line of the uterus moves to the right toward the ilium, thence toward the pubis, etc. Recorded statistics indicate that right torsion is the more frequent. The reason suggested is that the dorsum of the uterus rolls away from the rumen. Another unstudied possibility is that the tor-

sion is usually to the right when the pregnancy is in the right horn, and vice versa.

There are no pathognomonic symptoms of uterine torsion. The mild sagging of the atonic uterus, 45 to 90 or more degrees, passes unobserved unless accidentally discovered during rectal palpation. I incidentally discovered extreme uterine torsion in a cow during routine rectal palpation in the herd. The cow was apparently healthy. Yet the torsion was so extensive that the animal was rolled over six times in order to reduce the displacement. Her pregnancy continued to term, and she calved physiologically.

When torsion occurs at or near term, and progresses to such a degree that the uterine circulation is impeded or blocked, obscure signs of distress appear, varying according to the multitudinous lesions. When the torsion is so mild as 90 to 180 degrees, with the fetus in dorso-ilial or dorso-pubic position, the symptoms are merely those of the resulting dystocia. Vaginal and rectal palpation then reveal the nature of the difficulty. If the torsion is extensive, the vaginal walls are regularly thrown into spiral folds, which are readily recognized, and their direction indicates the direction of the torsion. When the torsion is very severe, the vagina becomes so narrowed that the cervix cannot be reached. If it extends further, the vagina may rupture transversely at its anterior end, or the uterus may rupture just anterior to the cervix, and the fetus may drop out of the uterus into the peritoneal cavity.

Rarely the torsion may be disseminated over a wide area and the revolutions be multiple without causing either the death of the animal or the rupture of the uterus. I have elsewhere described[246] a case of torsion of the uterus in advanced pregnancy, in which there were two complete revolutions of the pregnant organ. Although the cow was in indifferent health, she endured the displacement for about 23 months. Boyd[18] describes a similar case but designates it *mummification* with torsion present. I have regarded these cases as distinct from the typical bovine hematic mummy as illustrated in Figs. 177-180 inclusive, and have ventured to designate them as *static fetal cadavers*. There is no recognizable inter-placental hemorrhage in these cases, according to my observations. Neither is there visible desiccation of the fetus, nor signs of fetal decomposition if left undisturbed surgically. My conclusion has been that the fetal death occurs from a wholly unknown cause, which induces neither labor pains, fetal decomposition nor estrum, and the fetal cadaver remains in a state suggesting permanent *rigor mortis*.

Boyd attributed the death of the fetus to the uterine torsion. I have

observed the fetal phenomenon, in a range beef cow, without the uterine displacement. My conclusion is that the torsion is due to the presence of the fetal cadaver. The fetal fluids disappear, or rather fail to be longer produced, leaving the atonic uterus enlarged and flaccid, so that any violent movement of the cow may cause the mechanical displacement of the uterus. In the absence of fetal fluids and of uterine tone, there exists no automatic remedy and the displacement persists. The dearth of fetal fluids permits the uterus to revolve two or more times upon its axis without rupturing its walls.

Accounts occasionally appear in which the bovine uterus has made at least two complete revolutions without causing rupture of the organ or immediately mortal injury. It is not clear what ratio of such cases may be due to the *static fetal cadaver*. In one case the dairyman reported that at the expected calving period the cow had revealed signs of approaching parturition.

Rarely the torsion of the uterus occurs far forward. It has been stated that sometimes but one born—the gravid one—is involved in ruminants. The claim has not been verified and appears highly improbable. It could only occur in that portion of the apex of the cornu projecting anterior to the intercornual ligament, 9, Fig. 187.

In other instances, uterine gangrene occurs, with purulent decomposition of the fetus and early death of the female. Sometimes the uterus acts as an abscess cavity, and the fetal debris may slough out through the abdominal floor, as indicated in Fig. 164. Or the fetal debris may slough into the rumen, intestine or urinary bladder.

Uterine torsion in multipara is dfficult of diagnosis because the vagina is usually not involved and rectal palpation is not available. The patient is apparently unwell and the presence of pregnancy logically suggests the possibility of uterine displacement. The abdominal walls of carnivora are thin and pliable, affording an opportunity for the palpation of the uterus. This may reveal the tense, hardened area of torsion. The displacement may be quite overlooked, and, as indicated in Fig. 165, the segment which has undergone torsion becomes necrotic and the contained fetal cadaver, after some maceration, desiccates and remains indefinitely as a static mass of debris. The case is more fully described by Stephenson, Sweet, and Williams.[185] Uterine torsion, with rupture of the uterus and the extrusion of the living or fresh fetuses, is perhaps responsible for such extra-uterine fetuses as shown in Fig. 166.

The extent of the torsion and its effect upon the uterus and fetus vary infinitely. Probably most cases of 45 degrees or less become spontaneously adjusted, especially when labor sets in and the uterus

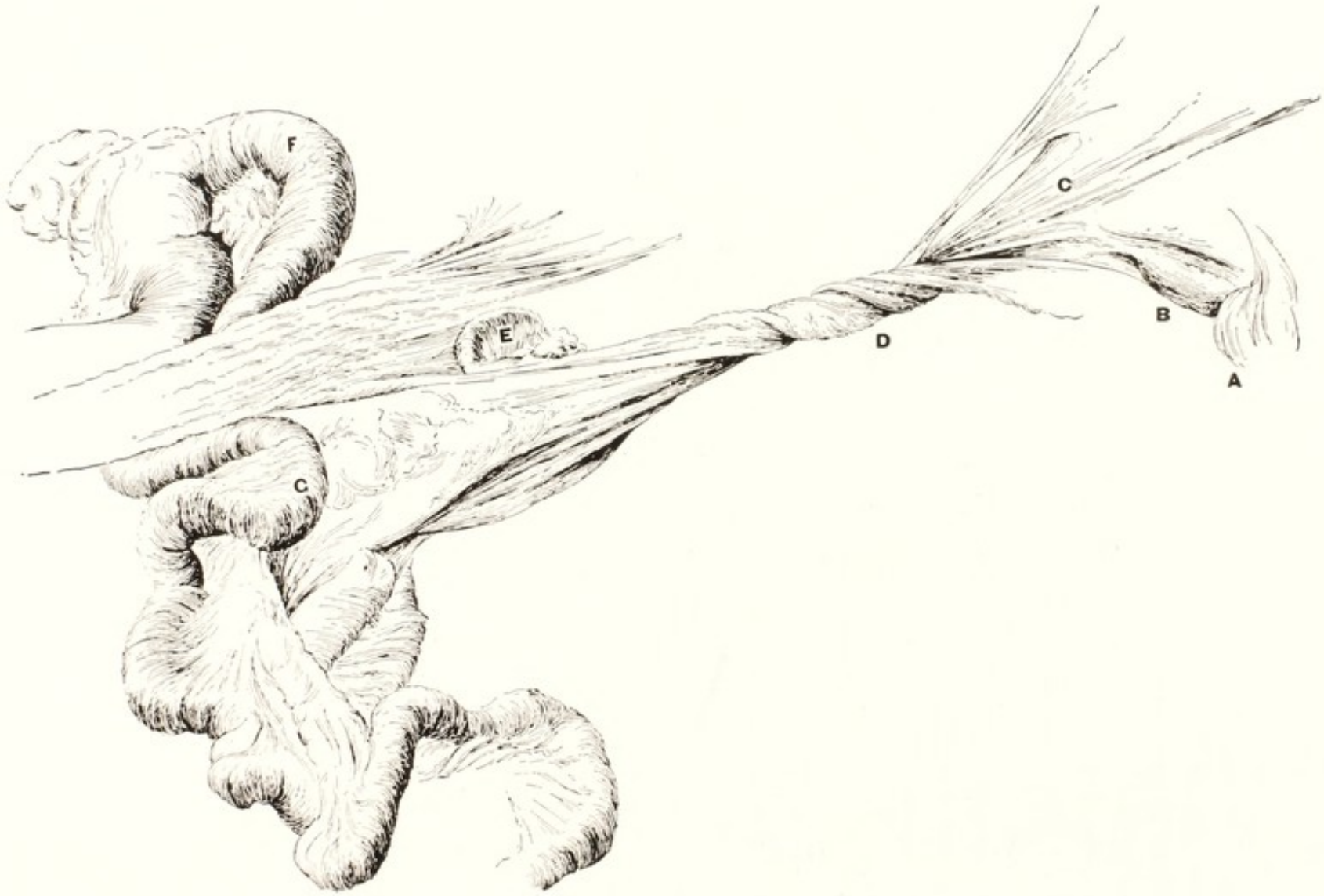


FIG. 164. Transverse Rupture of the Vagina of the Ewe due to Uterine Torsion.
Followed by sloughing of the putrid fetus through the abdominal floor.
A, vulva; *B*, vaginal stump; *C*, broad ligament; *D*, cord-like cervix entwined with broad ligament; *E*, ovary; *F*, uterine cornu; *G*, intestine.

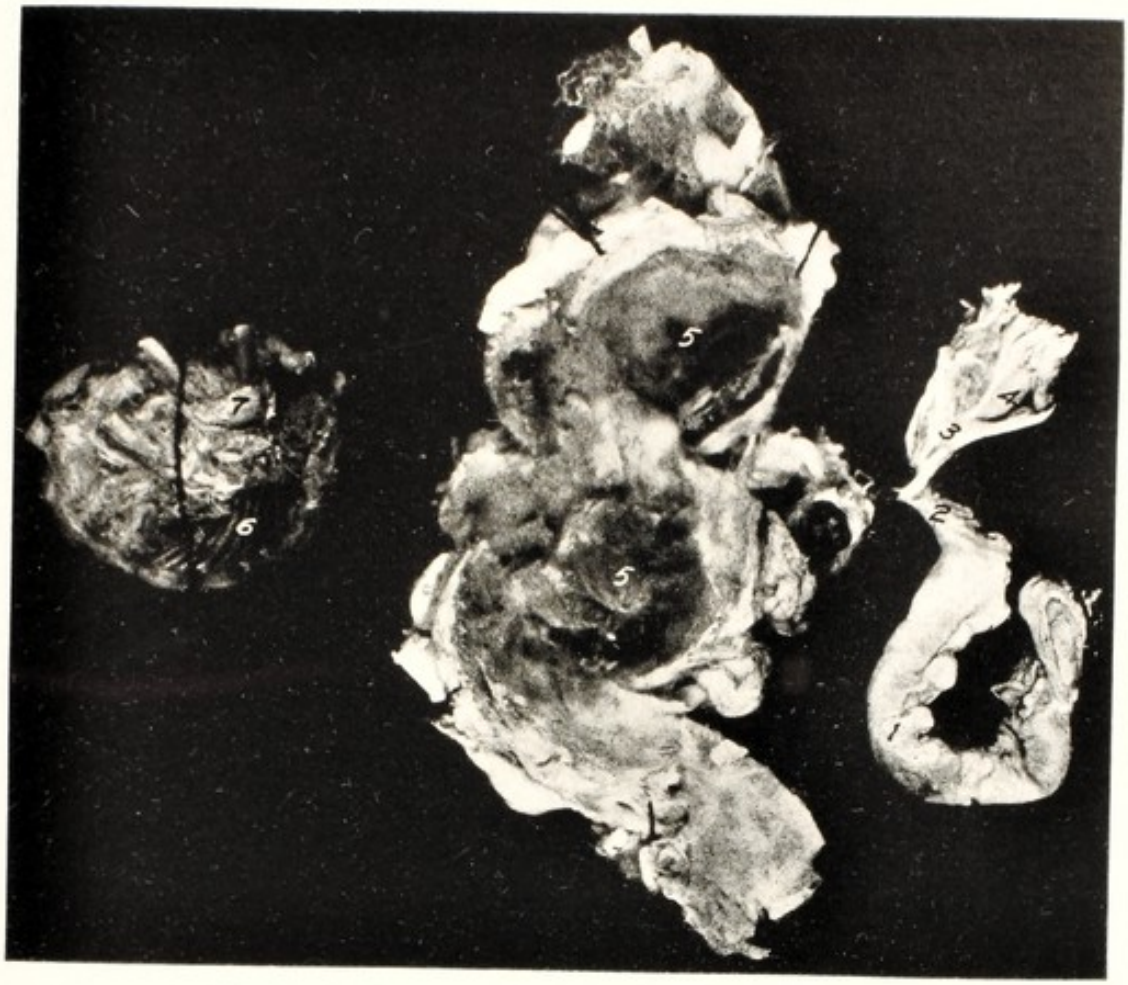


FIG. 165. Unicornual Uterine Torsion in a Cat.

1, Right uterine cornu; 2, base of left cornu; 3, ovarian pole of left cornu; 4, left ovary; 5, 5, fibrous capsule and remnants of necrotic ampulla isolated by uterine torsion and containing the fetal cadaver, 6, 7; 6, fetal ribs; 7, femoro-tibial articulation. *(Cornell Veterinarian)*

becomes aroused in vigor. Some cases are revealed by the dystocia due to the dorso-ilial position. When the torsion extends to 180 degrees in advanced pregnancy, if to the right, the left broad ligament is stretched over the vagino-cervical axis and tends to strangulate the uterus and to interfere with the blood supply. The fetus is then in dorso-pubic position and causes definite dystocia when labor sets in. When the torsion attains about 360 degrees, the tightly stretched broad ligament, containing the ovarian, uterine, and vaginal arteries, dangerously strangulates the uterus, and obstructs the blood flow to the uterus and fetus. The twisting of the uterus occasionally causes its transverse rupture. This may release the torsion, in which case vaginal exploration may reveal a free opening into the peritoneal cavity. The fetus falls out of the uterus and lies free in the abdominal cavity. Or the rupture may be hidden by the intact broad ligaments and fail to be identifiable until detorsion has occurred.

Extreme torsion imperils the life of the fetus and mother. As soon as the incarceration at the point of revolution seriously obstructs the return of venous blood from the placenta, asphyxiation of the fetus is threatened. In a bitch Benesch[12] observed violent fetal movements, evidently the effect of partial asphyxiation, thus indicating that the displacement of the uterus caused the violent fetal movements, not that the fetal movements caused the torsion. The obstruction of the

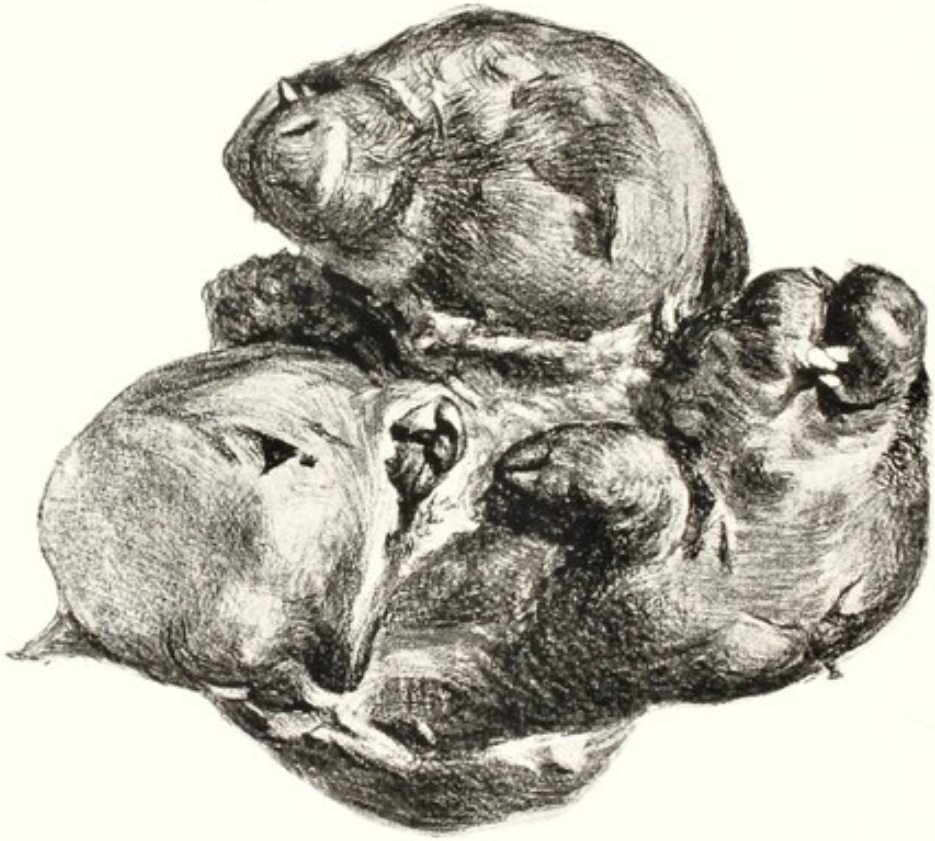


FIG. 166. Extra-uterine Swine Fetal Cadavers.
Presumably resulting from strangulated torsion of a uterine loop containing two fetuses.

circulation causes transudation of blood into the fetal sacs, reddening the fetal fluids. The uterine walls become engorged and bloody exudate appears in the peritoneal cavity.

Maceration of the fetus regularly follows its death, frequently accompanied by emphysematous decomposition. Sometimes, as in Fig. 165, the products of maceration are resorbed, and the dried skeletal debris remains indefinitely. Unless the uterus undergoes complete gangrene, adhesions occur between that organ and any contiguous structures. Occasionally, especially in swine, encapsulated extra-uterine fetuses are found, as in Fig. 166, which possibly may have dropped into the peritoneal cavity through the uterine rupture which had been caused by torsion. In some cases when the uterus ruptures transversely the fetus remains imprisoned for a time, as

shown in Fig. 164, the uterus retains sufficient vascular supply to avoid total gangrene, the walls adhere to the adjacent parts, the fetal cadaver decomposes and the pus and debris are discharged externally. Perhaps the cow to be described later, the uterus of which contained large volumes of whole grains of maize, mixed with fetal debris, had suffered from uterine torsion, the uterus adhered to the rumen and the two cavities interchanged contents. Similarly, the sloughing of the fetal cadaver through the abdominal floor, in Fig. 186, was possibly the effect of uterine torsion.

The prognosis of uterine torsion has been based chiefly upon the survival of the dam and her young. In this treatise we are primarily concerned with the question of the maintenance of fertility. From this angle the prognosis appears very bad. The data from this viewpoint are limited and vague. Various writers, many of whom are cited by Harms[80] record a mortality of the dam of 13 to 15 per cent. It is not clear what cases are included. A large ratio of ruptures of the uterus must end in death, and it is not clear that writers include those which when first examined are clearly doomed. I have described above one case in the mare. When I was called the uterus was gangrenous and the mare evidently moribund from sepsis. That gave me 100 per cent mortality for the mare. But I handled scores of mares in dystocia due to dorso-ilial or dorso-pubic position, possibly due to unidentified torsion, which largely recovered physically, though their later fertility was not learned. If the torsion is not extreme, if the vascular supply to the uterus has not been markedly interrupted, and if the torsion has been promptly reduced, the prognosis is good.

The outlook for the fetus is not so good as for the dam. Asphyxiation may ensue and the uterus remain viable. Or the fetus, already weakened in a uterus predisposed to torsion because of atony, and suffering from deficient blood supply due to the torsion, may well be born, or extracted, weak or non-viable. The prognosis is markedly individualistic, and depends upon a multitude of factors.

The prophylaxis of uterine torsion has not been directly discussed in veterinary literature. While accidents in endless variety have been invoked as causes, they are largely unpredictable and therefore unpreventable. The hypothesis that it is caused by the movements of the fetus, has no known basis in fact. The one possible means of prevention rests upon the conclusion stated above, that the fundamental cause consists of uterine atony or paresis dependent upon the methods employed in breeding, feeding, and handling. From this viewpoint, uterine torsion is largely preventable by avoiding the basic causes of genital diseases, as discussed in Section I.

The handling of the existing torsion has been considered in the companion volume, *Veterinary Obstetrics*.

Hysterocele. Uterine Hernia

The phenomena designated as uterine herniae vary in type. Inguinal uterine hernia, already mentioned in Chapter III as a teratological defect especially in the bitch and sow, consists of the migration of the ovary through the inguinal canal, analogous to the descent of the testicle in the male. Accordingly inguinal hernia in the female normally has as its content the oviduct, the ovary, and probably the ovarian pole of the uterine cornu. This presents a congenital tumor in the same region as the male scrotum, recognizable in the new-born female. She should be discarded as a breeding animal, along with siblings or other closely related individuals. If bred, inguinal pregnancy is probable. The growth of the fetus or fetuses causes notable enlargement, and eventually dystocia. It calls for hysterotomy or hysterectomy. If performed in due season, the prognosis is favorable for the lives of the both parent and offspring, after which all should be discarded as breeders.

Accidental ventral hernia of the uterus sometimes occurs during pregnancy or possibly, in rare cases, prior to conception. The additional load of pregnancy upon the abdominal floor facilitates rupture of the muscles, especially in solipeds and ruminants. Accident then plays its part.

Most observers state that in the cow the rupture is on the right side of the linea alba. Perhaps that is because the rumen occupies the left side of the abdomen, while the uterus and fetus are pushed to the right. When violence occurs, the impact of the fetus may be concentrated upon a limited area of the abdominal floor, causing rupture.

Early in pregnancy the uterus, closely confined in the pelvic region, does not drop into a hernia of small, or moderate size; later, when the uterus enlarges, the hernial ring does not readily admit the passage of the fetus. It follows that most herniae of this type develop late in gestation and are dependent upon a rupture during advanced pregnancy. Accordingly they usually appear abruptly and quickly attain their maximum size. Writers record instances of such herniae in cows, induced by the extraordinary pregnancy load of twins or of amnio-allantoic dropsy. It has been claimed in preceding sections that twin gestation is commonly pathological. The amnio-allantoic dropsy involves a general want of vigor of the female.

Rupture of the Prepubian Tendon

Rupture of the prepubian tendon occurs not infrequently in the

mare. Usually it appears without known violence having occurred, and in the absence of such phenomena as amnio-allantoic dropsy or twins.

In one of my cases, a draft mare in advanced pregnancy, and apparently in perfect health, became deeply mired while crossing a muddy brook. She struggled violently in her efforts to regain solid ground, with her anterior feet on the bank and hind legs engulfed in sticky mud. The strain upon the linea alba caused rupture of the prepubian tendon and an immense hernia which almost touched the ground. When I saw her a few hours later she was in near-collapse and was immediately destroyed.



FIG. 167. Rupture of Prepubian Tendon of Mare in Advanced Pregnancy.

The udder and one teat are shown displaced forwards. There is slight lordosis and marked depression of the supero-external angle of the ilium and elevation of the tuberosity of the ischium and of the caudal end of the sacrum.

The disease usually advances slowly, being first revealed by a marked edema along the abdominal floor. The mare shows slight disinclination to move about. The details of the symptoms have not been well studied. My impression is that the mare does not usually lie down. The horse is our greatest animal for persistently maintaining the standing posture whether awake or asleep. In the presence of disease which may interfere with getting up, the soliped largely

avoids the difficulty by not lying down. In the disease under consideration persistent standing, while avoiding the perils of getting up, may increase the edema and aggravate the disease which is weakening the linea alba.

It is claimed by some that the edema of the abdominal floor hides for a time a definite rupture. There appears to be equal ground for believing that the edema involves alike the linea alba and the subcutaneous connective tissue and that the edema is the forerunner of the rupture, rather than the mask which obscures its presence.

As indicated in Fig. 167, when the prepubian tendon begins to give way, it involves the two inguinal rings, and drags the mammary glands forward and downward along the floor of the abdomen. At the same time, the destruction of the linea alba, which normally serves as a stay between the pubis and sternum, permits the dorso-lumbar spine to become depressed and, as in nymphomania in the cow, the pelvis tilts forward upon the coxo-femoral articulations, the anterior or superior tuberosities of the ilia become depressed, and the caudal ends of the ischia and sacrum elevated.

The dystocia resulting from this rupture is discussed in the companion volume, *Veterinary Obstetrics*.

Strangulation of the Colon by the Gravid Uterus

I have recorded at some length[247] the occurrence of one case, in a thoroughbred mare, of the displacement of the great colon beneath the gravid uterus, with confusing symptoms, which prevented clinical diagnosis. The nature of the disease became apparent only at autopsy.

Prolapse of the Vagina

Vaginal prolapse may occur in any species of domestic mammal. It is observed chiefly in solipeds and ruminants and acquires its chief importance in dairy cows. In the cow the phenomenon generally consists, primarily, of the caudal displacement of cervix and uterus in such a manner that the vaginal end of the cervix moves backward to the vulva or protrudes beyond. This necessitates the invagination and final eversion of the cervical end of the vagina as a secondary complication. A study of Colored Plate I and Fig. 14 indicates that the displacement involves a relaxation of the broad ligaments of the uterus and cervix which permits these organs to move toward the vulva. A second type of cases may be primarily a prolapse of the vagina due to irritant vaginal lesions which induce straining by the patient. The first type is almost unknown in multipara because of the greatly elongated vagino-cervical canal and primitive develop-

ment of the cervix. Accordingly the phenomenon known to veterinarians as vaginal prolapse is most abundant and serious in the cow, where the cervix is most complex and suffers most frequently and seriously from disease.

Vaginal prolapse is most frequently observed in the pregnant cow. In the non-pregnant cow it is most frequent as a complication of nymphomania.

The symptoms as observed in the pregnant cow call for no extensive description. As a rule the prolapse becomes apparent only when the cow is recumbent. The cervix and portions of the vagina protruding through the vulva become irritated from exposure to the air and from stable dust, and in severe cases the protruding portion comes in contact with bedding. The vaginal and cervical mucosa become inflamed and swollen, and muco-pus is excreted. The vulva and the vaginal walls are atonic and may be edematous. The non-pregnant nymphomaniac presents a similar type of phenomena: there is relaxation of the vulva and vagina.

In these types there are no significant signs of distress which may be ascribed to the prolapse. There are no recognizable expulsive efforts which might push the uterus and cervix into the pelvis and vagina. There is no abnormal pressure from the abdominal viscera. Prolapse is not observed as a result of twin or multiple pregnancy nor in distension of the abdomen owing to amnio-allantoic dropsy. In the vaginal prolapse of nymphomania the cow is gaunt instead of having the abdomen distended with solids, liquids, or gases. In the presence of extreme tympany because of indigestion, the extreme intra-abdominal pressure causes a degree of vulvo-vaginal inversion for a brief interval, which subsides immediately when the tympany ceases: it is a purely mechanical prolapse wholly unrelated to the phenomenon under consideration. Reference has been made in Section II to prolapse of the vagina following coital injuries, but in that disease there are great expulsive efforts due to pain, and the consequent prolapse is in no wise related to the vaginal prolapse of pregnancy and nymphomania. In the one type the prolapse is due to severe straining; in the other it is the result of pathological relaxation and atony of the uterus, vagina, and vulva.

The prevention of prolapse of the vagina needs be based upon the cause. The prolapse in pregnant cows cannot be prevented except one can maintain the systemic and sexual vigor of the animal; the prolapse in nymphomania is only preventable by preventing the cystic degeneration of the ovaries. The injurious effects of the disease in the pregnant cow may be ameliorated by releasing the patient

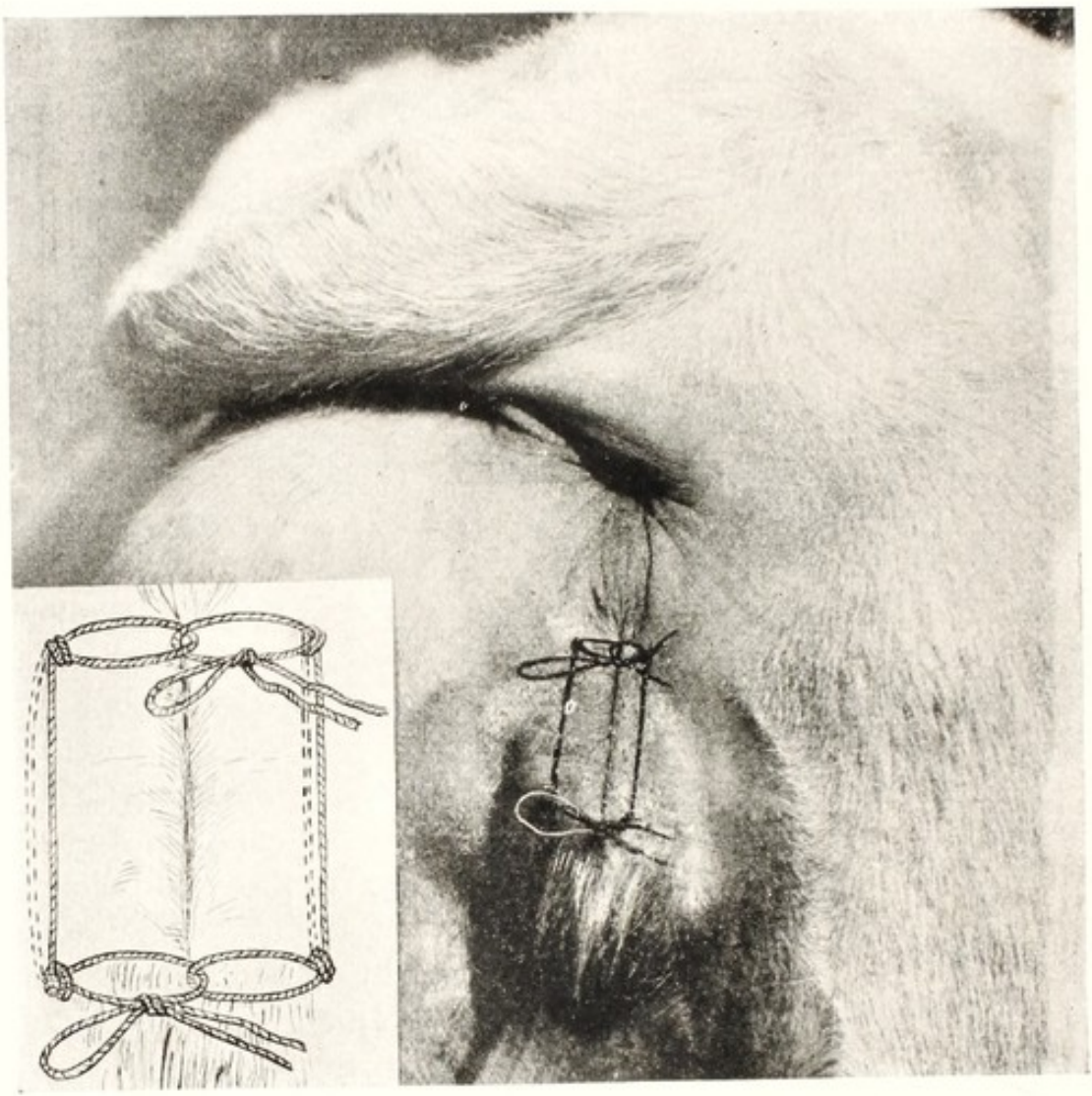


FIG. 168. Sutures for Uterine and Vaginal Prolapse.
Details of Sutures shown in inset.

from the stanchion, by allowing adequate exercise and by feeding moderately upon a laxative diet. The protrusion of the cervix from the vulva may be overcome by the application of vulvar sutures, as shown in Fig. 168, but the patient then needs be closely watched, lest labor set in and the sutures be torn out, or calving otherwise interrupted.

The cow which suffers once from prolapse of the vagina usually repeats the displacement at each subsequent parturition. There is present an inherent weakness in the animal, which detracts from her value and usually renders her discard advisable, not so much upon her own account as because her progeny, according to general observations, are not safe as foundation breeding stock.

Chapter XXX

THE DISEASES OF THE GRAVID UTERUS AND OF THE CONTAINED OVUM

The Early Death of the Ovum Due to the Union of Defective Sex Cells

IT has been held in the preceding pages that the maximum number and value of new-born animals is primarily fixed by the combined health and vigor of the ova as they are discharged from the ovary, and of the spermatozoa which fertilize them. Any defect or weakness of the fertilized ovum due to deficiency in one or both of the sex cells is not generally subject to recovery.

In Section I it was held that numerous fertilized ova are so defective that they perish very early and disappear. Or they may rarely persist indefinitely as cystic moles, as in Fig. 48. Virtually no researches have been made regarding early death of the ovum in domestic animals. Mall[133] and other writers in the field of human obstetrics record the finding of numerous teratological defects in ova which have perished early. It has been emphasized in Section I that defective ova, classified as amorphous, acardiac and asexual monsters, perish immediately unless the defective ovum promptly acquires vascular communication with a normal twin or litter mate. This vascular anastomosis is rare except in cattle. Presumably such defectives are prevalent in other species, but vascular anastomosis is infrequent. It is a fair inference that many of the necrotic swine fetuses, like those in Figs. 91 and 92, have perished because of teratological defects. Those defective ova which survive the early stages of pregnancy largely die prior to the completion of the normal duration of gestation. Many of them live to term, to be expelled dead, and others are born in a more or less viable condition. It has been urged in the first section that such defectives are largely weaklings, highly subject to invasion by pathogenic forces. Thus, I have described at length[252] a young, cryptorchid boar having hypospadias. When he had reached about 200 lbs. in weight, he was destroyed because of an abundant highly fetid discharge from the urethra. Autopsy revealed abscesses of the vesiculae seminales and of the ampullae of the vasa defferentia. The teratological or congenital anatomical defects branded him as a weakling, with low resistance to invaders. *Bacillus abortus* and other incriminated invaders are not commonly

reported by bacteriologists in early bovine abortions. A virus may be present, but, excluding that, a defective ovum provides the most logical hypothesis. Certain it is that defective ova account for many early abortions. Following fertilization, the individual, in addition to retaining any deleterious primary deficiencies, is constantly exposed to new perils at each step of its existence. The fertilizing semen may be polluted, and the infection meet the ovum within the tube, and destroy it, or the ovum or ova may evade the infection in the tube, and, as shown in Fig. 151, pregnancy and hydrosalpinx coexist. It is a fair inference that infections within the oviducts cause a highly important ratio of disease and death of the fertilized ova during their migration through the tube.

When the fertilized ovum enters the uterus, the perils to its life become more apparent and the diseases are more readily traced. The power of the ovum to resist invasion increases with age, but it is detained within the uterus more than twenty times as long as within the tube.

The perils of genital disease are cumulative. Any non-mortal deficiency of the fertilized ovum which survives the migration through the oviduct lowers its resistance to intra-uterine dangers. While discussing intra-uterine infection it was noted that many laboratory workers deny the frequent existence of infection within the non-gravid uterine cavity. Others of high standing record an abundance of intra-uterine bacteria in apparently healthy uteri. These invaders under favorable conditions, such as a weak ovum, acquire definite pathogenic power. The long period of time during which the young remains intra-uterine provides additional opportunity for the invasion of the uterine cavity from the blood stream. Accordingly the intra-uterine young is beset by the perils inherited from a defective ovum or spermatozoon: it may suffer serious injury in its transit through the oviduct; it is exposed to the perils from infection domiciled in the organ and to invaders which may reach the uterine cavity during pregnancy.

Diseases of the uterus and of its contained young are reciprocal. It is well nigh impossible for either to suffer from severe disease without involving the other. Now and then the clinician chances upon an embryo, not more than 1 cm. long, being discharged from the vulva of a cow. The inference is unavoidable that almost all such embryonic deaths pass unseen. The statement is frequently made that, in a given case of abortion, the fetal membranes appeared healthy. The studies of Williams, Udall, et al[243] emphatically challenge this view and indicate that, when an embryo dies, the fetus,

the fetal membranes and the uterus suffer. Clinically the injuries to the ovum and the uterus are not observable until the fetal membranes have been formed. From that time onward to birth or abortion, the fetal membranes of all adeciduates constitute an authentic replica of the endometrium with which they had been in contact.

The fetal membranes or placentae constitute the most important organs of ante-natal life. Through these all food elements, including oxygen, must pass and all waste products, except those stored in the meconium, must be eliminated. They are frail organs, abruptly formed and even more suddenly discarded at the birth of the young. They offer an illimitable number of disease changes, each of which, as a rule, is registered alike upon the endometrium and chorion.

THE NECROTIC TIP OF THE CHORION

The tubular fetal sacs of ruminants and swine regularly bear, at their apices, a dry, necrotic mass of grayish or brownish color. In some instances the necrotic segment remains tubular and is distended with fluid, as seen in the upper left specimen in Fig. 96. The figure illustrates some of the infinite variations observed in the chorion of apparently healthy fetuses. The necrotic tip is essentially universal in ruminants and swine and is commonly regarded as purely physiological, regardless of the degree of necrosis. Zietzschmann[278] considers the structure as the necrosed apex of the primitive (amniotic) chorion, and attributes the necrosis to an absence of blood vessels. The necrosis, occurring prior to the formation of the allantois, bars that membrane from the involved area.

Clinical study indicates that, while such necrosis generally presents no evidence of pathological significance, cases are by no means rare in which disease is undeniable. It is not essential to draw a clear line of demarcation between the healthy and the diseased necrotic tip. In the most ideally healthy bovine fetal sac, the necrotic tip is frequently not more than 50 mm. x 2 mm. in volume and is buried in the walls of the living allantochorion. Then follow the varied types shown in Fig. 96. The size of the necrotic segment advances in volume by imperceptible gradations. The upper specimen in Fig. 97 shows a chorion from a badly damaged uterus, in which the caruncles of one cornu had been completely destroyed during a prior pregnancy, with an extremely short non-gravid chorion at 3. In the lower figure the entire non-gravid chorion may be regarded as a necrotic tip and must be classed as pathological. Fig. 169 depicts a necrotic tip of the non-gravid horn, the necrotic area being 30 inches long. The membranes, from a second calving, were removed manu-

ally after 36 hours. The necrotic segment had been completely invaginated into the allantoic sac. It differs radically from the examples in Fig. 96. Few would attempt to deny its pathological significance. The excessive volume and the invagination of the necrotic tip were not the sole evidences of disease. The retention of the membranes for



FIG. 169. Necrotic Tip of Chorion, Thirty Inches Long, Completely Invaginated Into the Functional Base of the Non-Gravid Cornu.

The placenta was removed manually thirty-six hours after calving: it was thick and leathery as is described in Bacillus abortus infection, but cow was negative to tests for that invader.

1, Necrotic, frayed cotyledons at the ovarian end of the functional portion of the chorion; 2, narrow portion of the invaginated necrotic tip; 3, broad, flask-shaped apex of the necrotic end. *(Cornell Veterinarian)*

36 hours was admittedly pathological. The chorion of the gravid horn was thick, sclerotic, leathery, of that character which for a time was considered proof of *Bacillus abortus* infection. The cow was negative to the agglutination test.

Figure 91 shows five fetal sacs from a swine uterus, three of which had been dead for perhaps 15 to 20 days. The living ova have

rather extensive necrotic tips. There is no macroscopic difference between the dead tips of these and of the three dead ova. The same force which caused the death of the three ova may have caused the necrosis of the tips of the live sacs. Fig. 92 illustrates the wide variation in necrosis of the fetal sacs of swine embryos.

My observation has been that, the greater the volume of the necrotic tips upon the living fetal sacs in a swine uterus, the lower the ratio of living embryos to the number of corpora lutea in the ovaries. In the inset of Fig. 18, the apparently healthy fetal sac has relatively small necrotic tips. A somewhat different picture is represented in Colored Plate II, in which the entire non-gravid cornu is necrotic and immersed in a large quantity of adhesive exudate, formerly designated the exudate of contagious abortion. Certainly this is pathological.

Additional light may be thrown upon the significance of the necrotic tip by studying chorionic fusion. When twins or higher multiple pregnancies occur in ruminants, the rule is chorionic fusion, the allantoic sacs becoming one continuous tube. In the cow the allantoic arteries anastomose to constitute a single trunk into which all umbilical arteries empty; in the ewe, as shown in Fig. 21, the fusion of all tissues of the allantochorion is complete except for the visible anastomosis of the blood vessels. No traces of necrotic tips are observed in the fused ends of the fetal sacs. This suggests very early fusion, at which time blood vessels, or the elements from which blood vessels are developed are present in the abutting ends of the fetal sacs. Rarely the sacs of bovine twins fail to fuse. At times both ova come from one ovary and the two embryos lie in close contact in one horn. If mere contact were the cause of fusion, unicornual twins should always fuse. The want of fusion in unicornual bovine twins suggests that the tips of the membranes of the two ova had become necrotic before contact. That contact appears to occur between the rapidly elongating tubular vitelline sacs. Swine fetal sacs also fuse: I have observed six of eight fetal sacs thoroughly fused and a general allantoic artery extending the entire length. Here also the fusion must have occurred early and the formation of the necrotic tips must have been prevented or minimized.

The spectacular fetal deformity designated *Schistosomus reflexus* is limited in its occurrence to ruminants and swine*, in which domestic

* A solitary exception to this law is occasionally cited as having been observed by Schöttler (Berl. Tierärztl. Wochenschrift, 1922. S. 492) but a study of the contribution reveals that only a fission of the body cavity existed, without any suggestion of reflexed body walls.

animals alone is found a rapidly growing, greatly elongated tubular vitelline sac. The monster is characterized by a reflexion of the somatopleure of the blastoderm so that the thoracic and abdominal viscera lie free in the allantoic cavity. The spinal column is folded dorsally and the four limbs and head more or less completely enclosed within the body walls. Through the courtesy of Prof. Fincher, I was able to study, in 1936, the afterbirth, Fig. 170, of one of these monsters.[261]

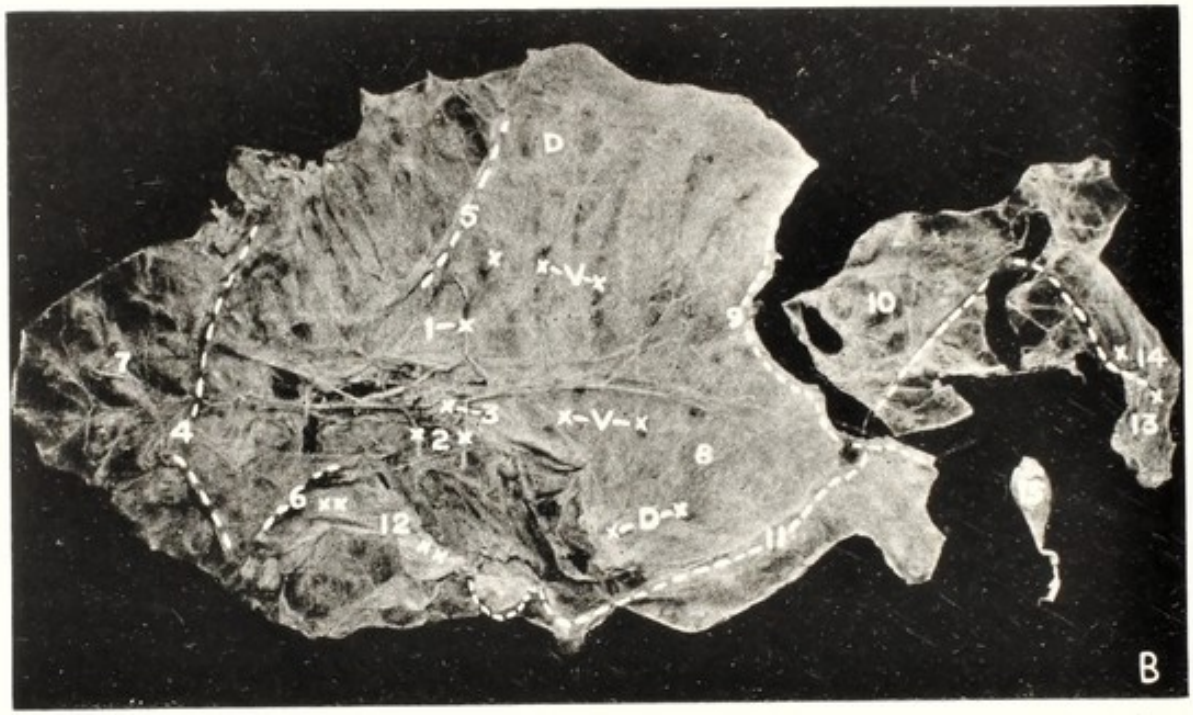


FIG. 170. Allantochorion from Bovine *Schistosomus Reflexus* Showing Necrotic Tip of Chorion and of the End of the Vitelline Tube (?), Viewed from Amniotic Side.

D, D, Dorsal rows of cotyledons seen through chorion; *V, V,* ventral rows; 1, umbilical vein; 2, umbilical arteries; 3, inter-umbilical anastomotic artery; 4, 5, 6, segments of the amnion rolled on rods in order to expose other parts; 7, allantoic cavity upon the ovarian side of the amnion; 8, allantoic cavity of the gravid cornu upon the cervical side of the amnion; 9, borderline between gravid and non-gravid cornua; 10, non-gravid cornu; 11, abnormal structure identified as the pathologically elongated, tubular vitelline sac; 12, funnel-shaped, fetal end of abnormal structure; 13, necrotic tip of chorion including the tip of the abnormal structure; 14, necrotic area in chorion. *(Cornell Veterinarian)*

The afterbirth revealed the presence of a tubular structure, 11, extending from the umbilical region, 12, to the apex of the non-gravid cornu, into the necrotic tip, 13. The tube, filled with fluid, varied between ¼ and ¾ inch in diameter and was 5.5 feet long. The structure was located near the center of the superior or dorsal chorionic wall in that area where Zietzschmann[278] and others locate the vitelline tube. It was readily seen from the placental side, but was more conspicuous from the internal surface, protruding somewhat into the allantoic cavity. Not long after having studied the mem-

branes described, a second afterbirth was obtained in the ambulatory clinic, in which the same tubular structure was present. So far as known, these are the only fetal membranes from *Schistosomus reflexus* thus far studied. Essentially identical, they forcibly suggest that this monstrosity has its origin in an abnormally developed vitelline tube, the apex of which, becoming caught in the necrotic tip of the chorion, fails to retract and disappear. This offers an insuperable barrier to the ventral folding of the somatopleure to form the body walls, restricts the formation of the amnion and causes reflexion instead of ventral folding of the body walls. A study of Fig. 169 in comparison with the afterbirths of the two cases of *Schistosomus reflexus,* furnishes the interesting suggestion that in the former there had been a similar incarceration of the vitelline tube in the necrotic tip, the chorion yielded and became inverted for 30 inches, after which the vitelline tube disappeared. In other words, the conditions illustrated in Figs. 169 and 170 had a common ancestry in an abnormal development of the vitelline duct: in Fig. 170 the apex held and the embryo was everted; in Fig. 169 the umbilical end prevailed and the apex of the chorion was inverted.

The thought is of additional interest in inversion and prolapse of the uterus. Occasionally the study of the afterbirth indicates that the apex of the uterine cornu has been inverted or intussuscepted during gestation. Following the termination of pregnancy, it is now and then found that the apex of a uterine horn is inverted.

It is of further interest to observe that, in the cases of *Schistosomus reflexus* cited, the umbilical vein in the one instance adequately studied, instead of passing through the liver to the heart via the posterior vena cava, evaded the liver and emptied into the anterior vena cava at about the point where the vitelline vein is presumed to connect. This would suggest that the reflexion occurred prior to the development of the amnion and allantois. Concurrent with the failure of the umbilical vein to attain its physiological relations with the liver, that highly important fetal organ is grossly defective in all cases I have observed. The liver, the seat of numerous cysts, contains little hepatic tissue macroscopically recognizable as such.

The acardiac or amorphous monsters so common in the cow have no umbilical cord, but instead have an afferent artery and efferent vein. It is not easy to understand that these were vascular extensions from the host to the parasite, but it would rather appear that the artery and vein are the vitelline vessels of the parasite which primarily fused with the vitelline tube of the host.

The problem of the basic origin of the necrotic tip and its possible

relation to important disease of the embryo or fetus seems to merit greater study than has yet been accorded.

As studied in the slaughter house, the necrotic tip of the fetal sac, when of the volume shown in Fig. 96, does not reveal a tendency to irritate the contiguous endometrium. If somewhat larger, there appear, with increasing emphasis, evidences of irritation. Pus or pus-like exudate is present in the apex of the cornu, the endometrium is thickened, calcification is visible and palpable, and other signs of limited disease are present without definite evidence of peril to the mother or young. As the area involved increases, the danger to mother and fetus becomes apparent. When the entire non-gravid cornu of the chorion becomes necrotic, as in Fig. 97 and Colored Plate II, the pathological significance becomes indisputable and the volume of the exudate enormous. When the volume of necrosis has reached that shown in the lower chorion of Fig. 97, the non-gravid cornu ceases to function thereafter and pathologically the uterus becomes unicornual as seen in both upper and lower figures. According to the general conception of disease, a necrotic mass of this character, within the living tissues or in a body cavity surrounded by vulnerable parts, serves as an inviting nidus in which pathogenic organisms may multiply in volume and virulence. Clinically the extension of disease of the uterus and fetal sac in ruminants and swine is largely from the ovarian poles toward the cervix. This may be ascribed: 1, to the presence of the necrotic tip; 2, to invasion from the oviduct; or 3, to the low resistance of the ovarian pole of the non-gravid cornu of the ruminant fetal sac, owing to its great distance from the fetal heart. Logically the three forces may cooperate.

Occasionally it is found, when an inverted necrotic tip of considerable size is present, that at the point of contact between the inverted tip and the living fetal sac, a definite contact necrosis is present. Rarely the area of contact necrosis, two or more inches in diameter, drops out of the chorion, leaving a broad avenue of communication between the allantoic cavity and the utero-chorionic space.

Although the necrotic tip of the fetal sac is not usually associated with visible evidences of interference with reproduction, there are numerous cases in which there is definite proof of its pathological importance. These are not authentically recognizable in the closed uterus of the living animal. Rectal palpation of the animal from which Fig. 38 was made would show an absence of development of the non-gravid cornu. The dwarfed condition might be due to early necrosis of the non-gravid cornu of the fetal sac during the current

pregnancy, or the caruncles of the uterine cornu may have been destroyed in a prior gestation, thus barring physiological development.

Placentitis. Metritis. Chorionitis

Placentation in domestic mammals varies widely. These differences profoundly influence the types of disease involving the uterus and the fetal sac. Placental disease is universal in abortion. If the placentitis be non-mortal for the fetus, it disturbs the duration of pregnancy, either abbreviating it, as is usually the result in the cow, or prolonging it, as is frequently observed in the mare. The uterine placentitis, if non-mortal, leaves a permanent defect in reproduction power.

EQUINE PLACENTITIS

Scant study has been accorded the placentitis of the mare, the chief attention being given to the bacteriology of sterility and abortion. I briefly described in 1926[245] some of the more common lesions, and with Gibbons made some additions in 1929.[249] The diffuse equine placenta tends largely toward equally diffuse placentitis. It is possibly this uniformity of the disease which causes it to escape identification in many instances. Perhaps it is this which causes some writers to state that abortion might occur without the presence of maternal and fetal placental disease. Careful study shows clearly that macroscopically recognizable placental lesions are present in all cases of abortion, as well as in prolonged gestation with a viable or non-viable foal. Thus in Fig. 30 is shown the placental side of the chorion of a foal born at 387 days (1 yr. 22 days). The foal was unable to stand until 2 days old. The afterbirth was grossly overweight. The mare had produced two viable foals during 11 breeding years. The placenta was thickly sprinkled over with necrotic patches, which appeared like old scabs with free borders, as shown at A. The membranes of another mare, aborting at 120 days, or about 30 days after the formation of the allantochorion, showed that extreme calcification had developed uniformly throughout the placental surface of the chorion. Doubtless parallel lesions existed in the endometrium. Figure 93 shows necrotic areas upon the non-vascular or inner side of the chorion of a non-viable foal born at term. The membranes weighed 18.5 lbs. A fibrinous clot, 1, measured 12 x 2 x 1 inches, while upon the opposite, placental side was a necrotic area 2 x 3 inches, so that the necrosis extended completely through the chorion. The necrotic area was held in situ merely by the great fibrous clot shown at 1. A smaller necrotic area dropped out while the membranes were

being handled. Calcification is common upon the placental side of the diseased equine chorion, and logically upon the endometrium as well.

The diffuse character of the placenta renders possible the formation of an inter-placental abscess. This I have observed in one instance in well advanced pregnancy. While the mare was at work in the field the owner observed the sudden expulsion from the vulva of a liter or two of pus. Abortion followed a day later. The membranes showed clearly the outlines of the abscess at the os uteri internum.

The outstanding macroscopic lesion in numerous cases of equine abortion consists of great distension of the allantoic veins and capillaries, giving them the appearance of aneurisms, as shown in Fig. 95. The details of the lesions are more extensively described in the cited literature. The diffuse placentation of the mare apparently acts as a deterrent to extensive purulent or septic metritis during pregnancy. Instead the fetus is expelled. When uterine torsion is neglected, fatal gangrene may occur. When dystocia goes unrelieved after the dilation of the cervical canal, mortal metritis generally follows quickly.

THE PLACENTITIS AND CHORIONITIS OF PREGNANT RUMINANTS

The placentation of ruminants provides opportunity for the development of types of pathological phenomena which are unknown in other groups. A study of Fig. 15 and of Colored Plate I shows that a vast utero-chorionic space exists in the cow. Regularly distributed over the entire endomentrium are 100 to 140 uterine caruncles, arranged in four parallel rows. In advanced pregnancy the caruncles project two or more inches above the general plane of the endomentrium and push apart the non-placental area of the uterus and chorion. At their summits the caruncles bear the physiological uterine placentae. Reciprocal placental areas form upon the surface of the chorion. The caruncular columns constitute a vast colonnade. There is formed between the non-placental portions of the endometrium and chorion a closed sac which at term is about 8 or 9 feet long, from ovarian pole to pole, and about three to four feet in circumference. Hence the utero-chorionic cavity offers a superficial area of twenty to thirty square feet with a depth of 1.5 to 2 inches, crossed by more than 100 caruncular columns. The inter-caruncular walls of the uterus and intercotyledonal walls of the chorion have only a moderate vascular supply; the placental areas command by far the greater volume of blood passing through the uterine and umbilical vessels.

In the abattoir there is repeatedly found in the utero-chorionic

space extensive volumes of an intensely adhesive, yellowish or brownish mass, generally known as "the exudate of contagious abortion." More extended studies have shown that its presence is not proof of identifiable invasion by *Bacillus abortus*. It is common in abortions ascribed to spirillae, streptococci, and other organisms, as well as in other pathological states of the pregnant uterus in which abortion does not occur. *Bacillus abortus* is now known to invade the bodies of all domestic mammals and is alleged to cause abortion in some,

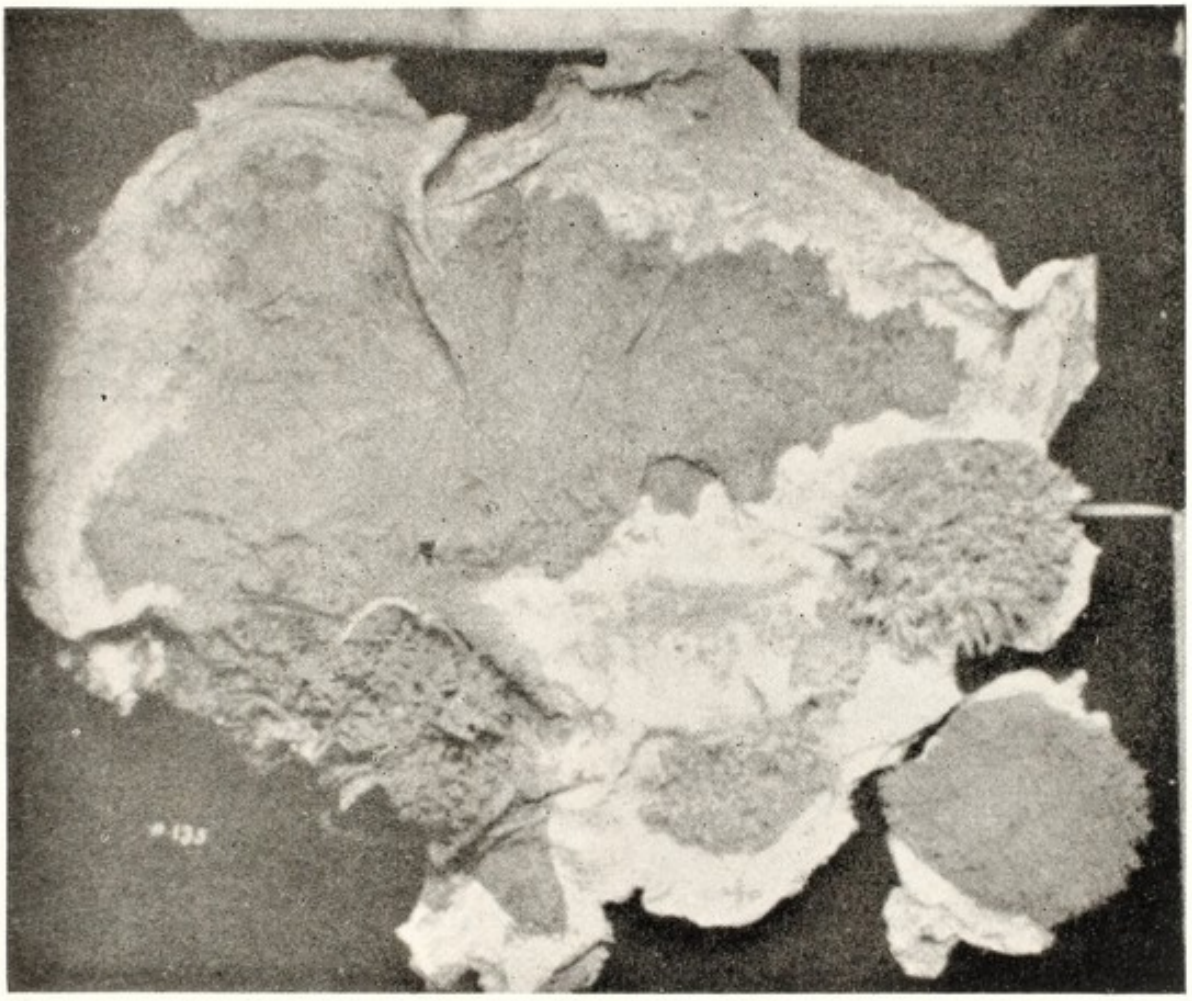


FIG. 171. Penetrant Necrosis of the Bovine Allantochorion.

The necrotic (dark) area occupies one-half the field. Along the lower margin are three necrotic, clumpy cotyledons; at the lower right a healthy cotyledon has been inserted for comparison. (Cornell Veterinarian)

but the *abortion exudate* as observed in the utero-chorionic space of ruminants is not known to be formed in solipeds or swine. *Bacillus abortus* is convicted of causing orchitis, semino-vesiculitis, carpal hygroma and other phenomena in bulls, and mastitis in cows, but the exudate of contagious abortion does not occur in any of these. The same is to be said of fistulous withers, poll evil and gonitis of horses. Clinically it would appear that the utero-chorionic space of ruminants

provides the chief, if not the sole environment in which "the exudate of contagious abortion" may be developed. It is apparently the result of a chronic infection or irritation involving the walls of the utero-chorionic cavity. Judging by recorded bacteriological studies, *Bacillus abortus* appears to be the most frequent invader.

The exudate is apparently produced collectively by the non-placental uterine mucosa, the non-placental surface of the chorion and the placental surfaces proper. The chorion contiguous to the exudate becomes injected, thickened, and indurated. Sometimes calcification is identifiable. In the abattoir, the uterine walls are correspondingly affected. Rarely, necrotic areas develop in the chorion, as shown in Fig. 171, which are penetrant and involve the entire thickness of the membrane, similar to the necrotic area of the equine placenta Fig. 93. Such necrosis in the bovine chorion apparently originates within the allantoic cavity, as it needs to do in the mare, and has no relation to the exudate within the utero-chorionic cavity. It has not been authentically determined what degree of injury "the exudate of contagious abortion" may cause to the uterus and contained fetus. Perhaps its chief significance is as a meter of pathological changes occurring within the placentae.

The associated placentitis imperils the health and life of the fetus. At birth, the diseased placenta is discarded. It has already implanted dangers in the fetus, which persist. Lesions corresponding to those of the chorionic placentae, occurring in the crypts of the caruncles, likewise remain permanently. The most frequent and significant changes readily observed are the necroses of the chorionic tufts. These profoundly delicate structures, as shown in Fig. 46, penetrate completely the crypts in the placental areas of the uterine caruncles. The crypts form subsequent to conception and their depth increases as pregnancy advances. The fetal placentae form upon the chorion, their location and extent being regimented by the uterine caruncles. Each chorionic area in contact with a functioning caruncle develops placental tufts. The tubular ruminant chorion develops very early and with great rapidity. As soon as it has extended from ovarian pole to pole, it is held in a permanent position by the placental areas of the caruncles. The force which holds the given chorionic area in permanent contact with the caruncular summit, is unknown. The adeciduate placentae are not held in contact by any anatomical anchorage: there is no continuity of tissues or known adhesive tissue between them.

The delicate chorionic tufts shown in Fig. 46 consist of capillary loops, which during pregnancy are distended with blood, as are the

corresponding capillaries of the placental crypts. At the close of pregnancy, when the naval cord ruptures, any residual blood in the chorionic tufts readily escapes, vastly decreasing the volume of the previously distended capillaries. When the separation of the fetus from the membranes interrupts the force previously holding the maternal and fetal placentae in contact, the latter falls away.

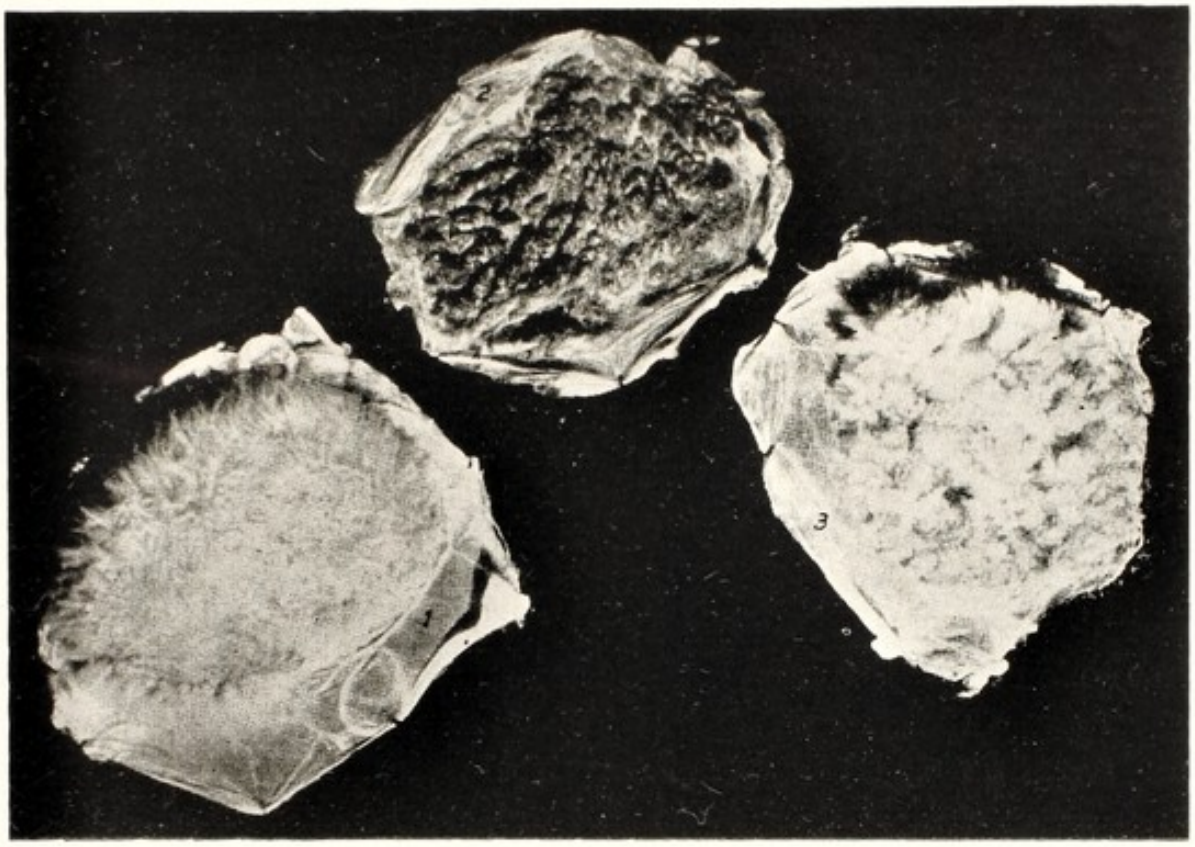


FIG. 172. Healthy and Diseased Cotyledons from Three Cows.

1, A cotyledon from a sexually healthy, highly fertile cow, showing densely packed, very fine placental villi; 2, a cotyledon from the ninth calf of a highly fertile cow; this one cotyledon has lost all its villi and is in advanced calcification; 3, a cotyledon from a uterine caruncle which had suffered severely in a prior pregnancy, causing the villi to be coarse and clumpy. *(Cornell Veterinarian)*

With the advent of disease, necrosis of the chorionic tufts frequently occurs. This, so far as known, is universally linked with necrosis of the placental epithelium within the crypts of the caruncles, into which the delicate tufts extend. At the beginning the necrosis is largely peripheral, and is shown, upon autopsy of the pregnant cow, as a girdle of dirty gray tufts, freed from the crypts. Clinically the necrosis is readily observed immediately after the close of pregnancy, by a study of the afterbirth. Thus in Fig. 37, the afterbirth shows clearly the profound placental necrosis which had led to abortion at 237 days. Once the placental necrosis has occurred, the afterbirths

of all future pregnancies authentically show the original injury unless the record has been obliterated by additional destruction in later pregnancies: a caruncle, although it may have been damaged in a given gestation, may still continue to function imperfectly, but the involved caruncle may again be invaded and the entire organ completely destroyed. The damaged caruncle, which continues to function, reveals the prior disease in the manner shown at 3, in Fig. 172. Here the necrosis, during a prior gestation, had destroyed a large proportion of the placental crypts, and the fetal placenta thereafter was characterized by sparse, coarse, clumpy chorionic tufts.

Once the placental epithelium of the crypts in the caruncle has been destroyed, all clinical evidence indicates that it cannot be renewed. At each succeeding parturition the chorion bears authentic proof that the damage has been permanent. The nutrition and health of the fetus are regimented by the extent of placental necrosis occurring in any and all prior gestations. Numerous other pathological phenomena are observed. In 2 of Fig. 172 extensive calcification is present, somewhat analogous to the equine placental calcification shown in Fig. 30. I saw in the abattoir a case in which all fetal and maternal placentae were so completely calcified that, had the cow been permitted to live to the close of gestation, the afterbirth could not have been thrown off or removed without all the uterine caruncles accompanying it. There is conflict of opinion regarding both the date and avenue of the invasion of the utero-chorionic space of pregnant ruminants. Accepting the conclusion of those who assert the frequent presence of bacteria or other invaders in the clinically healthy non-pregnant uterus, it is claimed that such invaders lead a clinically neutral life. Under the stress of pregnancy they may acquire definite pathogenic power.

Clinically, as has been indicated in preceding chapters, mares bred very early after the termination of pregnancy show a low ratio of conception, a high prevalence of abortion, prolonged gestation and a high ratio of non-viable foals. Cows re-bred early show a low ratio of identifiable pregnancies, a high incidence of abortion, a marked tendency to abbreviated duration of pregnancy, and a high prevalence of retained afterbirth. One observer may attribute the unfavorable result to the presence of disease-producing elements in the uterus at the date of conception; another may conclude that the early breeding places a dangerous burden upon the reproductive system and lowers its resistance to blood-borne infections. The decision is not vital. Each leads eventually into the same port—disaster. The avenue of invasion provides equally favorable ground for conflict of opinion.

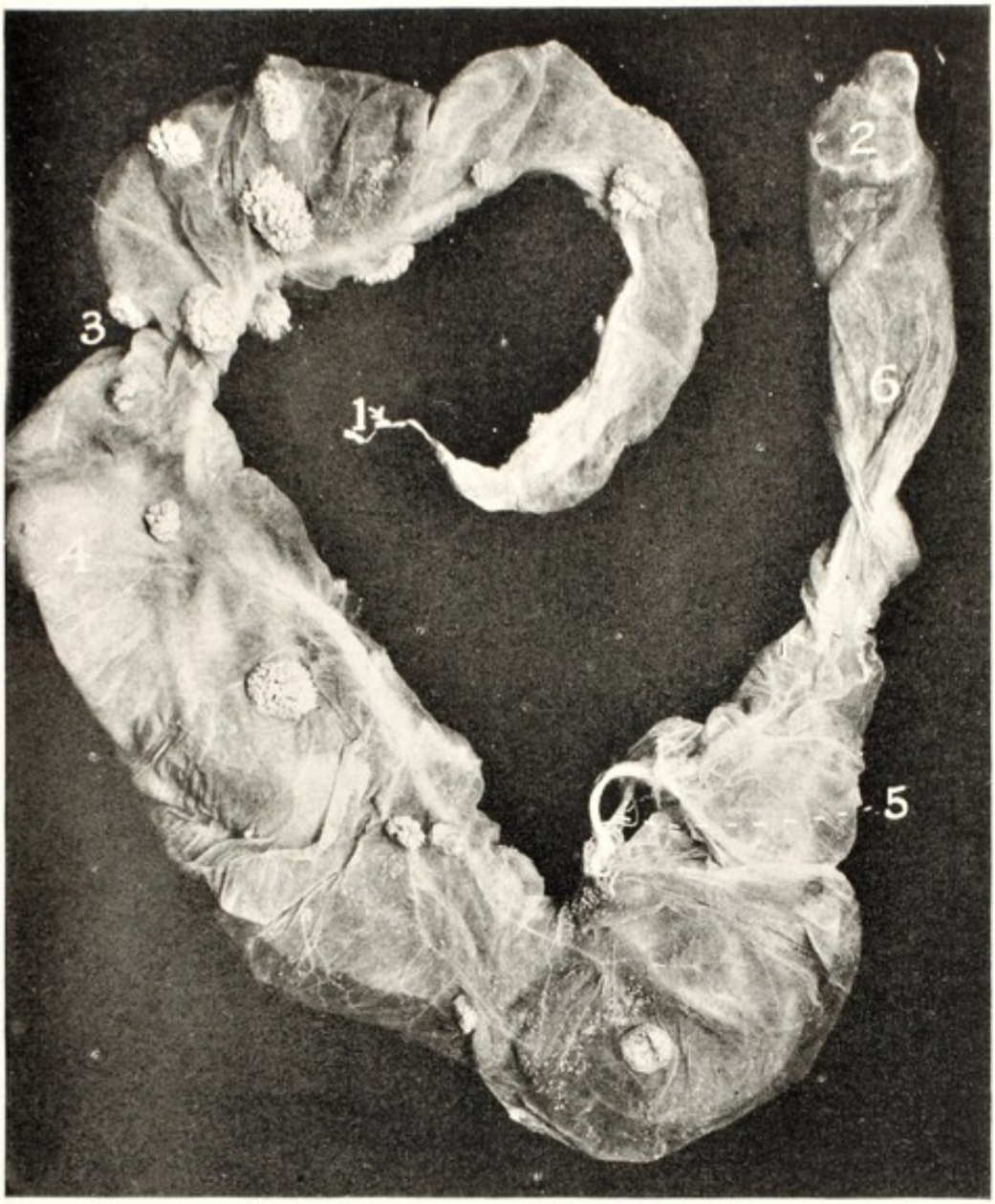


FIG. 173. Bovine Allantochorion from a Uterus in Which Most Caruncles Had Been Destroyed During or Following a Previous Pregnancy.

The cow was slaughtered three and one-half years after having aborted, presumably absolutely sterile, but found about three months pregnant.

1, Necrotic tip of gravid chorion; 2, necrotic area at apex of non-gravid horn; 3, constriction in fetal sac evidently due to partial atresia of the gravid uterine cornu, probably caused by metritis following abortion over three years previously; 4, main portion of gravid chorion enveloping the fetus; 5, the cervical axis dividing the right and left cornua; 6, the non-placental chorion of non-gravid cornu, showing by its thinness and transparency that the endometrium of the cornu had been destroyed prior to present pregnancy. *(Cornell Veterinarian)*

Clinically a study of Fig. 37 indicates with much emphasis that the infection, *Bacillus abortus*, entered the utero-chorionic space at the ovarian pole of the gravid cornu and travelled toward the cervix. The life of the fetus was maintained by the placentae of the non-gravid cornu up to the 237th day, when abortion ensued. The observation is

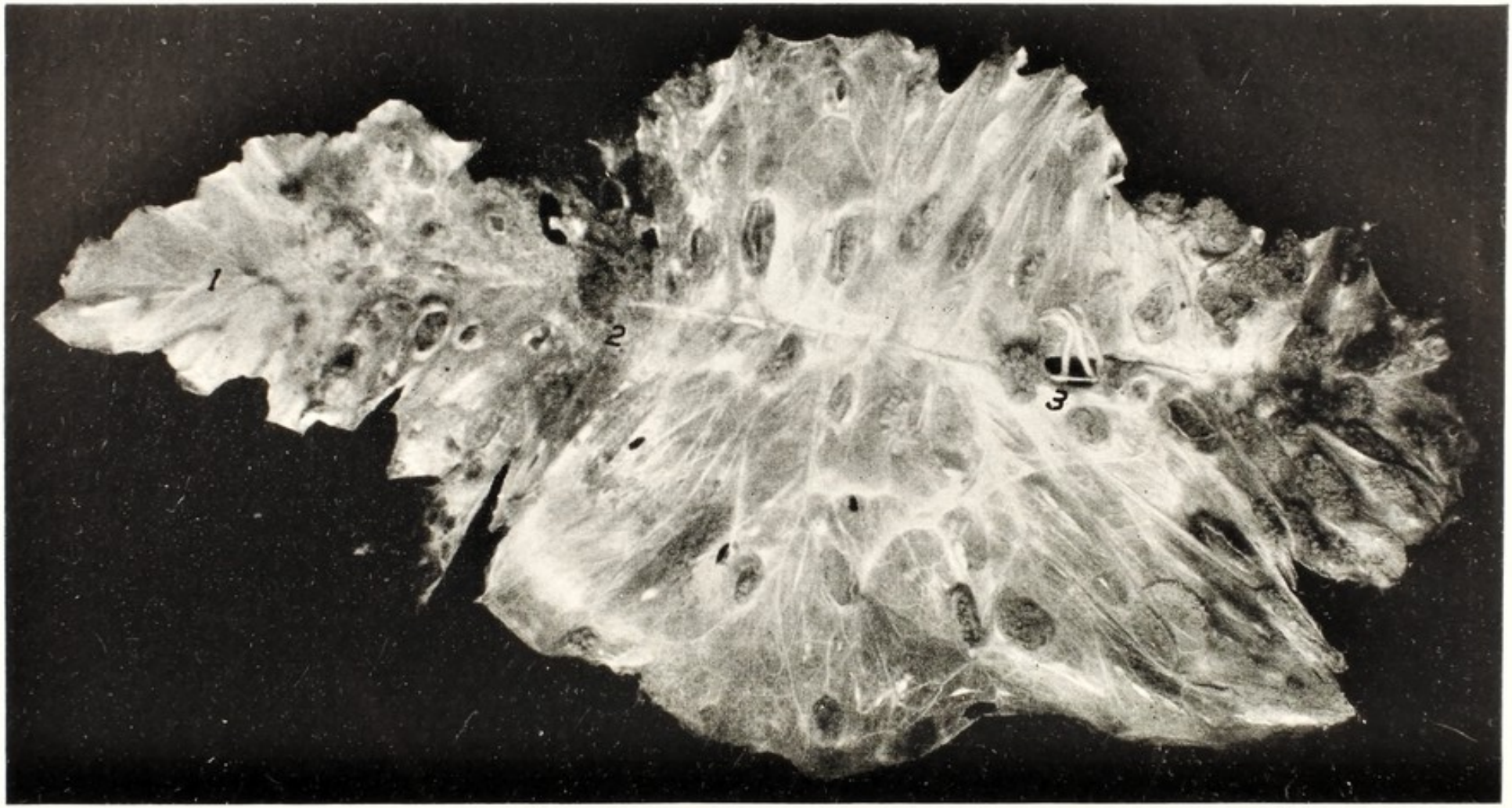


FIG. 174. Chorion Indicating Mild Cervical Endometritis.

1, Naked ovarian pole on non-gravid chorion indicating destruction of the uterine caruncles prior to last pregnancy; 2, cervical area much thickened and covered by hemorrhagic adventitious placental growths; 3, allantoic navel. *(Cornell Veterinarian)*

a rare one. Admittedly the infection may have been carried to the area after conception, via the blood-stream. After having calved twice, she was sterile and required repeated breedings for the pregnancy under study. This would suggest that disease existed within the uterus prior to the conception and may have killed other fertilized ova which disappeared unseen. Far more frequently the clinical evidences of the advancement of the disease point to the ovarian pole of the non-gravid horn as the original focus. Such are the indications in Colored Plate II and in Figures 97 and 169.

The recuperative strength of the animal may enable the cow to conceive after virtually all caruncles have been destroyed. Thus, in Fig. 173, the cow had aborted at 8 months and afterbirth was irremovably retained. The caruncles virtually all became necrotic. After repeated breeding for 3.5 years, she was slaughtered and unexpectedly found pregnant about 90 days. In a third group of cases the disease is apparently concentrated about the cervix, as shown in Fig. 174. In a prior pregnancy destructive disease had occurred at the overian pole of the current non-gravid cornu. The disease had largely recovered with the loss of uterine caruncles. In the pregnancy illustrated the non-functional cornu had remained comparatively free from active disease at its ovarian pole and the lesions of the current pregnancy were concentrated about the cervical area.

There is abundant evidence that, when a virulent infection having a marked affinity for the pregnant uterus and its contents, such as *Bacillus abortus,* is injected directly or indirectly into the blood-stream of a susceptible cow, its invasion of the utero-chorionic space is highly probable. The probability of its penetration may be enormously increased if the endometrium and chorion are already diseased.

ADVENTITIOUS PLACENTAE

Physiologically the bovine placentae are confined to the summits of the uterine caruncles. Pathologically adventitious placentae sometimes develop while the caruncles are comparatively healthy and the adventitious structures are not recognizably needed for the nutrition of the fetus. They seem to be the result of irritation of the intercaruncular mucosa. Generally they follow extensive destruction of the caruncles during a prior gestation, associated with severe metritis falling short of the actual destruction of the endometrium. If the intercaruncular endometrium be destroyed, no adventitious placentae can be formed and the chorion, if free from disease, is a thin, feebly vascular, transparent membrane like the amnion, as shown at 1, in

Fig. 175 and at 3, in Fig. 40. Sometimes the caruncles are all destroyed as in Fig. 176 and a large part of the endometrium develops frail, hemorrhagic placentae. Rarely, when almost all caruncles have been



FIG. 175. Membranes of a Fetus Aborted at 116 Days, Seen from Placental Side.

Cow had suffered from obstinate metritis and cervicitis following her first calving and failed to again become pregnant for fifteen months; she aborted at 116 days. 1, Amniotic cavity from which the fetal cadaver has been removed; 2, some apparently primary cotyledons surrounded by profuse adventitious placental formations; 3, naked apex of gravid horn; 4, approximate line between the two cornua. *The non-gravid chorion, from the cervix at 4, to the ovarian pole shows no recognizable primary cotyledons, indicating that all uterine caruncles in the area had been totally destroyed by the metritis following the first calving. The chorion is thickly covered with black, hemorrhagic adventitious placental growths; 6, the necrotic naked apex of the cornu, markedly flask-shaped, indicating that it had been invaginated into the area of the cornu beginning at, 5; 7, necrotic tip.* (Cornell Veterinarian)

destroyed, the secondary structures succeed in maintaining fetal nutrition and an apparently healthy calf is born after an abbreviated period of gestation. I have observed one such cow. Usually pregnancy in such a uterus ends in early abortion. In such cases as Fig. 40, the adventitious chorionic placentae are markedly hemorrhagic.

Human obstetrists describe *placenta praevia* as a serious abnormality. The placenta of woman is physiologically located at the fundus of the uterus. In some cases the placenta is, instead, situated near the cervix, and as pregnancy advances severe hemorrhages occur through the cervix. Finally when labor sets in the skill of the obstetrist is severely tested to save the lives of woman and child. According to J. Whitridge Williams[226] the incidence of placenta praevia increases with parity: the greater the number of pregnancies experienced, the higher the incidence of placenta praevia. He cites Doranth as showing an incidence of 0.17 per cent in primipara and 5.51 per cent when the number of births were between seven and ten. He also cites Strassman as observing an increase in incidence according to the rapidity of births. Human obstetrists are not positive regarding the cause of placenta praevia. The citations above suggest that in woman, as in the cow, placental structures abnormal in character and location occur and that the phenomenon may have been preceded by placentitis in prior pregnancy or by a heavy reproductive load. The inference is justifiable that in ruminants the abnormal location of the placentae is necessitated by injuries to the physiological placental areas and that the adventitious placentae are substitute structures.

The securely barred cervical canal of the cow, Fig. 49, defies the external escape of blood during pregnancy. The hemorrhage, however, is copious, as shown in Fig. 40. According to some obstetrists, the hemorrhage in woman is caused by placental dehiscence; in the cow the inter-placental hemorrhage apparently causes the separation. Placental hemorrhage is a common accompaniment of adventitious placental structures, and is not known to require any abnormal uterine contractions. The hemorrhagic condition shown in Fig. 40, was already present when the supposedly sterile cow was slaughtered at 90 days.

Profuse Caruncular Hemorrhage in the Cow

BOVINE HEMATIC MUMMIFICATION

The cow is the outstanding, if not the sole domestic animal exhibiting a marked tendency to non-traumatic hemorrhages of the reproductive system. It has been noted in Chapter XI that within three or four days after ovulation she often has a well defined menstrual flow. In the abattoir, when the ovisac has just ruptured, abundant droplets of blood are seen upon the placental areas of the uterine caruncles. It was further observed that occasionally there is recognized, by rectal palpation, copious hemorrhage from the crater

of the freshly ruptured ovisac, although no visible vessels are ruptured at ovulation. It is recorded in Chapter XII, and illustrated in Fig. 42, that fatal hemorrhage sometimes follows dislodgment of the corpus luteum. Closely associated with placentitis is interplacental hemorrhage involving one or many cotyledons, as shown in Fig. 176 from a heifer aborting at term. Sometimes there is a very diffuse

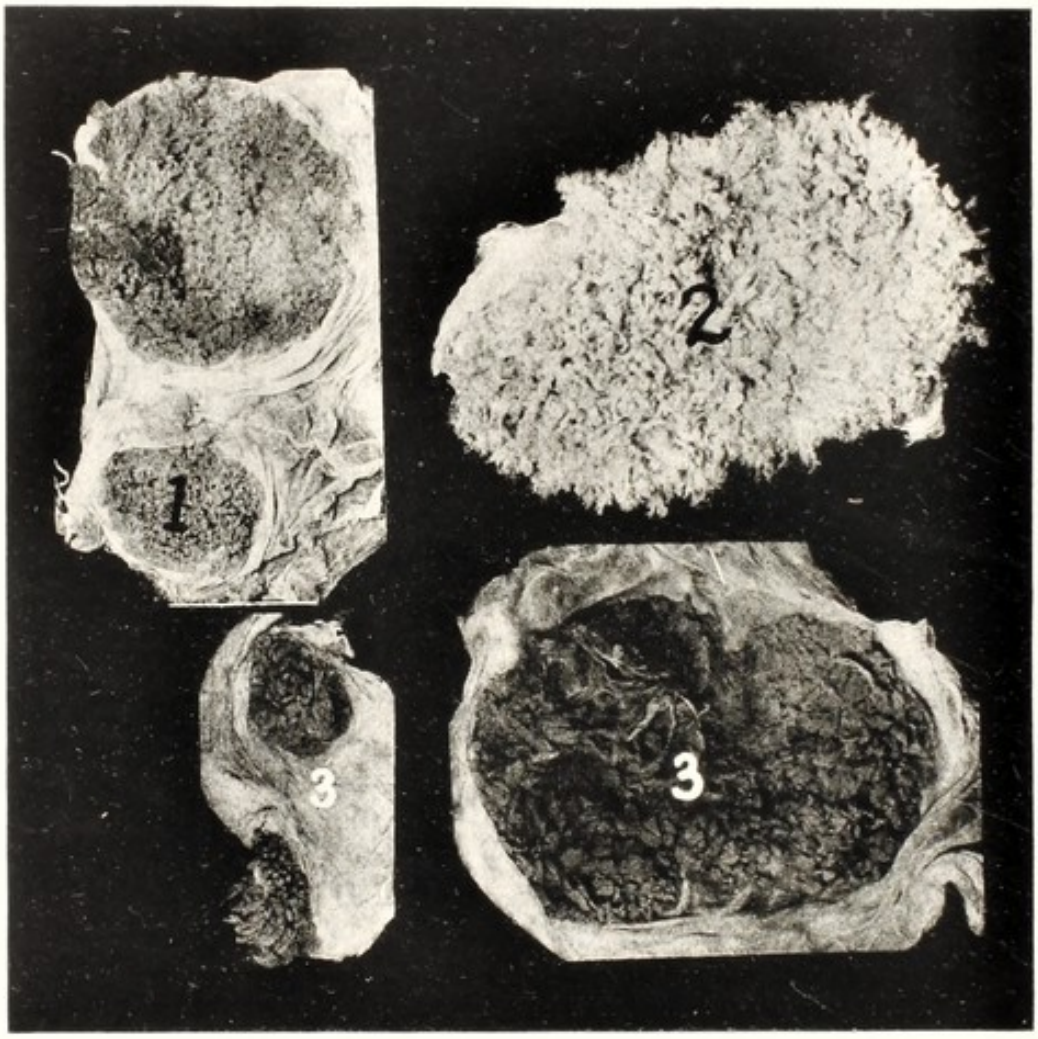


FIG. 176. Dystocia and Dead Fetus at Term in Two-year-old Heifer, Showing Inter-placental Hemorrhage.

Cotyledons in cervical area show old hemorrhages and necroses; the apex of the gravid horn had been invaginated. 1, Arrested placental growth; 2, a necrotic cotyledon; 3, 3, hemorrhagic cotyledons. *(Cornell Veterinarian)*

placental hemorrhage in the adventitious placental structures in cases where all or nearly all caruncles have been destroyed, as shown in Fig. 175. The Bang-negative cow, after a long period of sterility, conceived, but aborted at 116 days.

There frequently occurs in cows a specific type of fetal mummification not known in other species of domestic animals. Veterinary writers generally have not set it apart as a distinct phenomenon, but

group it with wholly different types of fetal desiccation observed in swine and in one of twins in the mare and the cow. It has been designated *lithopedion* or *steinkalb* by many writers. This has led some human obstetrists to assume that bovine fetuses frequently become calcified.

The hematic mummy is the direct consequence of a profuse interplacental hemorrhage involving all placentae and pushing apart the maternal and fetal placental structures, as indicated in Figs. 177 to 180. It is common in heifers. It is not known that the hemorrhage

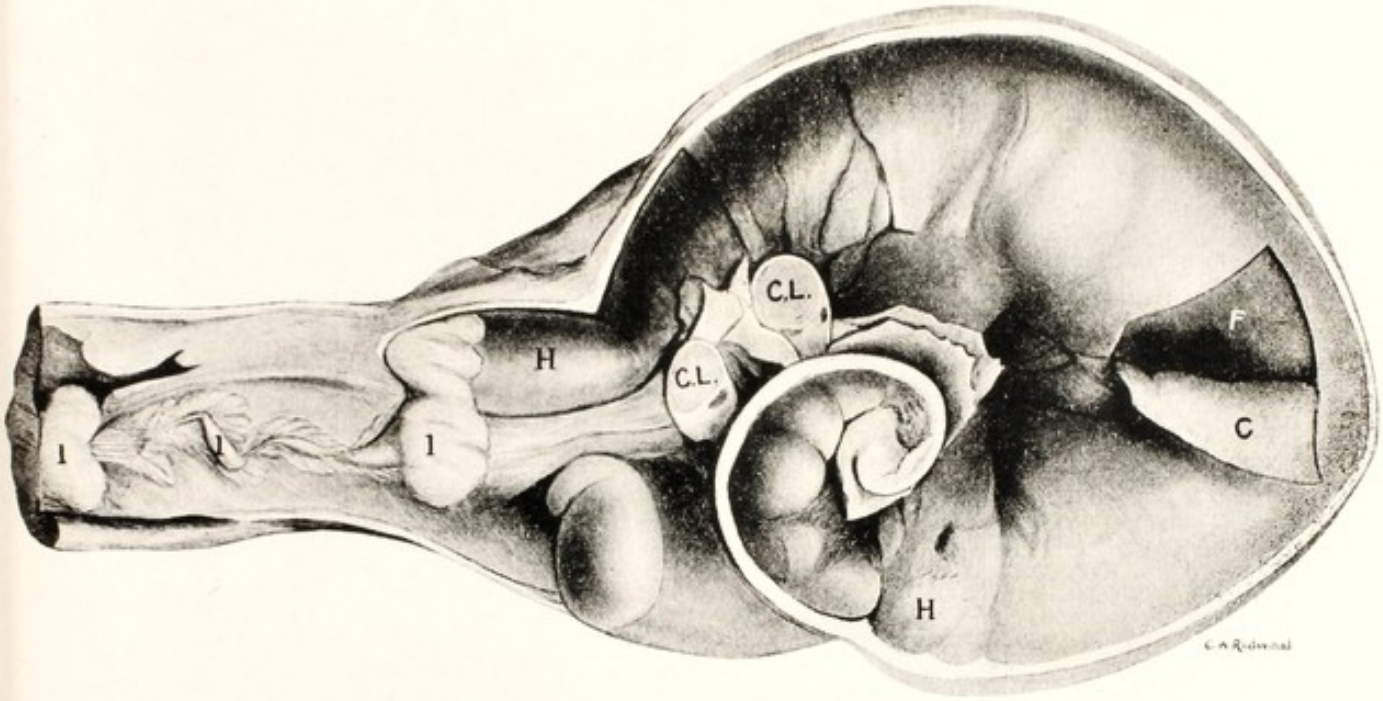


FIG. 177. Mummified Fetus (Hematic Mummy) in Situ in Uterine Cavity.

The interplacental blood coagulum has not yet become desiccated, and the fetus, with its membranes, is surrounded by a thick layer of blood wholly separating it from the uterine placenta. I, Cervical canal; *H, H,* hematoma distending the uterus and covering the fetal sac; *C,* chorion incised and turned back to expose the fetus, *F; CL,* the sectioned corpus luteum of pregnancy.

ever proves fatal. Perhaps the lowered maternal blood-pressure, with the increased intra-uterine tension, serves to terminate the hemorrhage.

In the closely analogous puerperal intra-uterine hemorrhage, to be described later, the milk of the cow has a highly characteristic red color, attributed to the breaking down of the red blood cells in the process of desiccation and their excretion by the mammary glands. It seems highly probable, when interplacental hemorrhage occurs in a pregnant lactating cow, that her milk will attain the same scarlet color indicating the beginning of fetal mummification. It occurs, at least

generally, in apparently healthy cows or heifers and occasions no signs of disease or disturbance. When clinically diagnosed, it is usually discovered by rectal palpation after the supposedly pregnant female has gone over time without signs of approaching calving. Sometimes the mummy is expelled after months or years; sometimes the condition is discovered at slaughter.

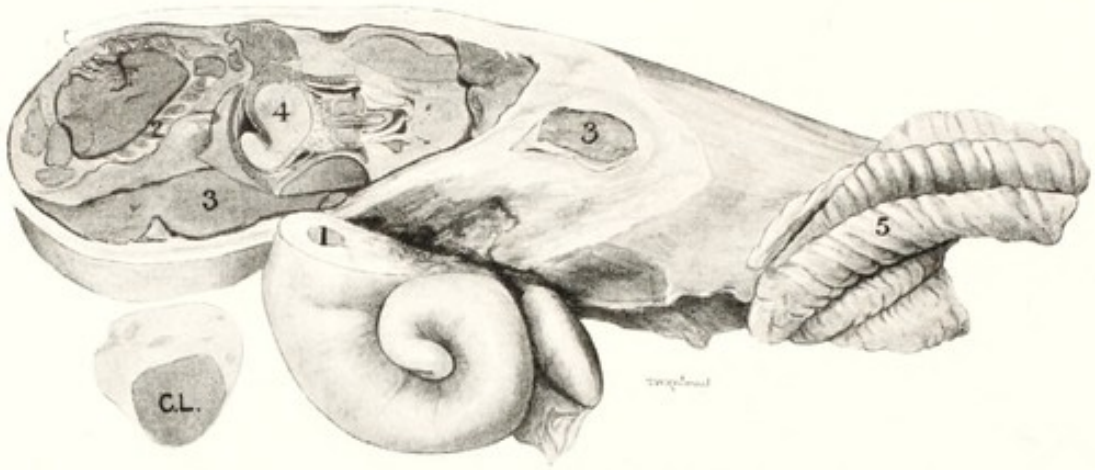


FIG. 178. Sagittal Section Through Bovine Uterus Containing a Fetus in the Early Stage of Mummification.

1, Portion of the hematoma extending into the non-pregnant horn; 2, transverse section through the fetal ribs; 3, 3, hematoma in the pregnant horn; 4, cranial bone; 5, vagina; *CL*, embedded corpus luteum.



FIG. 179. Completed Mummification of Bovine Fetus.

In company with W. W. Williams, I have had the opportunity to study the phenomenon early after the hemorrhage had occurred and to follow it through to the establishment of definite fetal mummification. The observations were verified by a series of cases personally studied in the abbatoir, as shown in Figs. 177 to 180. Clinically, at the outset, there is easily recognizable by rectal palpation a doughy

mass distending the uterus. Too firm for the fetal liquids of pregnancy, it consists of a vast, soft blood clot completely enveloping the ovum (fetus and fetal membranes). The fetus is not directly palpable but may be obscurely felt through the coagulum. The clot, even in

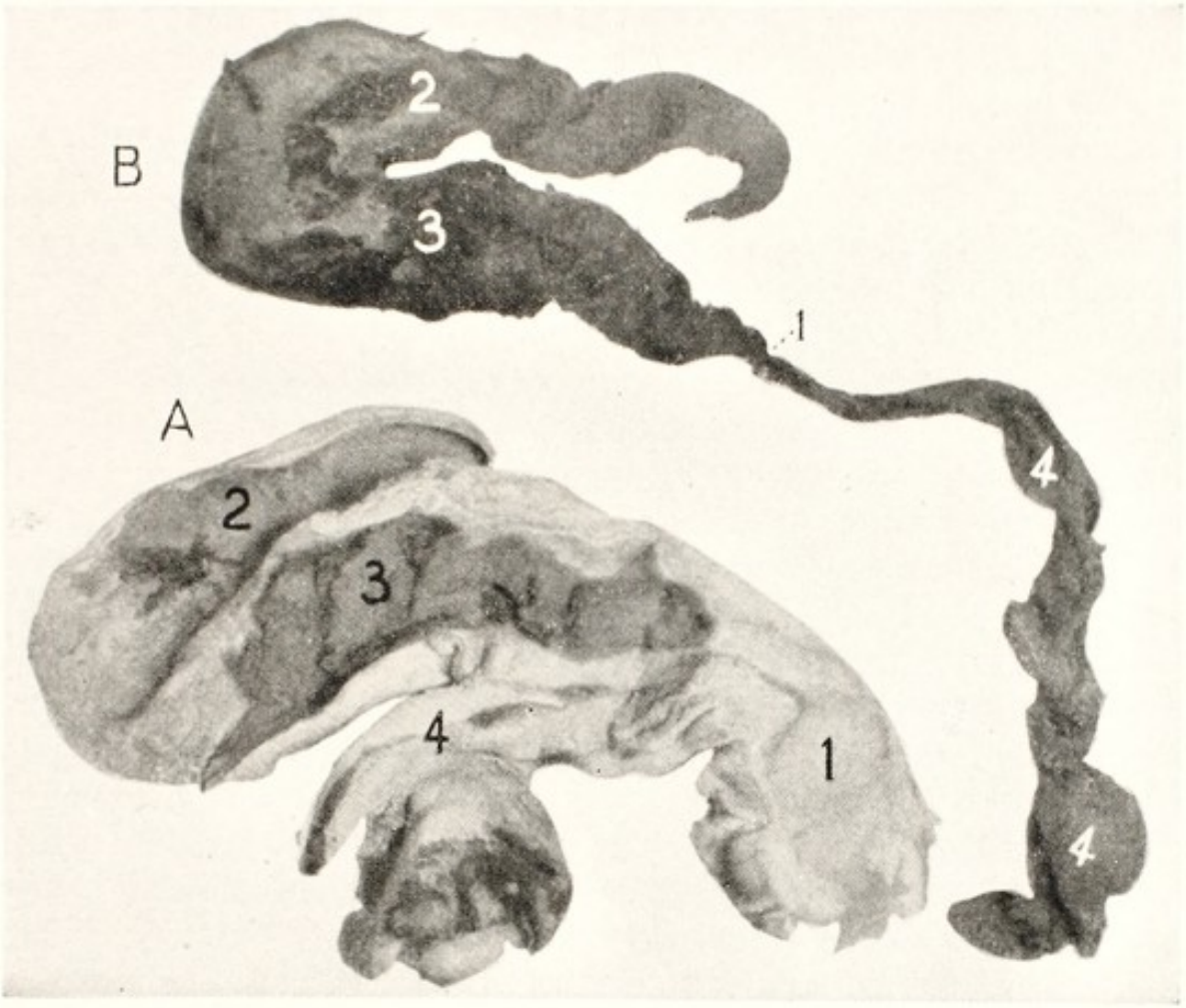


FIG. 180. Bent Mummified Fetus in the Uterus of a Heifer.

The fetus had been dead seventeen or eighteen months when the heifer was slaughtered. A, The uterus from which the fetus has been removed. 1, The cervical canal with seal; 2, the ovarian pole of the gravid (right) cornu; 3, the base of the gravid cornu; 4, the left cornu. B, The bent mummy in its sac. 1, The cervical area of chorion; 2, the portion of the mummy occupying the ovarian pole of the bent uterus; 3, the portion of the mummy from the base; 4, 4, the non-gravid chorion. *(Cornell Veterinarian)*

contour, conforms to the normal outline of the pregnant organ, as indicated in Fig. 178. The blood coagulum promptly begins to desiccate. Necessarily the allantoic and amniotic fluids, as well as the liquid portions of the fetus, must be resorbed at virtually the same period. The size of the intra-uterine mass, rapidly decreasing and becoming more firm to the touch, within a few weeks is like Fig. 178. The duration of time required for complete mummification has not been au-

thentically determined, but it is apparently between three and six months, and the cadaver then remains indefinitely as an inert foreign body, like Fig. 179. The blood cells of the coagulum have been broken down, their water resorbed and much of the hemoglobin removed. Some of the coloring matter remains and the mummy and its membranes retain the dark stain indefinitely, as shown in Fig. 179. The uterine seal remains intact. The cow appears well and does not come in estrum.

It has been stated that, when a fetus dies and air is admitted to the uterus, the cadaver macerates; if air is not admitted, it mummifies. Neither is correct. Harms[80] correctly states that, in the bitch and sow, macerating, emphysematous fetuses repeatedly occur located between two healthy ones. I have repeatedly observed, as will be describd later, acute septic metritis in the cow with rapid emphysema of the fetus, the uterus closed and sealed. Emphysema of the fetus is common in torsion of the uterus in all species, although the cervical canal is closed so tightly that the vagina or uterus may rupture transversely. Neither does an open cervical canal necessarily cause fetal emphysema. The peculiar phenomenon to be later described, which I have provisionally designated static fetal cadaver, may exist without emphysema or mummification. In that type of puerperal uterine hemorrhage in the cow illustrated in Fig. 188, the hemorrhage is seemingly identical in character with the caruncular hemorrhage of pregnancy. The blood is poured out into a wide-open uterus from which a calf has been recently expelled. The uterine cavity is usually, if not always polluted. The blood coagulates, desiccates (mummifies) and, like the mummified fetus, remains indefinitely as an inert body.

The forces which prevent putrefaction and emphysema of the blood clot and fetal cadaver are unknown. Rarely the corpus luteum of pregnancy atrophies, an ovisac ripens, estrum follows and the cadaver is expelled.

The cause of the caruncular hemorrhage is unknown. Kitt[107] states that the mummification—and therefore the caruncular hemorrhage—is due to invasion by *Bacillus abortus*. Hutyra and Marek[98] also attribute the hematic bovine mummy to *Bacillus abortus* invasion. The phenomena attributed to the abortion bacillus are illimitable, and at times amazingly contradictory. It is commonly alleged that *Bacillus abortus* is the chief cause of retained placenta in the cow. In direct conflict with this view, high authorities declare that the invasion by this same bacterium causes the most spectacular and abrupt detachment of the placenta known to veterinary science. Presumably the fetus is alive and free from marked lesions when the caruncular

hemorrhage abruptly sets in and destroys the young. Why this bacillus should cause retained placenta in one cow and abrupt dehiscence of the placenta of the intra-uterine fetus of another has not been explained.

Franck[64] apparently studied in the laboratory the mummy without the uterus, and concluded from the wrinkled exterior of the cadaver that it had been due to uterine torsion. Boyd,[18] also attributing hematic mummification to uterine torsion, advises the dislodgment of the corpus luteum as "the most satisfactory and by far the safest form of treatment." Just how the dislodgment of the corpus luteum would eliminate the uterine torsion, Boyd fails to explain.

Specific means of prevention are unknown. Since it occurs almost exclusively in dairy cattle, the inference arises that it may be avoided by the application of the general rules of breeding hygiene.

The prevailing treatment is the dislodgment of the corpus luteum. Since the corpus is more or less embedded, it may be necessary or advisable to incise the yellow body, with the ovarian scalpel, Fig. 43. Some have advised cesarean section and others the dilation of the cervix.

The economic problem of the handling of fetal mummification merits consideration. If the condition is recognized early, before the desiccation has been completed, and the corpus luteum is dislodged, the expulsion of the fetus will probably cause no dangerous laceration of the cervix or vagina. If the mummification occurs in early gestation, resulting in a small cadaver, and its expulsion is brought about by the dislodgment of the yellow body, important injury to the birth canal is improbable. Slatter[181] records two cases in which the mummies were eleven and eighteen inches respectively, after which each again conceived, calved apparently normally and was pregnant a second time. In a third case a very small mummy was expelled spontaneously and the cow later became pregnant. No data are available covering a large number of consecutive cases.

When the mummy is large, badly distorted and extremely hard like that shown in Fig. 179, the advisability of attempting to cause its expulsion, or its removal by cesarean section, as some might suggest, is quite debatable. Lacerations of the cervix of a type to interfere with future reproduction are almost unaviodable. The cesarean section is far more spectacular than economic. Yet less appealing is the forcible dilation of the cervical canal. The dilation will probably seriously injure the cervix, and the passage of the mummy through the canal will extend the lacerations. Such cases as that shown in Fig. 180, although small in volume, present further complications in

expulsion or extraction owing to the bent condition of the cadaver. In this instance the heifer was not worth the cost of handling, and was therefore slaughtered.

The presence of the mummy does not detract from the value of the carcass of the cow for beef. The decision of the course to pursue should be based upon the potential breeding value of the cow, the cost of handling, the delay until she may be safely bred and the salvage value of the animal for beef. If handling be decided upon as justifiable, the corpus luteum should be dislodged. If the corpus lueteum is so deeply embedded that it cannot be safely removed by compression, the capsule of the ovary should be incised with the ovarian scalpel, Fig. 43, and the dislodgment completed by compression. The cow should be closely watched after a day or two, and examined upon the first signs of labor, and all available precautions taken to avoid lacerations and contusions. The passages should be kept well lubricated, projecting limbs amputated or broken, and other precautions taken as conditions may demand.

UTERINE SAND

There commonly exists in the utero-chorionic space of cows in advanced pregnancy numerous minute bodies which I have designated *uterine sand*. These are free, spheroidal masses, 1 or 2 mm. in diameter, translucent and of a pale amber color. They are very hard, and are insoluble in water and alcohol. They vary greatly in numbers and volume. By detaching the fetal membranes from the uterus, washing both surfaces in a vessel of water, and then decanting it off, there remains behind a mass of *uterine sand* sometimes as great as one ounce, feeling much like sand when grasped between the fingers. Its exact character and origin are unknown. It appears most probably due to the disintegration of red blood cells arising from small placental, or endometrial hemorrhages.

EDEMA OF THE ALLANTOCHORION

Edema of the fetal membranes is a common phenomenon in bovine ova invaded by various bacteria. Moderate edema of the amnion, allantochorion and umbilical cord is recognizable in most cases of abortion and serious utero-fetal disease associated with bacteria. I have recorded an extreme case, in *Veterinary Obstetrics*. A cow in second pregnancy appeared well up to about 225 days, when her appetite failed and expulsive efforts appeared. It was only with difficulty that she could arise. At the autopsy, in company with Dr. Cushing, I found the uterus and chorion ruptured, the allantocho-

rion about five inches thick, and the fetus highly anasarcous. The peritoneal cavity of the cow contained an estimated 400 pounds of thin, reddish fluid. The source of the entire volume of fluid was not accurately determinable. The cow may have had some degree of ascites. Much of it was doubtless from the amniotic and allantoic sacs, and an unknown quantity from the ruptured allantochorion itself. The detached allantochorion, with the liquid caught while detaching it, weighed 88.75 pounds. The estimated weight of the uterine contents with fluid free in the peritoneal cavity was 558.75 pounds.

The non-gravid cornu was devoid of caruncles, like the uterus shown in Fig. 182. They had evidently been destroyed during her first puerperal period, presumably owing to retained afterbirth. The disease had passed unnoted or had been forgotten.

Dropsy of the Fetal Membranes

AMNIO-ALLANTOIC DROPSY. HYDRAMNIOS. HYDRALLANTOIS. POLYHYDRAMNIOS

The ova of all species of domestic animals have an amniotic cavity lined with ectoderm and filled with amniotic fluid, and an allantoic cavity lined with endoderm and filled with allantoic fluid. The two cavities are in communication through the urethra, urinary bladder, and urachus. The origin and function of the two fluids have not been satisfactorily determined. Zeitzschmann[278] gives the normal volume of amniotic fluid in the cow as 1.1 to 4 liters and of the allantoic fluid as 3.5 to 12 liters. No precise line of demarcation between the normal and pathological volume of the fetal fluids can be drawn. When the volume of the fluids becomes notably abnormal, and definitely affects the size of the abdomen, interferes with the general well-being of the pregnant animal, causes dystocia, etc., it becomes clearly recognizable as disease. Pathologically the volume may reach 50 gallons (200 liters or 400 pounds) in the cow. At about this point the increase is stalled because the cow can no longer rise, the essential body functions are vitally disturbed and the patient dies.

In the absence of definite knowledge of the origin and physiological functions of the fetal fluids, the etiology of dropsy of the amnion and allantois is an unsolved riddle. As in many other diseases, there are numerous hypotheses, each of which is disturbed by various clinical facts.

It is recorded in Section I, that in the hybridization of the American bison bull with the domestic cow severe amnio-allantoic dropsy

regularly follows and leads to a high ratio of death of both cows and young. Naturally the hypothesis presents itself that the hybridization is the cause. On the other hand, the hybridization of the male ass with the domestic mare is not accompanied by any notable amnio-allantoic dropsy.

In the same section mention is made of a destructive prevalence of amnio-allantoic dropsy in connection with bulldog calves among Dexter, Tux-Zillertaler, and other cattle. These are not hybrids as generally understood, but are crosses between two dissimilar breeds of cattle, apparently followed by inbreeding, in order to fix the type. It is further related that by intense inbreeding of both cattle and sheep, many non-viable *muscle contracture monsters* are produced, associated with high mortality of the pregnant females owing largely to the severe amnio-allantoic dropsy. It is next stated that the inbreeding of horses sometimes leads to atresia of the colon, hydrocephalus, and cerebral glioma, without, so far as known, inducing amnio-aliantoic dropsy. Hence it appears that some types of hybridization and of inbreeding lead largely to destructive amnio-allantoic dropsy, while other forms of hybridization and inbreeding show no such tendency.

Writers upon human obstetrics state that twin pregnancy is a fruitful cause of dropsy of the amnion (the human ovum has no allantoic cavity) and with this veterinary writers agree by recording a notable prevalence of the disease during twin pregnancy in the cow. But the not infrequent twin pregnancy in the mare is rarely associated with amnio-allantoic dropsy. Fleming,[62] citing Cartwright, mentions one such case. My first twelve years in the profession were in a highly concentrated horse-breeding district where dystocia was the major obstetrical problem, but I saw no case of dropsy of the fetal membranes. Though my later experience with horse breeding has been considerable, my sixty-four years in the profession have failed to reveal a case in the mare. Nevertheless, the mare is subjected to all the hypothetic causes enumerated: in one way she is far more exposed. The hypothesis is advanced by some veterinary and medical writers, that torsion of the navel cord, by interfering with the circulation, causes amnio-allantoic dropsy. Yet the disease is infinitely more prevalent in the cow, where the cord is short and rarely twisted, than in the mare with the long and abundantly twisted navel cord. Human and veterinary obstetrists believe that in some cases the hydrops is caused by diseases of the fetus or of certain fetal organs. There is room in some cases for the suspicion that the cause lies in a diseased uterus. Thus Fincher, Frost, and Williams[55] record two cases

of extreme hydrops which, like the case of edema of the chorion cited above, occurred in cows in which the uterine caruncles had been largely destroyed as the result of a prior pregnancy which left the uterus incapable of proper nurture of young. This invites fetal disease. One of the two cases yielded at slaughter forty gallons (320 lbs.) of dropsical fluid, the other fifty gallons (400 lbs.). In the latter

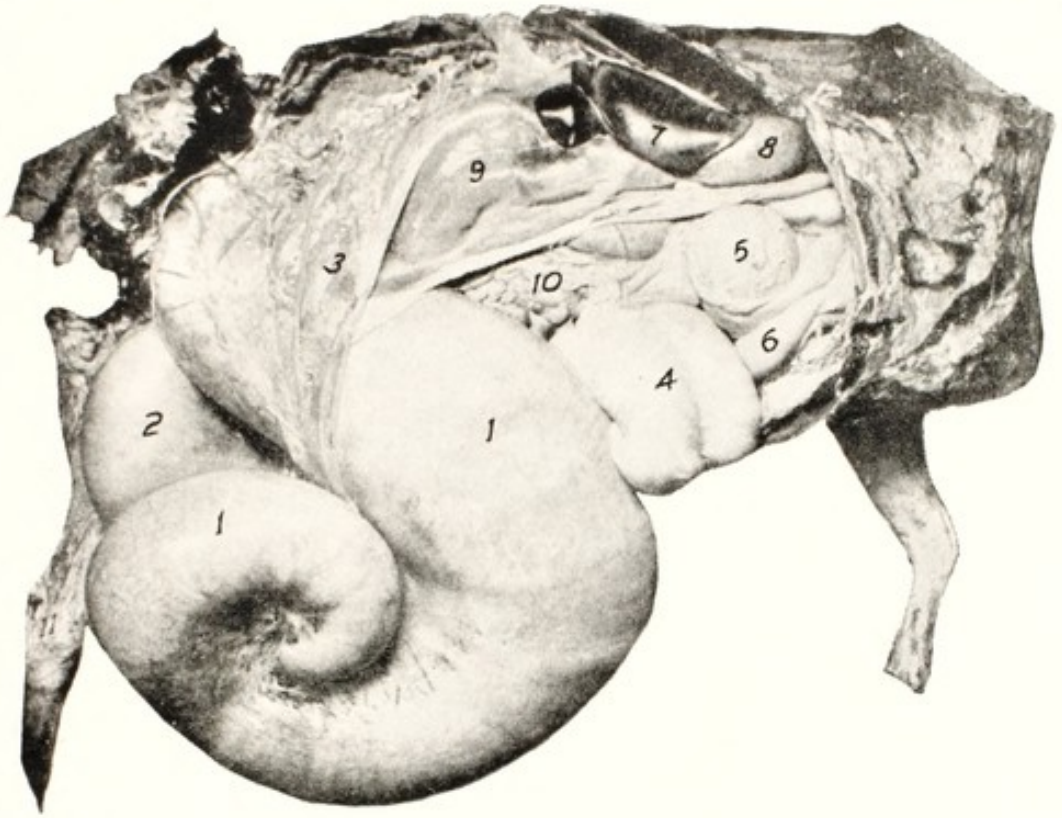


FIG. 181. Amnio-Allantoic Dropsy in the Cow; Sagittal Section of the Body of Affected Animal.

1, 1, The right (gravid) cornu, 12.5 feet long; 2, ovarian pole of non-gravid cornu; 3, the broad ligament of the uterus; 4, the empty rumen, displaced far forward by the uterus; 5, omasum; 6, abomasum; 7, liver; 8, gall bladder; 9, large intestine; 10, small intestine; 11, left tarsus; 12, sixth rib. *(Cornell Veterinarian)*

case, as shown in Fig. 181, the distended uterus represents almost one-half the total volume and weight of the pregnant cow. As shown in Fig. 182, the uterine caruncles had been destroyed in the non-gravid cornu prior to the current pregnancy. The destruction of most of the caruncles, with unavoidable serious damage to those which remained, invites fetal disease because of deficient nutrient supply.

Studies concerning the origin and function of the fetal fluids, and the etiology of excessive amounts or pathological composition of the fluids provide opportunity for much speculation. It is claimed by some that the fetal kidneys are non-functional or become functional only in a moderate degree in fetal life, while others deny this. Human

obstetrists record cases in which the urinary bladder becomes so enormously distended that the expulsion of the fetus is impossible. Apparently this is due to atresia of the urethra, and indicates a copious secretion of urine. In domestic animals the urinary bladder may optionally discharge its contents into the amniotic sac through the

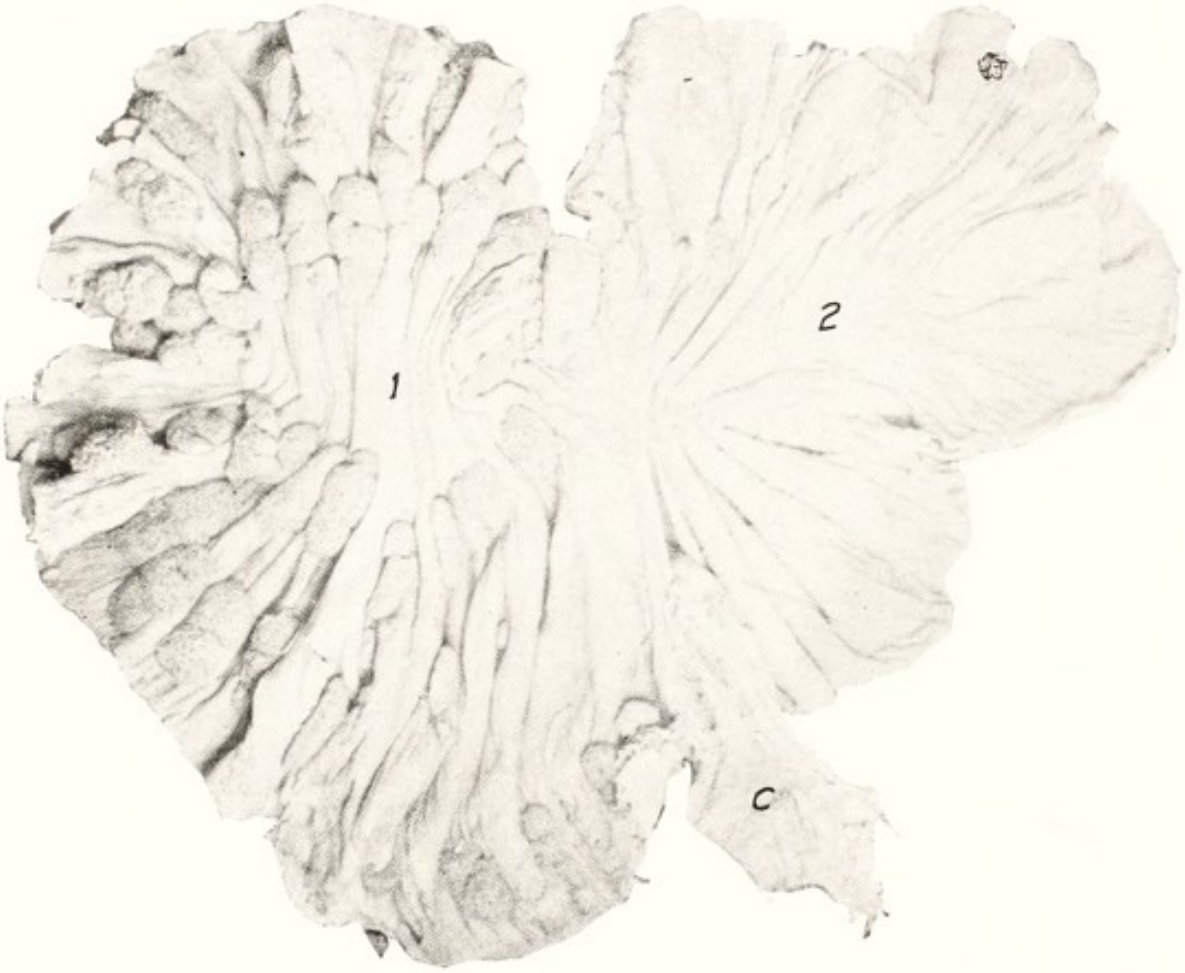


FIG. 182. Opened Uterus from Fig. 181, Showing Destruction of Uterine Caruncles in the Previous Pregnancy.

1, The gravid (right) cornu with numerous, near-normal caruncles; 2, the nongravid cornu from which all caruncles had disappeared before the pregnancy under study; *c*, cervix. *(Cornell Veterinarian)*

urethra or into the allantois sac through the urachus. As a consequence, distension of the urinary bladder rarely occurs. Harms[50] cites one case each in the mare and cow. These cases suffice to indicate that the fetal kidneys function. Very little urea is found in the fluids. That is to be logically expected, but the manner of its elimination is unknown. The kidneys of the fetus in Fig. 181 were three times the normal size. They probably secreted an abnormal amount of urine of unusual quality. There is scant reason to believe that the kidneys secreted the 400 pounds of fluid present. Most

writers believe it largely emanates, whether physiologically or pathologically, from the epithelium of the allantoic walls.

The possible sources of the amniotic fluid are less restricted. When the meconium of a calf or foal is suspended in water and the liquid decanted off there remain great quantities of hairs, not merely the typical lanugo of the human and carnivorous fetuses, but full-grown hairs, from which one may readily and authentically determine the color of the young. This indicates clearly that the skin contributes generously to the volume of the amniotic fluid. The liquid secretions from the skin, the shed hairs and other substances are promptly swallowed. Physiologically they do not pass through the alimentary tract. The liquid portions are rapidly resorbed and the solids remain stored in the intestine as a part of the meconium. In amniotic dropsy, the amniotic fluid is usually mucoid, quite unlike the normal fluid. This suggests that the mucus has come from the skin. Some of it may be from the mouth. This leads to the further inference that amniotic dropsy may be due more to the failure of the fetus to swallow the fluid and filter out the solids than to an excessive formation of it. The suggestion is apparently supported by a study of the monster, *Schistosoma reflexus* (Gurlt). This monster, confined, so far as known, to ruminants and swine, is formed early and consists virtually of the dorsal, instead of the physiological *ventral* folding of the somatopleure. The embryonic skeleton is everted while the thoracoabdominal viscera are exterior, attached along the mid-dorsal line to the parietal pleura and peritoneum. Writers upon veterinary obstetrics state that the monster is commonly associated with dropsy of the fetal membranes. Evidently the swallowing of the amniotic fluid is rendered difficult. I have described three highly interesting cases[261] in which the swallowing or digestion of the fluid was profoundly affected. In one of these cases, the reflection was so complete that there had apparently been almost no amnion. The cutaneous, reflected sac had an opening only 4 or 5 inches in diameter, which was virtually barred by the compression of the deformed feet and limbs. In this sac there had accumulated about ten gallons of viscid, mucoid fluid, which rendered expulsion of the fetus impossible until the cutaneous sac had been incised and the fluid permitted to escape. The conclusion seemed warranted that the pathological volume and type of fluid had come from the skin of the fetus and that the deformity prevented swallowing.

In another of the three cases, the rumen contained an estimated ten gallons of fluid identical in character with that of the preceding case, the fetus had swallowed the fluid as far as the rumen, where

it became stalled and remained undigested. The third case also had distension of the rumen, but in a lesser degree.

The additional observation was made that, owing to the early reversal of the somatopleure, prior to the formation of the allantois, the anlagen for the liver were removed from the physiological path of the umbilical vein, and consequently the liver was deprived of its normal blood supply. The liver consisted chiefly of large cysts, with apparently almost no hepatic tissue. It would seem extremely difficult for proper digestion of the amniotic fluid to occur in the absence of bile. In harmony with this, both human and veterinary obstetrists mention hepatic disease or defects as probable causes of the dropsy.

The abnormal fluids necessarily emanate from the maternal blood. In the most acute cases the transfer of the water from the vascular system of the dam is amazingly rapid. Early in my practice I attended a cow which could not get up because of the immense load of fetal fluids. I passed a trocar into the sac through the right flank and allowed an estimated forty gallons of fluid to escape. She then got up readily and moved freely. She drank great volumes of water, the fetal sacs quickly refilled, and in two or three days the volume of hydrops was as great as ever, the cow was unable to rise, and succumbed.

The disease is usually observed in advanced gestation. It is associated with a varying rate and degree of abdominal enlargement. This may cause it to be confused with twin pregnancy. Upon rectal palpation the uterus is found greatly distended and somewhat atonic. The fetus may drop far forward and be impalpable in the great mass of fluid. In the cow, the uterine caruncles are palpable. This, with the presence of the enlarged uterine arteries of pregnancy, aids in differentiation from the pyometra of trichomoniasis.

When the accumulation of fetal fluids approaches or exceeds 200 pounds in the cow, the patient loses flesh, but the increase of the fetal fluids may maintain or increase the gross weight of the animal. The appetite decreases; and, as indicated in Fig. 181, the rumen and the alimentary tract generally are empty. Presumably the weight of the abnormal fluid, and the entrance of toxic material into the maternal blood stream, combine to lower the vigor of the dam, so that finally she is unable to rise when down.

The prognosis varies with the intensity. Quite uniformly the outlook for the fetus is very poor and the mortality of the cows is high. When diagnosed early and the fetus extracted, some cows recover sufficiently to breed again. The emptying of the uterus is considered in the companion volume, *Veterinary Obstetrics*.

FETAL ANASARCA

Closely allied to amnio-allantoic dropsy, and not rarely concurrent with it, is fetal anasarca. The abnormal deposit of fluid is largely in the subcutaneous connective tissue. It is most common in cattle, and is observed in other ruminants. It is very rarely seen in the mare. The degree of anasarca may be very extreme, as shown in Fig. 183,

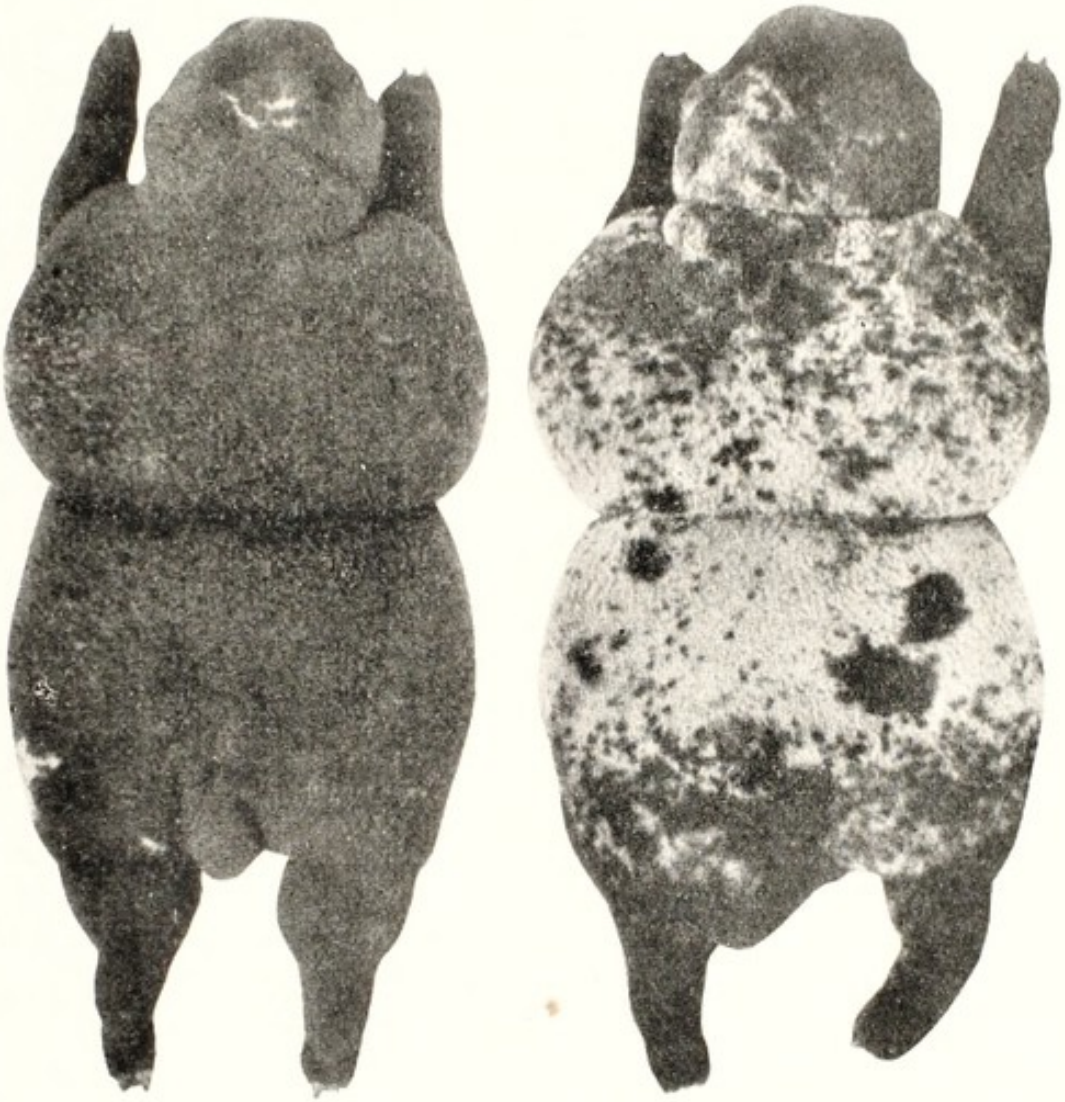


FIG. 183. Fetal Anasarca of Twin Lambs With Total Weight of 58 Pounds.

the twin fetuses being fully thrice the normal volume. Moderate fetal anasarca is not rare in cases of amnio-allantoic dropsy. Occasionally also there is present ascites or hydrothorax. The anasarcous fetus usually perishes before term.

TORSION OF THE UMBILICAL CORD

The tubular uterus of domestic mammals renders the revolution of the fetus upon its transverse axis essentially impossible in advanced pregnancy. Probably the vast majority of torsions of the umbilical cord are the result of the revolution of the fetus upon its long axis,

followed by the gliding of the cord over the cephalic or caudal end. During early gestation any elasticity in the cord would tend to automatically overcome torsion.

In most species of domestic animals the umbilical cord is too short to permit readily the revolution of the fetus upon either the longitudinal or the transverse axis. This may play a part in the relatively greater frequency of torsion of the uterus than of the umbilical cord. In the equine fetus, having by far the longest navel cord, some degree of torsion is general and physiological. At what degree the torsion becomes pathological has not been determined. Neither has it been determined in which direction the torsion usually occurs, nor that the torsion is more probable in one than in the other direction. Evidently the individual fetus must repeatedly revolve in the same direction, or extensive torsion could not occur.

At my request, Professor Danks has made observations upon the torsion of the umbilical cord in a few available equine fetuses. In all cases the torson was to the right, or clockwise. Fetal death, amnio-allantoic dropsy and other pathological phenomena are attributed to the torsion of the navel cord, but authentic evidence of such result is difficult to establish. Limited personal observations apparently indicate that in the equine fetus the more extensive and virulent the existing disease of the maternal and fetal placentae, the more extensive the torsion of the navel cord. The equine fetus, in advanced pregnancy, visibly moves more abundantly and violently than observed in other species. The ordinary mare used jointly for breeding and farm work is under the most constant observation of any pregnant domestic animal. When the farmer observes exaggerated fetal movements, he suspects fetal disease and fears abortion. The horse is preeminently subject to colic and rolls violently. A prominent cause of colic is impaction of the large intestine, which is common in the horse and rare in ruminants. The equine fetus suffers abundantly from impaction of the intestine with meconium and in numerous cases the new-born foal is in violent colic which may authentically be designated as congenital colic, paralleling in a way the fetal diarrhea of the calf. Hence it is none too clear whether torsion of the navel cord causes fetal disease and death, or whether fetal disease causes torsion of the cord.

AMPUTATION OF FETAL EXTREMITIES BY THE NAVEL CORD (?)

Amputation of fetal extremies by the navel cord has been described as not infrequent in the human fetus, in which a detached hand or foot has been found under such conditions that the ob-

servers have ascribed the amputation to incarceration within a coil of the navel cord. The detached member is much smaller than the living stump upon the central side of the division. Writers fail to explain how the cord might be drawn so tightly as to cut off the circulation to the parts distal to the amputation without obstructing the blood flow in the cord and causing the death of the fetus.

Veterinary literature records one solitary case of alleged intra-uterine amputation of an extremity by incarceration in the navel cord. Harms[89] cites Gayzi as having observed a living foal in which the right hind foot had undergone intra-uterine amputation. The fact of the amputation seems beyond question. The navel cord was not observed in the process of incarcerating the foot, and no evidence was submitted of damage to the cord at the supposed point of contact. Recently Streeter[190] has submitted convincing evidence that, in the human embryo, the available facts indicate that the navel cord amputation hypothesis is erroneous. Streeter submits incontestable evidence showing that intra-uterine amputation of the fingers and toes is equally frequent to that of the hands or feet; the lesions are identical in both cases; and it is mechanically impossible for the navel cord so to incarcerate the fingers, and especially the short toes, as to cause necrosis or other injury. While it may be imprudent to say that amputation of a fetal extremity by the navel cord is impossible, it may be safely stated that the hypothesis is unproven.

Utero-fetal Disease and Dystocia

In 1931[251] I took the position that dystocia in domestic animals is not usually the consequence of accident, but is preponderantly the result of defect or disease of the fetus or its membranes or of the reproductive system of the mother: all three factors commonly participate. All obstetrists admit that abnormalities in the form, volume and consistency of the fetus interfere with its expulsion. The evidence seems overwhelming that the healthy fetus assumes the physiological presentation and position upon the advent of labor. This is improbable if the fetus is diseased. Yet more frequent is dystocia when the fetus is dead or non-viable, even though the pregnancy has been abbreviated. It is not rare to meet dystocia in a cow aborting at seven months when the fetus is little, if any more than half the normal volume.

In some cases of amniotic dropsy, the amnion is sclerotic and must be surgically divided in order to permit the expulsion of the fetus. When the maternal placentae (bovine caruncles) are diseased, so that the retention of the afterbirth, is foreordained, dystocia prevails, the

expulsive powers are subnormal and the fetus is largely in pathological presentation or position. The endless list of causes of dystocia, when carefully traced to their ultimate origin, are, with infrequent exceptions, referable to abnormalities of the fetus, fetal membranes, or uterus.

FETAL DEATH

In the preceding pages numerous causes of fetal death have been named and the ultimate destiny of the fetal cadaver mentioned. The destiny of the cadaver is largely influenced by the cause and the environment or by the species of animal.



FIG. 184. Twin Equine Aborts.

The lesser fetus died at about five or six months and underwent papyraceous mummification: the other died and the two were expelled or removed at about ten months. The afterbirths are shown in Fig. 19. *(Cornell Veterinarian)*

Abortion. When fetal death and expulsion occur relatively early in gestation it is customarily classified as abortion; if it dies late in gestation, the writer may call it either abortion or stillbirth.

Fetal death is not uniformly followed by the prompt expulsion of the fetal cadaver. The cause of the generally prompt expulsion of the cadaver is not known. This is not readily determinable so long as the cause of physiological birth remains a collection of hypotheses. Abortion is preeminently a phenomenon of uniparous pregnancy. The death of one or more fetuses in a multiparous uterus is usually followed by retention of the cadaver in a non-perilous state and the

survival and birth of other living litter-mates. There are exceptions. Twin and multiple gestations in mares and cows largely end in abortion. Under some conditions their behavior resembles that of pluripara, and when one of the twins dies, the other may continue to live for a period, as indicated in Fig. 184, until it finally succumbs and the unequal twins are aborted simultaneously. Or in both cow and mare, the dead fetus may be expelled and the living one go to term and be born viable. Rarely, a cow in twin pregnancy expels one viable twin prematurely and carries the other to term, when it is also born viable.

FETAL DEATH WITH PAPYRACEOUS MUMMIFICATION

The term "mummification" is elastically used in veterinary science to cover wholly unrelated pathological phenomena. The most familiar type is that already described as bovine hematic mummification. Quite apart from this type is the desiccation of embryonic cadavers of swine, such as those shown in Figs. 91 and 92. A somewhat similar condition is seen in the twin equine aborts of Fig. 184.

The type is limited in domestic animals, so far as known, to swine, and to twin gestation in the mare and cow. Apparently it is comparable to a type of mummification in human twins wherein one fetus dies and the other survives. The form of the human uterus, Fig. 12, is such that the fetal cadaver may be compressed by the live twin and thus become markedly flattened or discoid. This cannot readily occur in any species of domestic animal, because the form of the uterine cavity does not permit two fetuses to lie side-by-side. The appearance of such cadavers suggests that a partial solution of the soft tissues with resorption has occurred. If infection is present, it is held in abeyance in such a manner that ordinary putrefaction is excluded and no serious injury to the uterus occurs.

Extra-uterine pregnancy, which inevitably ends in fetal death, barring cesarean section, is not authentically known to occur in domestic animals. Some writers, like Foster[63] and Goodfellow[72] have reported extra-uterine pregnancy in the mare without authentic details, under circumstances to indicate that they were dealing with bicornual pregnancy instead.

Extra-uterine fetuses are sometimes found in slaughtered swine, and possibly more rarely in other species in which fetuses are in the peritoneal cavity completely encapsulated within a fibrous sac, as shown in Fig. 166. The phenomenon is presumably due to rupture of the uterus with escape of the fetuses. The cadavers sometimes remain static in volume, persisting indefinitely as passive bodies. They

are frequently designated as *mummies,* although wholly unrelated to other fetal cadavers so classified.

The *static fetal cadaver,* as I have chosen to designate it,[248] has been observed rarely in the cow. The fetal death occurs at about term and the fetus remains plump and rigid as in *rigor mortis* for an indefinite period of time. I[248] have described a case in which the fetus remained static for 23 months. Boyd[18] describes a case which had so remained for 20 months. Both cases were associated with uterine torsion but it was not determined in either case which phenomenon occurred first—the torsion or the fetal death. I have observed a second instance of the fetal phenomenon in which there was no uterine torsion. The cause of the phenomenon is unknown.

Disintegration of the retained fetal cadaver occurs in a variety of forms. The type of disintegration is modified by the character of the invader associated with the fetal death. There is nothing in veterinary literature indicating that fetal cadavers in which the death is ascribed to a virus are retained long in the uterus, to undergo extensive disintegration.

In the preceding pages it has been stated that fetal death is ascribed to two separate protozoa, the *Trypanosoma equiperdum* of dourine, and the *Trichomonas fetus* of the trichomonad disease of cattle. Fetal death due to the former is but vaguely described and is certainly rare. Recently the trichomonad disease of cattle has abruptly forged its way to the front in order to explain many of the bovine abortions for which *Bacillus abortus* could not be held responsible.

In trichomoniasis, retention of the fetal cadaver, and its disintegration, are outstanding phenomena, constituting a valuable, if not the most important, symptom by which the presence of the protozoon may be diagnosed. After a cow has been apparently pregnant for a few months, there appears a distention of the uterus defined as pyometra, and frequently, if not regularly, there is within the uterine contents a fetus undergoing disintegration. The uterine contents are designated *pus,* although the fluid is not wholly like the substance ordinarily so called. It is thin, flocculent, grayish or yellowish. It does not promptly become fetid when exposed at room temperature. Apparently bacteria and the trichomonad cannot readily live together. The pyometra-distended uterus does not usually, if at all, suffer from peritoneal involvement. Consequently peritoneal adhesions of the uterus apparently do not occur. The general health of the cow is not profoundly affected.

BACTERIAL MACERATION OF THE FETAL CADAVER
PUTRID DECOMPOSITION OF THE FETUS

Bacterial destruction of the retained fetal cadaver and its membranes occurs in all species of domestic animals. In multipara, the fetal death and decomposition may occur in one or several fetuses, while some litter-mates remain apparently healthy. If the embryonic death occurs early, the disintegration and resorption may be so com-

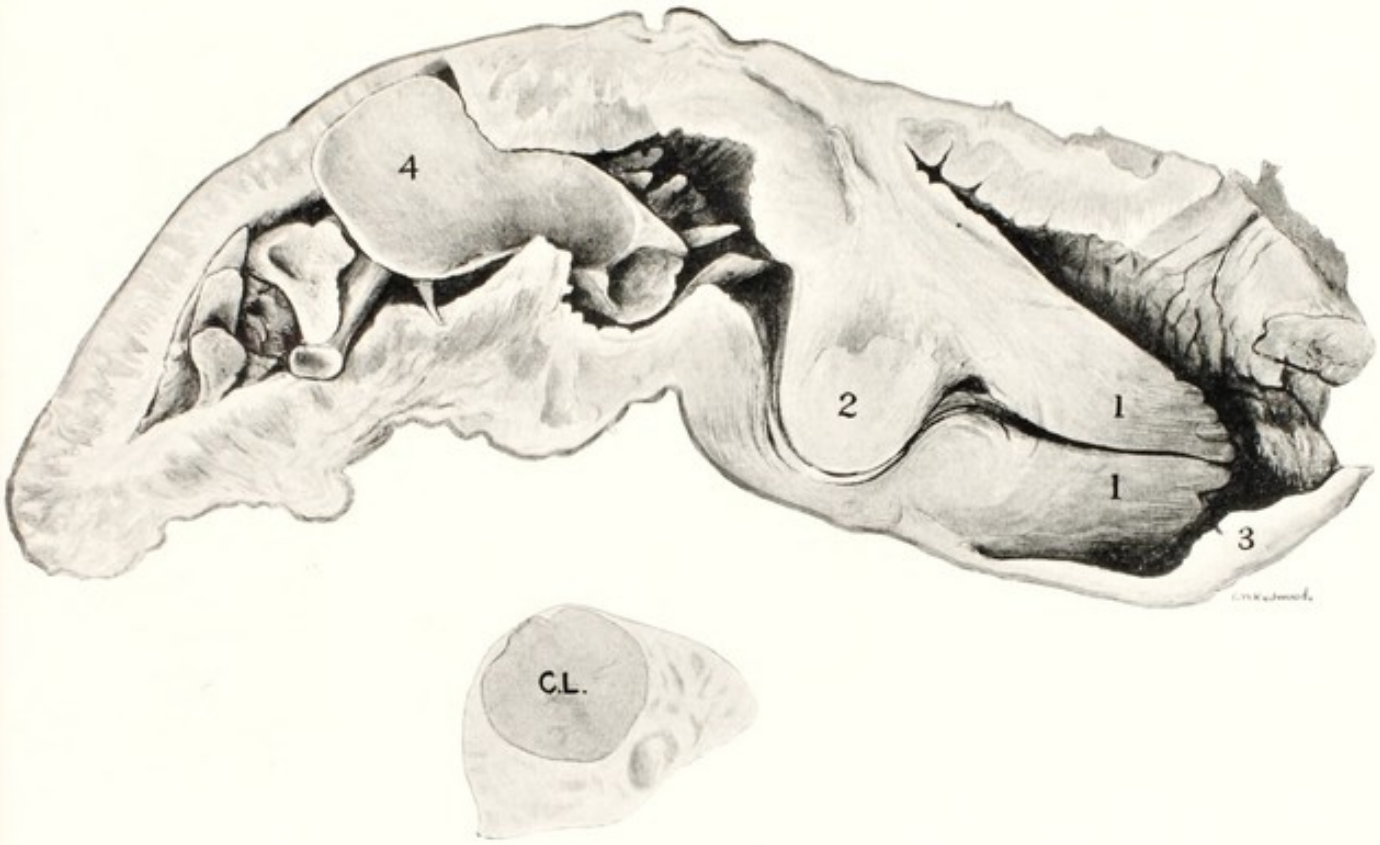


FIG. 185. Maceration of Bovine Fetus With Retention of the Bony Skeleton in the Uterus.

1, 1, The cervical lips; 2, greatly enlarged, sclerotic annular cervical fold; 3, vagina; 4, parietal bone of fetus, resting against a confused mass of fetal bones; *CL*, embedded (retained) corpus luteum of pyometra.

plete, for example in swine, that the only evidence of the occurrence in the abattoir is the presence of more corpora lutea than of embryos or a small mass of detritus of such appearance and location as to suggest the remnants of an ovum. The completeness of the disintegration decreases as pregnancy advances. Thus, in a supposedly pregnant heifer which had passed her expected date of calving, I recognized, upon rectal palpation, a roundish tumor of even contour at the bases of the two cornua. Upon autopsy, the enlargement was found to consist of a mass of inspissated pus about three or four inches in diameter. Embedded within this were found several shafts of long bones

about one-half inch long. The disintegration had proceeded under such control that no observed disturbance in the health of the heifer had occurred beyond the absence of estrum, without signs of advancing pregnancy. Had the true character of the disease been recognized by rectal palpation, the dislodgment of the corpus luteum would quite certainly have caused the expulsion of the fetal debris, and the heifer might possibly have proven fertile.



FIG. 186. Maceration of Bovine Fetus With Fetal Cadaver Sloughing Through the Abdominal Floor. (Guard)

In the sow the decomposing fetuses may be located at either the ovarian or the cervical pole of the cornu, or at any point between. When labor sets in, the decomposing fetal cadaver causes dystocia, in which it differs from the papyraceous mummy described above. The dystocia is partly caused by the increased size of the decomposing cadaver, and partly by the concurrent localized metritis. When the death and disintegration occur in advanced gestation, the complete liquefaction of the cadaver becomes impossible: the bones, hair, and other structures defy destruction. The bones and hair develop later in carnivora than in solipeds and ruminants and disintegration of dead ova is under more effective control. When a bovine fetus dies at four or five months its osseous skeleton is well advanced. If the cervical canal remains passable, as in Fig. 185, the soft parts of the fetus may disintegrate and, mixed with pus, be discharged through

the vulva to constitute *pyometra*. If the genital tube be obstructed at any point and fetal development has passed beyond the stage where total disintegration and resorption may occur, the decomposition of the fetal cadaver, if the female survives, may cause perimetritis with adhesions to surroundings organs and tissues. Thus, in the torsion of the uterus of the cat shown in Fig. 165, the incarcerated fetal cadaver became encysted in the peritoneal cavity, the system was competent to resorb the soft parts, and the skeletal debris remained as a neutral foreign body. In contrast, the uterus of the ewe, shown in Fig. 164, acted as an abscess cavity, so that the owner observed the expulsion of the fetal cadaver through the abdominal floor. The case reported by Guard[74] in which a bovine fetus sloughed out through the abdominal floor, as shown in Fig. 186, may also have been due to torsion of the uterus, especially since Guard states that the vulva was too small to admit the hand and that the vagina was so occluded that the cervix could not be seen with the aid of a small speculum.

Uterine torsion is not essential to putrefaction of the bovine fetus and the sloughing of portions of the cadaver through the uterine walls. In one case of fetal maceration the vulva and vagina of the cow were comparatively normal. The cervix was extremely sclerotic, somewhat like that shown in Fig. 185. The cervical canal was severely constricted, but permitted the escape of pus and some portions of fetal debris. The heifer had repeatedly suffered from fetid diarrhea, with dark feces. I dilated the cervical canal by incision, passed my hand into the uterus, and removed fetal bones, fragments of skin and whole grains of maize and oats from the uterine cavity. Evidently the uterus had ruptured into the alimentary canal, presumably the rumen. Fetal debris had entered the alimentary canal and caused the attacks of diarrhea, and the alimentary canal had discharged into the uterus grains of maize and oats. After the removal of the fetal remains from the uterus, the heifer made an uneventful recovery and was sold for beef without opportunity for post-mortem observation.

There is no known law controlling the path of the escape of the disintegrating fetal remnants. It has been noted above that detritus may escape through the cervical canal, the abdominal floor, and via the rumen through the alimentary canal. Cases are reported of the discharge of the fetal cadaver into the urinary bladder. Sometimes the fragments become impacted in the cervical canal. W. W. Williams observed the sloughing of a two-months bovine fetus into the rectum,

CHAPTER XXXI

THE PATHOLOGICAL PHENOMENA SURROUNDING THE PERIOD OF BIRTH

THE birth of young constitutes the supreme crisis in reproduction. If there has been error in the method of the breeding of animals, as discussed in Section I, and the ovum and pregnant female have survived, many perils arise to both mother and young just prior to expected birth, during that act, or immediately following. Any diseases or defects of the gravid uterus or of the contained ovum which have failed to terminate the pregnancy are brought prominently into the foreground, threatening the health and life of the dam and young. There occur in all domestic animals about the close of pregnancy, just before, during or soon after birth, an extensive series of phenomena in the mother, apparently analogous to the group of diseases classed by J. Whitridge Williams[226] and other human obstetrists as the *toxaemias of pregnancy*. Prominent among these is the milk fever or parturient paresis of the cow, and less frequently of other ruminants, which may develop just before, during, or soon after birth; the parturient eclampsia of the mare, observed after birth; the pregnancy disease of ewes; and the various types of dystocia already described in the companion volume, *Veterinary Obstetrics*.

In domestic animals there is no clear line of demarcation between the diseases of the mother and fetus during late gestation: the diseases of mother and young largely cooperate during the birth act, and continue during the puerperal period along somewhat parallel lines. Birth is not a cure for uterine or fetal disease. If metritis exists during late pregnancy, it continues through the birth act to cause dystocia. During the puerperium the metritis persists and tends to fulminate. It largely reveals itself, especially in the dairy cow, in retained afterbirth. The freely open birth canal may modify the prevalent type of bacteria or other invaders, but the metritis remains and may either increase or decrease in virulence. So the bovine fetus may suffer from diarrhea or the equine fetus from colic impaction, but birth is not a remedy for either disease.

Metritis of Advanced Pregnancy

The metritis of late gestation, and during birth and the puerperium, offers an infinite variety in symptoms, intensity, and eventual result.

It may be profitable to discuss the disease under the two indistinct types of acute septic metritis and sub-acute metritis.

Acute septic metritis is not very common during pregnancy in other animals than dairy cows, and is frequently obscured in them by dystocia and retained placenta. During my experience for one year in the Holstein herd illustrated in Table X, I observed two terminations of twin pregnancy. One of these, a primipara, born at 269 days, was herself a twin—and the only heifer calf out of a dam of low fertility. She dropped dwarf, defective, worthless twins from a pasture breeding. The heifer suffered from irremovably retained afterbirth, with severe sepsis and extensive necrosis of the uterine caruncles. Since her breeding outlook was hopeless and the twins worthless, all three were destroyed. The second twinning cow, nine years old, had produced six viable calves, the last three at intervals of 11 months. Her twin pregnancy began twenty-six days post-partum, and dead male twins were removed by traction at 301 days. The cow had been losing flesh for some weeks and suffered severely from pyaemic gonitis. Her uterus was severely inflamed and paretic, and the afterbirth retained. Since extensive necrosis of the caruncles destroyed all hope of future breeding, she was killed. Both cows might have survived, but were worthless.

Septic metritis with uterine gangrene is not rare in dairy cows. The cow near term, apparently well in the morning when turned to pasture, is found down unable to rise in the evening, with portions of the afterbirth protruding, accompanied by dark fetid discharges and, in many cases, by diarrhea with dark fetid feces. The uterus is dead, the fetus is emphysematous, and powerful traction is required for the removal of the cadaver. The cow dies. The uterine gangrene may occur in a heifer without history of prior disease. Thus Heifer No. 1 in Table I was apparently well in the evening and was moribund when I saw her the next morning. No effort was made to remove the fetal cadaver, which was enormously emphysematous.

At times septic metritis becomes general in a given herd. Wing[271] reports a serious outbreak of acute metritis in the Cornell University herd in 1910 and 1911, but gives no details. Herd veterinarian at that time, I computed the losses at twenty per cent mortality, and twenty per cent absolute sterility. Wing's bulletin deals only with Holstein-Friesians, but all breeds were involved. According to Wing's Table 8, there were twenty-three Holstein pregnancy terminations in 1910-1911, and nine of these cows are recorded as having calved or aborted in 1911-1912—a decrease of 65.2 per cent. Some of these may have been sold. It was during this outbreak that the eighteen heifers in

Table I were born. It seems that the herd was saturated with the infection, and that this seriously depressed the reproductive powers of those cows which bore viable calves and survived the outbreak.

Writers upon human obstetrics largely attribute septic metritis to errors in asepsis by the obstetrist, midwife, or nurse. These cows and heifers were not exposed to possible manual infection until after the acute metritis had developed. A large ratio of the fatal cases and of the cases of permanent sterility were primipara and most of them were free in pasture at the onset of the disease. Dystocia was common and retained afterbirth highly prevalent. Material was handed to the Department of Bacteriology at the Veterinary College. The invader was reported as a micrococcus. There was no suggestion that it was the *Micrococcus melitensis* of Bruce. I worked assiduously upon the problem, without any recognizable effect. If I removed the afterbirth manually, the cow died; if I did not remove it, the cow died. If I failed to douche the uterus, the cow died; if I tried to douche the uterus, that organ ruptured from the weight of a moderate volume of water. A junior colleague thought that I erred in my technic in the removal of the afterbirth. When the next case occurred I asked him if he would be so kind as to remove it for me. He did so cheerfully, and reported the cow safe. She was dead in 48 hours. The cows were doomed before labor set in. Dystocia was general and the progress of birth was slow in those not requiring assistance. As soon as the fetus was out, the infection exploded and I was helpless, both upon my own resources and with any aid I could command from colleagues, including clinicians and bacteriologists. Several lesser outbreaks of acute septic metritis have since come to my notice. One colleague was nonplussed by the prevalence of retained afterbirth in a large public herd, where virtually all cows died after his removal of the afterbirth. Having founded his conclusions upon an extensive clinical experience, he had come to regard retained afterbirth as a minor problem and could not understand his humiliating helplessness in the virulent outbreak of retained afterbirth and septic metritis which he faced.

Septic metritis occurring in pregnant cows may reveal its presence by pyaemic arthritis or serious disease of other organs. When the date for parturition arrives, the labor is atonic, and not rarely the cow goes down early, the sepsis is profound, the fetus emphysematous, and there is a thin fetid vaginal discharge. Milder cases suffer from dystocia. The temperature is erratic and may be either abnormal or subnormal. After the expulsion of the fetus the placenta may be completely retained, the appetite lost, and an acute diarrhea present with thin, black, fetid feces. In such cases the temperature is markedly

subnormal. The profuse, fetid diarrhea, in either the pregnant or puerperal cow, may lead to an erroneous diagnosis of primary digestive disease. I have known an able practitioner to diagnose acute enteritis in a puerperal cow with complete retention of the fetal membranes within the necrotic uterus. No portion of the umbilical cord or other part of the fetal membranes was visible. The fact that the animal is in advanced pregnancy, or has recently calved or aborted, should direct attention to the uterus, an examination of which authentically reveals the character of the disease.

The sub-acute or milder type of metritis reveals itself in a variety of ways. It disturbs the duration of pregnancy by abbreviation or prolongation. There is inevitably more or less uterine inertia, which leads to dystocia. The expulsive powers may be subnormal and lead merely to prolonged labor; the fetal membranes may be sclerotic, delaying or preventing their rupture by the weak uterus; the sick or dead fetus is irresponsive and one or more of its extremities become misdirected and the expulsion of the fetus obstructed. After the expulsion or extraction of the fetus, retention of the placenta is highly probable; necrosis of the uterine caruncles or other portions of the organ usually occurs; and in the bovine uterus pus and necrotic debris accumulate in the dependent ovarian pole. Sometimes this is actively expelled, causing a relatively constant purulent vaginal discharge; frequently uterine inertia exists which prevents active expulsion, and the pus and debris are passively expelled by external compression when the animal is recumbent. Less frequently the pus is largely or wholly retained within the uterus for a varying period. Resorption of the products of necrosis occurs, which may cause pyaemic disease of the articulations, and pyaemic abscess of the sublumbar lymphatics and of various organs. Rarely the cervical canal becomes blocked and uterine abscess follows. A large ratio of the animals recover their general health and varying degrees of fertility. The prognosis and handling are discussed in connection with retained afterbirth.

Retained Afterbirth. Retained Placenta

Retained afterbirth is difficult to define accurately.

There exist important anatomical differences in the various species. There are two major groups of placentae—the deciduous and adeciduous. The human placenta is the most highly developed in the former class, which is represented in veterinary medicine by the placentae of carnivora. An extreme example of the latter is the equine placenta.

It is not known, especially in the adeciduates, by what force the

fetal and maternal placentae are brought into contact and their physiological relation maintained until the fetus is born. When the fetus has been expelled, the force which causes the afterbirth to drop away is not known. Owing to these uncertainties, the cause of the abnormal retention is, in some important technical respects, unknown. When labor sets in, the life of the fetus is primarily dependent upon the integrity of the placental contact until the young animal may begin pulmonary respiration. This must be within a very brief time, since otherwise death occurs from asphyxiation. The equine afterbirth is sometimes completely detached and expelled in advance of the fetus, when, if the fetus was not already dead, it promptly becomes asphyxiated.

Whether deciduate or adeciduate, the fetal membranes abruptly fall away immediately after physiological birth. The deciduate placenta of woman, according to authorities in this field, is detached from the healthy uterus within thirty minutes after the birth of the child; in healthy parturition in the mare the fetal membranes have been completely expelled within half that time. The placenta of woman is relatively larger, in relation to the weight of the fetus, than it is in domestic animals, and is more compact, forming a considerable mass which after separation continues to offer some difficulty in expulsion through the cervix and vagina.

The multiparous domestic animals physiologically have independent fetal sacs with zonular placentae. After one fetus has been expelled it is essential that the placenta follow promptly in order to permit the expulsion of the next fetus in that cornu. On the other hand, when twins or multiples are present in the cow, with chorionic fusion including vascular anastomosis, the life of the second fetus is dependent upon the continuance of the placental contact of the entire chorion. If unicornual, the second fetus passes through the chorionic canal of the first in order to be born. It may therefore be needful that the relations of the placenta of the first fetus be continued for several hours in order that the second may survive. In fact, however, the afterbirth in such cases is communal and the same law which causes it to retain its relation with the uterus in single pregnancy applies to the second twin: it is the afterbirth of the second, as well as of the first twin.

The line of demarcation between the physiological expulsion of the placenta and its pathological retention varies according to the view of the writer, and commonly differs for the various species of animal. It is generally agreed that physiologically the mare expels the fetal membranes within thirty minutes after the birth of the foal.

Schmaltz[178] states that the bovine fetal membranes are normally discharged within 3 to 8 hours. Generally the afterbirth of the cow is said to be retained if it has not been expelled within 6 to 8 hours. Retention of the fetal membranes occurs in all animals. The incidence and virulence in the cow are many times greater than in all other species combined. It is claimed by writers on veterinary obstetrics that the long, finely branched chorionic tufts, Fig. 46, which sink deeply into the crypts of the uterine caruncles, constitute a logical anatomical background for retention of the fetal membranes. But a careful study of the problem tends strongly to deny such hypothesis. In the ewe and goat, which have quite similar placental arrangements, retained placenta is not at all common. I have seen little of goats, but have had considerable experience with sheep, without having observed an instance of retained placenta. Howarth[93] records a high incidence of retained afterbirth in a flock of over 800 ewes, in which more than twenty-five per cent aborted. It appears that, when abortion rages in a flock of ewes, retained afterbirth prevails as virulently as in groups of cows in which sterility and abortion are common. The difference is that abortion in sheep is not common. However, when it does affect a herd, the consequences are parallel to those in cattle.

Further doubt is cast upon the hypothesis, that the high incidence of retained afterbirth in the cow is due to the placental anatomy, by the great variation of incidence of the phenomenon in different breeds and under various methods of handling. Thus in Table X there were in the Holstein-Friesian herd less than 100 pregnancy terminations during my residence for a year as herd veterinarian. Retained afterbirth was a major problem which commanded my daily attention. During this period there were 5000 pregnancy terminations in the closely surrounding market beef herd of Herefords. Passing among these daily I observed no instance of retained afterbirth, and no case was mentioned to me by the personnel having charge of the Herefords. Doubtless retained afterbirth occurred, but not being of that frequency which constitutes an economic problem, was not mentioned to me. So far as known, the placental anatomy and physiology in Holstein-Friesians and Herefords is identical, but the incidence of retained afterbirth was spectacularly unlike.

It is further to be noted that the hypothesis mentioned is in sharp antithesis with the hematic mummy described in Chapter XXX. The hematic mummy, known only in the cow, is dependent upon an abrupt separation between the fetal and maternal placentae owing to profuse interplacental hemorrhage. It seems illogical that the

anatomical character of the placentae should cause the abrupt dehiscence of the fetal placenta in one case and its pathological retention in the other.

Going back to early pregnancy, as soon as the allantois is formed, cotyledons or fetal placentae appear upon the allantochorion. They correspond accurately with the placental areas on the summits of the uterine caruncles, in number, size, and contour. In the slaughtered animal the ovum lies free within the uterine cavity, with each chorionic placental area located directly facing the uterine caruncle to which it belongs. I have not discovered a single instance in which this relationship had been disturbed. No trace of a fetal placenta has been found indicating, or faintly suggesting that the ovum had been displaced and that any one of the 100 to 140 fetal placentae had shifted its position so much as a hair's breadth during the life of the cow. Much speculation has been indulged in regarding the grave injuries which may follow falls, kicks or other forms of physical violence to pregnant females. It has been claimed that such violence may detach the fetal placenta and cause abortion or other disaster. But no authentic record is found in which such violence has directly caused abortion. Abattoir studies of females in early pregnancy fail to reveal evidences of placental displacement owing to the violence of slaughter. The fetal and maternal placentae are not held together during early pregnancy by the anchorage of the chorionic tufts within the caruncular crypts: neither structure exists. There is no anatomical arrangement or structure which may be identified as the force which maintains the accurate and vitally essential contact between the fetus and uterus. The force responsible for the maintenance of the placental contact, in the early pregnancy of adeciduates, is wholly unknown. When birth occurs and the umbilical cord is ruptured, that force ceases to exist in the healthy female, and in thirty minutes the fetal membranes *fall* away: they need not be expelled. The fetus, on the other hand, cannot *fall* away, but needs be *expelled,* owing to its large size compared with the dimensions of the birth canal.

Retained Afterbirth in the Mare

Retained placenta in the mare is of two somewhat distinct types. The entire fetal membranes may be retained, or only the non-gravid chorion.

Retention of the entire placenta is usually observed following abortion or prolonged gestation. Abbreviated pregnancy or premature birth of a viable foal is rare. I recall one case only, birth occurring at about 300 days, followed by retained placenta. Prolonged

gestation is common in the mare and is frequently associated with retained afterbirth. Thus the retained afterbirth shown in Fig. 30 followed a pregnancy of 387 days. The foal shown in Fig. 190 was carried 380 days and retained afterbirth followed.

In a group of nineteen thoroughbred mares in a stud of low fertility and mated with famous stallions, infamously diseased, the afterbirths of fifteen of them were expelled within fifteen to thirty minutes, after an average duration of pregnancy of 345.9 days; the other four mares with average gestation periods of 356.5 days retained their afterbirths more than thirty minutes. Two of the four mares which had retained afterbirth were barren the following year. The symptoms of this type of retained fetal membranes are too obvious to require description.

The second type of retained afterbirth in the mare consists of the retention of the non-gravid cornu only. I have observed this only in draft mares. The chorion of the body and gravid cornu drop loose promptly and hang to the ground. The slender, frail non-gravid cornu is torn through at its base, either by the massive weight of the protruding portions or by the mare stepping on them. The fragment is usually but six to ten inches long, and is quite firmly retained. Although there may be a somewhat unusual vaginal discharge, the placental retention is not usually identified for one to three days, when acute laminitis ordinarily appears. This leads to an examination of the uterine cavity, and the retained placental fragment may be found, or the fragment may have been expelled and purulent metritis remain. It is a good example of the apparent existence of bacteria located deeply within the pregnant uterus. At the time of birth, the retained non-gravid chorion is located at a point three or four feet from the exterior. The rupture is produced by linear tension within a few minutes after foaling. No accidental exposure of the uterus or manual exploration can be incriminated in the invasion.

Parturient laminitis is the most common result. The mare usually maintains recumbency in order to lessen the pressure upon the feet. When compelled to rise she shows great pain and walks upon her heels in order to lessen the pressure upon the sensitive laminae. The symptoms of parturient laminitis do not differ from those caused by indigestion. The disease is very severe and frequently ends fatally. Rarely parturient laminitis also accompanies retained afterbirth in ruminants.

When retained afterbirth of this kind is suspected because of laminitis, purulent genital discharge or other signs, the character of the disease is authentically determinable by manual exploration if

the fragment is still retained. It may drop away at any time and disappear. In one case, as my client and I approached the recumbent mare, she got up, and the placental fragment dropped. My client's dog was just ahead of us and swallowed the fragment before we could interfere.

The prevention of retained placenta in the mare has not been placed upon a secure basis. Its incidence has not been compared with the breeding history of the mare. It is presumably due to causes parallel in principle with those applying to the dairy cow. Among these may be suggested ninth-day breeding, the breeding of racing fillies soon after retirement from the turf, the breeding of mares of low fertility, and the mating of mares with stallions of low fertility.

The consequences of the fragmentary retention are quite controllable by the very simple means of inspecting the afterbirth. Any intelligent groom, after casual instruction by a veterinarian, can spread out the afterbirth and determine if the fragment is missing. If retained, its immediate removal with aseptic washing is usually advisable.

The handling of retained placenta in the mare has not been extensively studied. The general custom is to remove the membranes manually, although some would leave them undisturbed for a day or two. By removing them manually within an hour or two after the mare has foaled or aborted, the average results appear better, because one removes thereby many membranes which, if left alone, would automatically have dropped away shortly afterward. If the membranes are destined to remain for a day or two, an effort to remove them an hour or two post-partum may fail, or, if undue force be applied, the maternal placenta may be perilously injured. Upon the other hand, the great weight of the fetal membranes may tend to cause uterine prolapse.

When the membranes are long retained, the protrusion of the exposed parts is increased when the mare lies down; when she rises, the weight of the uterus pulls the membranes back into the vagina and drags with them bacteria with which they have come in contact. This is notably observed in the occurrence of puerperal tetanus. Accordingly the plan of immediate manual removal, and that of delay, each has its dangers, and the clinician needs treat each case individually. I believe it advisable, when the membranes have been retained over thirty minutes, to make a careful examination and, if they can be readily detached, remove them, and wash the uterus with warm physiological salt solution. The removal may be accomplished by grasping the protruding membranes with one hand and,

exerting gentle traction, introducing the other hand between the detached portions of the membranes and the uterine walls until the point of retention is reached. The detachment may then proceed by insinuating the finger tips between the two placentae and gently pushing them apart. Or the detached membranes may be partly or wholly encircled by the thumb and fingers and, while equal traction is applied to the protruding membranes by the one hand, the uterine wall may be pushed forward, as by a ring, away from the membranes. In the second type of disease, when it has passed undiscovered until the copious discharge of muco-pus occurs, frequently accompanied by laminitis, the retained fragment is to be carefully removed, and the highly infected endometrium thoroughly cleansed by douching with warm physiological salt solution. This should be repeated once or twice daily until the danger from laminitis has passed or, if it be present, until the uterus has undergone involution. The laminitis is best handled by standing the mare in cold water or, if recumbent, by applying ice poultices to her feet. The outcome is none too promising.

When the membranes are not removable by the application of prudent force, their putrefaction may be somewhat held in abeyance by the introduction into the ovarian end of the chorion of moderate quantities of a slowly-dissolving antiseptic like iodoform, thymol iodide or bismuth subnitrate, suspended in a pint or two of mineral oil.

Retained Placenta in the Cow

Retained placenta in the cow is not defined in a manner universally acceptable. Many classify the membranes as *retained* when they have remained in the uterus for six hours after having calved or aborted, while others designate a far greater duration of time. I[235] made observations upon the duration of the retention of the fetal membranes within the uterus, and associated phenomena, in a small number of cows which I believe were representative of the large herd of which they constituted a part. The herd was badly diseased. Twenty cows were in labor from 0.33 to 35 hours with an average of 3.21 hours. Nineteen of these retained their afterbirths from 1.2 to 43.4 hours, with an average of 14 hours. The calves were removed manually from two of the cows at 3 and 35 hours respectively. Three of the fetal membranes were removed manually at 34, 36 and 25.3 hours respectively. Six, or 30 per cent of the calves died before fifteen days old. Their afterbirths had been retained 24.3 hours as compared with the group average of 14 hours. Only two afterbirths were retained less than 1.5 hours, and these followed pregnancies

of 281 and 288 days respectively, as compared with an average of 278 days for the group. The group was of low fertility, with an abortion rate of 27 per cent. In the group 73.3 per cent were positive to the agglutination test for *Bacillus abortus*. The common statement in veterinary literature, that a given cow or a given ratio of cows in a herd has suffered from retained afterbirth, is not precisely informative. It would be vastly better if the writer recorded the duration of the retention. Generally the reader may draw the inference that when retained afterbirth is recorded the writer means 6 to 24 hours, with the latter period predominating.

The character of the phenomenon varies. The usual type of bovine retained placenta requires no material description. There is present the perfectly obvious mass of membranes hanging from the vulva. With that may be associated symptoms of metritis; there may, or may not be expulsive efforts; the appetite may be affected in any degree; the temperature may be abnormal, normal, or sub-normal; and there may be constipation or diarrhea. The recognition of the retained afterbirth is the basis for diagnosis. The retained placenta may at times elude detection when the clinician or breeder is off his guard. I have observed an experienced practitioner handling a cow for a violent fetid diarrhea as a disease of the alimentary tract. Suspecting error, because the cow had calved or aborted a few days before, I examined the uterus and found it filled with putrid, highly fetid fetal membranes. In another case, while visiting with a colleague, he had occasion to examine the genital tract of a heifer, the pregnancy of which had recently terminated. She appeared well in general. When my colleague passed his hand into the uterus, he was completely mystified concerning the contents which he palpated. The fetal membranes, or a major portion of them, lay in the uterus with all uterine caruncles detached from the uterus and firmly attached to the fetal membranes. I have observed the same condition upon the killing floor of the abattoir. The cow had been passed by the yard veterinary inspector, she looked clean and well, her viscera were passed by the inspector on the floor, and her uterus, passed over to me as pregnant, for study, was filled with decaying membranes. When meeting with these cases I failed to determine whether the membranes were actually complete. My suspicion now is that they were not. The cow, as is well known, swallows her afterbirth as quickly as possible, unless it has become repulsively putrid and fetid. If a considerable portion of the membranes protrude from the vulva, she promptly grasps them and begins swallowing. This may lead to complications if portions of the membranes are firmly retained. The late Professor Law related a rare case in which he was hurriedly

called to attend a cow which had swallowed a large part of the membranes sufficiently to have them firmly incarcerated in her esophagus, while the other end was held with equal firmness in her uterus. Since the membranes were highly resistant, the cow was held with her nose to her tail. Usually the membranes rupture, and the cow swallows part, while the remainder tends to spring back into the uterus out of sight. The rupture of the membranes usually occurs at one of two weak points. The parturient rent at the cervix, through which the fetus is expelled, leaves a wide tear at the juncture of the gravid and non-gravid cornua, which favors separation when one cornu protrudes and is grasped by the cow. The great allantoic arteries, extending from ovarian pole to pole, offer much resistance to the transverse rupture of the chorion. The weakest spot in the length of the membranes is at the allantoic navel, where one of the great arteries is directed toward the ovarian pole, the other toward the cervix. They are connected, as shown at 8 in Fig. 20, by the inter-arterial anastomotic artery. Hence the rupture usually occurs in one of these two weak areas. If the non-gravid horn is first to protrude, it may be broken off at the parturient rent and the entire gravid cornu be retained out of sight. Or if the base of the gravid cornu has also become detached, the latter may break in two at the allantoic navel and its ovarian half be retained. If the gravid chorion first becomes detached, and is grasped by the cow, the rupture usually occurs at the cervix, rarely toward the center of the non-gravid horn, and the fragment is retained at the ovarian end. Thus the retained placental fragments, as in the mare, are hidden from view and tend to escape detection. In such failure of diagnosis lies the great danger.

The authentic proof of complete retained placenta, the protrusion of the navel cord, with portions of the membranes, is not always present in the cow. The navel cord is so short, and may break so near the allantois, that the entire mass of membranes may be hidden, but this rarely occurs. The failures in diagnosis are usually due to the cow having broken off, and eaten the protruding parts. The retained portions then undergo decomposition, with great peril to the uterus and to the breeding life of the cow. The most usual evidence of such fragmentary placental retention is the presence of that form of genital discharge in dairy cows which is commonly attributed to mild endometritis, usually revealing its presence by the escape of a few ounces of pus when the cow is recumbent. Hence only a minority of cases may be recognized. The phenomenon is probably of far greater importance than has yet been realized. Thus, in the amnio-allantoic dropsy illustrated in Figs. 181 and 182, no history of retention of the placenta of a prior gestation was available. How-

ever there had been complete destruction of the caruncles in one horn, and by far the most frequent basis for this is retained placenta. So, in the case of extreme chorionic dropsy already mentioned, extensive necrosis of the caruncles had occurred. The dropsy of the chorion occurred in the second pregnancy, the cow had not changed hands, and the owner was intelligent and alert, but no history of retained placenta, metritis or sloughing of uterine caruncles was obtainable. The autopsy revealed the indisputable fact.

So far as I can see there are but two means by which the existence of such hidden retained placentae may be diagnosed early and authentically—the careful examination of the membranes after their expulsion in order to determine that they are complete, or the manual exploration of the uterine cavity at an early period. In herds where genital disease is prevalent, such examination should be regularly and faithfully conducted.

The many hypotheses regarding the etiology of retained afterbirth hark back to the era when superstition reigned supreme. The advancement of veterinary science has not yet fully annihilated tradition. Writers upon veterinary obstetrics commonly mention as an important, if not the most important cause, the atony of the uterine walls and the absence of *after-pains*. The need of contraction of either uterine or abdominal walls for the expulsion of the fetal membranes may well be challenged. I have observed the equine fetal membranes completely expelled while the fetus, probably alive at the onset of labor, remained within the naked uterus. The uterus could not have contracted greatly in size while the fetus was retained. The placental arrangement in the ruminant excludes any direct action of either the uterine or the abdominal walls upon the detachment of the placentae. The uterine caruncles are said by histologists to be devoid of muscle fibers, and hence unable to contract actively. The caruncles stand up from the musculosa of the uterus in such a manner that, however much the uterine walls may contract, they only bring the caruncles nearer to each other and can exert no detaching force upon the fetal placentules. During physiological birth the allantochorion becomes detached about the cervix and, when the fetus is expelled, portions of the membranes, including the navel cord and part of the amnion (all of the amnion in the mare) hang outside the vulva, the weight of which serves to draw out other portions as they become detached. The last placentae to become detached are those at the ovarian pole of the pregnant horn, in which no part of the fetus lies. It is this progressive detachment of the placentae, from the cervix to the ovarian pole, which protects the fetus against asphyxiation during birth. The blood supply from the

base of the pregnant horn is cut off when the placentae of that area are separated to permit the first "water-bag" to pass through the cervix and vagina, and the blood supply from the non-gravid cornu is probably largely or wholly cut off by compression of the allantoic artery of that area by the presenting parts of the fetus. So physiologically, and usually also pathologically, the placental dehiscence begins at the cervix and ends at the ovarian pole. If uterine contraction caused placental detachment, the placentules at the ovarian pole of the gravid cornu, containing no part of the fetus, would be the first to be detached, and the fetus would be asphyxiated. Uterine atony is undeniably present in most, if not all cases of retained afterbirth. Violent straining is sometimes present, but it is not known that the uterine walls are contracting; certainly such straining is pathological. It may be due to inversion of the apex of the gravid cornu, to vaginitis, to injury to the bladder, etc. Pomayer[158] believes that the chief cause of uterine atony is prolonged dystocia. This hypothesis is readily reversible and it may be said even more logically that, when there is present placental disease which foreordains retained afterbirth, parturition or abortion is difficult and prolonged, and, when the fetus has finally been expelled, the already atonic uterus has been further damaged. Thus in the herd mentioned above with 73.3 per cent positive to the agglutination test for *Bacillus abortus*, all births were slow. Six of the calves died young. Of these 50 per cent of the dams were positive and 50 per cent negative. The average duration of retention for the three positives was 21 hours and for the negatives 27.5 hours. The negative cows suffered more from retained afterbirth than the positives. The evidence in this small group indicated that other infection than *Bacillus abortus* was present and virulent.

Various accidents are said to cause abortion and retained afterbirth. Thus Pomayer[158] reports histological studies of retained afterbirth in a cow destroyed three days after having aborted twins. Pomayer attributed the abortion of the twins to the cow's having been gored three days previously. Pomayer failed to record any signs of traumatism at the autopsy, but considered the fact that the cow had been gored sufficient proof that it had caused abortion. It has been claimed on preceding pages that twin ovulation and twin pregnancy are pathological and that most twin gestations in cows end in abortion, premature birth or retained afterbirth. The cow need not be gored in order that the pregnancy may end in disaster. Unimportant physical injuries are invoked to explain a wide variety of diseases and abnormalities of the intra-uterine young.

It is frequently stated that *Bacillus abortus* infection is the chief

cause of retained afterbirth. This was based upon the belief, following Bang's discovery of the bacillus, that all cattle abortions due to or associated with infection, were caused by the one organism. Since abortion during late gestation is largely followed by retained placenta, it was concluded that *Bacillus abortus* was the chief factor. The claim that *Bacillus abortus* infection is the most important cause of retained placenta in the cow loses much of its force when it is shown that both abortion and retained placenta abound in herds negative to the agglutination test for the abortion bacillus. Udall, Cushing and Fincher[201] record that in the Ambulatory Clinic of the New York State Veterinary College, in an area heavily infested with Bang's disease, 73 per cent of the cows having retained placentae were negative to the agglutination test and 23 per cent positive. Search of the retained membranes, from the positive reactors, revealed the presence of *Bacillus abortus* in 44 per cent of the cases studied. The report of Udall, Cushing and Fincher is representative of the available statistical studies.

The lesions of retained afterbirth advance by imperceptible gradations from those which drop away within twelve to twenty-four hours, followed by the appearance of rapid recovery, insignificant genital discharge and little interference with reproduction, to virulent septic metritis as already described. The retention, or rather the abnormalities which lead to the retention, early causes injury to the placental layer of the caruncles as in Fig. 172, or the total destruction of the caruncles as indicated in Fig. 182. The necrosis may go yet one step further and completely destroy the endometrium, leaving a purulent fistula as the sole evidence of a prior uterine cavity, as shown in Fig. 185.

The prognosis varies with the lesions. Some cows breed comparatively well afterward; far more breed hesitantly. They are frequently absolutely sterile on one side, where the lesions were most severe, and weakly fertile upon the other side. Many are totally sterile. Many die. The permanent sterility is apparently largely due to salpingitis (including pyosalpinx and adhesion of the pavilion to the ovary). It is one of the most destructive types of genital disease in cattle.

Retained placenta in dairy cattle is largely preventable by the application of conservative methods of breeding, feeding and handling. Twin pregnancy, so ruinously followed by retained afterbirth, can be largely excluded by avoiding the errors which lead up to twin ovulation and pregnancy, already discussed. Retained afterbirth prevails after amnio-allantoic dropsy. This is largely preventable by avoiding the errors in mating which lead to the dropsy. Thus it has

been stated, while discussing the hybridization of the American bison bull with the domestic cow, and the forms of mating which lead to *bulldog calves*, and to *muscle contracture monsters*, that the pregnancy largely leads to amnio-allantoic dropsy. These errors are readily evaded.

Cows bred at 30 to 60 days post-partum largely retain their afterbirths, as indicated in Tables XI and XII and the contiguous text. This is readily controllable. The pregnancies of the bull whose genital organs are shown in Fig. 139 were uniformly abnormally abbreviated and followed regularly by retained afterbirth. The slaughter of the bull terminated the defect. At the opposite extreme, the pregnancies of Bull X, Table VIII, were abnormally long and retained afterbirth highly destructive. The retained afterbirth ceased only after the bull and all his progeny had been discarded. When the gestation periods of the cows bred to a given bull mostly fall outside the optimum duration of 279 to 288 days inclusive, whether above or below, retained afterbirth, according to my observations, will certainly be highly destructive. In a herd with two or more sires, if the pregnancies of one are of abnormal duration, while those of the others are normal, the cause and remedy become clear. If a single sire is in use, the examination of the stained spermatozoa is helpful in determining whether the sire or the cows are responsible.

The heifers in Table I were the progeny of diseased dams. They were very unhealthy as calves and their reproductive systems were defective when they began to ovulate and show estrum. They were then granted but a few months for imperfect restoration to vigor when they were bred at fifteen months in an effort to have them calve at two years. Abortion, retained afterbirth and other disasters abounded. The causes related are definitely subject to control, and the retained afterbirth avoidable.

The heifers shown in Table X were largely from weak cows, were imprudently fed during their first six months, and nine months later were bred. Since they were immature and genitally hypoplastic, retained afterbirth logically was rampant and destructive. When the feeding plan was revolutionized, and the heifers were not bred until two years old, retained afterbirth disappeared as a major peril.

When the subject is carefully studied from various viewpoints and the fundamental causes learned, the phenomenon is preventable: it is one of the most readily and successfully preventable diseases in the field of genital pathology.

In an important sense *retained afterbirth is incurable*. When the disease becomes identifiable, owing to the termination of pregnancy,

placental lesions have already become established in the uterine caruncles, which remain throughout the life of the female to interfere with reproduction according to their extent and character. No method of treatment can operate retroactively to restore destroyed placental tissues.

Fortunately the placental area is so generous in extent that, after destruction of important portions of the placental tissues, the remaining areas are competent to sustain reproduction in varying degrees of efficiency. In ruminants also the inter-caruncular endometrium is capable in an emergency of developing adventitious placental tissues with some power for the nutrition of young.

Timely and prudent interference may halt the disease in its course and salvage a large measure of breeding power, may largely protect the milk yield and, the fertility failing, may greatly enhance the salvage value of the animal for slaughter.

Few diseases known to veterinary science have bred more "cures" and conflicts of opinion. It is one of the ancient diseases of history. Harms[80] cites literature upon the subject antedating the Christian era. He cites one writer at about 300 A.D. who advised as a cure a handful of fennel steeped in water, the water mixed with olive oil and wine and poured into the nostrils. The cure seems doubtful now, but many other remedies introduced during the sixteen intervening centuries are perhaps of no greater value. The principal points to be considered in treatment are:

1. *The manual removal of the afterbirth.* Some advise the removal in six to twelve hours, while others would wait one to five days and yet others would not remove the membranes at all. Pomayer[158] cites Hering and others as advising the injection of cold water into the umbilical veins and expecting this to result in their timely expulsion. The early manual removal of the membranes—when actually removable—opens the way for highly optimistic statistics, because the plan includes so many which do not need removal. Mention has been made above of a cow in which the membranes lay in the uterus as an inert putrid mass with the uterine caruncles completely detached from the uterus and firmly attached to the decomposing membranes. No one would advise against their removal at the time of their discovery. I was incidentally in a herd in which an esteemed colleague was the attending veterinarian. His rule was to abstain from handling retained placentae until two days had elapsed. I was shown a cow which had calved about one day previously. Both cornua of the allantochorion were hanging from the vulva, a few cotyledons were held by caruncles in the base of the gravid horn, and the weight of the membranes had dragged the cornual base out through the vulva.

I violated my colleague's rule by detaching the adherent caruncles, just as he would have done, had he been present. There are numerous cases in which prudence dictates the prompt removal of the membranes. Each case determines the date at which the membranes should be removed.

It is not always clear what a writer means by the manual removal of the fetal membranes. Ideally, removal should signify the complete withdrawal of the chorionic placental tufts, Fig. 46, from the placental crypts of the uterine caruncles. As already stated, placental dehiscence physiologically begins at the cervical end of the cornu, and normally the final separation occurs at the ovarian pole of the gravid horn. In the vast majority of cases this area constitutes the focus of peril, and any course of handling needs have as its ultimate goal the freeing of this area from all dead tissues and pathological contents. There is no virtue in removing the membranes from the other parts and leaving them behind at the ovarian pole of the gravid cornu. As may be seen in Fig. 14, the apex of the gravid horn constitutes the most dependent point of the uterine cavity, into which decomposing membranes or pathological excretions drop by gravity and from which the atonic uterus is powerless to expel them. Here they decompose, tending to cause necrosis of the uterine caruncles and other portions of the endometrium. The infection frequently extends into the tube and causes salpingitis. Spermatozoa cannot usually pass the oviduct. If they do so there is no opportunity for nutrition of the ovum, and it perishes. The cow is incurably sterile upon the affected side.

If therefore the membranes cannot be completely removed at this point, their removal from other areas is open to serious criticism, especially if the membranes toward the cervical pole are torn off and no longer exert feeble traction upon the apical portions.

Sometimes, instead of the withdrawal of the chorionic tufts from the crypts within the uterine caruncles, the chorion is torn away about the periphery of the caruncle, leaving the placental area firmly fixed to the caruncle. It is difficult, if not impossible, to show that such handling is in the least beneficial, or that it does not add materially to the already established peril.

My course during recent years has been to make a careful manual examination within one day after parturition and remove the membranes if I find that the chorionic placentae can be completely detached without injury to caruncles. In such cases, most of them would drop away spontaneously not long afterward. The possible advantage in their removal is a somewhat earlier elimination of the decomposing mass and opening the way for direct handling of the

diseased uterus. If it is found impracticable properly to remove the membranes at the first visit, the examination may be repeated at intervals of not less than one day, according to economic conditions, until the membranes drop away or are removed. The method of removal is simple. The operator, grasping the protruding membranes with one hand, exerts gentle traction upon them. The other hand is passed into the uterus until the area of attachment is reached. Each caruncle is grasped between the thumb and index finger, pressure is exerted at the periphery along the line of demarcation between the two structures, and the chorionic placenta gently detached. While acting as resident veterinarian I have repeated my examinations every day or two for five to ten days before it was possible to remove the afterbirth, when frequently the caruncles, having become necrotic, came away with the membranes.

There is unnecessary confusion regarding the removal of the uterine caruncles. If they are already detached from the uterus, there is no question regarding the desirability of removing them. When mention is made of manually detaching them, it sometimes arouses supercilious criticism. As in the separation of the placentae, so in the removal of the caruncles, it is a question of prudence in each case. I once attended a cow for retained afterbirth at about two days post-partum. The highly intelligent student accompanying me, who under my directions attempted the manual removal, was apparently making good progress, when to my amazement the first portions of the chorion coming through the vulva bore the firmly attached uterine caruncles. Immediately making an examination, I found that the caruncles dropped off without the exertion of any material force, unaccompanied by laceration, pain, or hemorrhage. The operation was continued, and the cow made an uneventful physical recovery. I have met with no other such case. Had they not been removed, they would have spontaneously dropped off a few hours later and, attached to the membranes, would probably have remained within the atonic uterus until manually lifted out. Within four to five days post-partum it is not at all rare to find, when attempting to remove the membranes, that some or many caruncles have become detached from the uterus and remain attached to the membranes. In such cases other cotyledons drop off when the operator attempts to detach the chorion. While this foretells the probable sterility of the cow, it is not highly perilous to her life. It indicates generally that the uterine walls are reacting rather favorably and establishing an effective barrier against mortal sepsis.

At times writers mention with well merited scorn the accounts of removal of the caruncles by force; it is equally unprofessional in

some cases not to remove the caruncles. The afterbirth may fall away spontaneously, or be removed in a proper manner, and the uterine caruncles later become necrotic and drop off to be retained in the atonic organ. This I have seen occur after the cervix had contracted. The necrotic uterine caruncles drop down into the apex of the involved horn and become surrounded by thick, purulent debris. The necrotic masses cannot be removed by the usual method of douching. In my experience it is advisable to introduce down to the ovarian pole of the cornu, a soft rubber catheter, with a single fenestrum, preferably the long catheter used for douching the sheath of the bull. A pint or two of warm, physiological salt solution is then introduced, after which the end of the catheter is compressed, lowered, and the liquid contents siphoned out. When most of the liquid has been siphoned out, the catheter becomes blocked by one of the decomposing caruncles. The distal end of the catheter should then be closed by compression and slowly withdrawn. The vacuum within the catheter retains the decomposing caruncle in the fenestrum of the instrument, and the necrotic mass is withdrawn. It is a tedious task when a large number of the sloughed caruncles are present, but the method has proven successful in my hands.

The sloughed caruncles do not always undergo rapid decomposition. One heifer without noticeable genital discharge, and from which the membranes had presumably fallen away promptly, completely surprised me by appearing on the twentieth day post-partum with a compact mass of a pound or two of desiccated caruncles in her vagina and more in her uterus. In another case, in which the afterbirth had presumably been entirely removed, rectal palpation revealed a month later two or three caruncles lying free within the uterine cavity.

The manual removal of the afterbirth involves important questions of professional tact. Many, if not most dairymen think that a competent veterinarian can remove the afterbirth of any cow at any date post-partum; the intelligent practitioner of experience knows otherwise. He can tear the afterbirth in pieces and remove some of the fragments; he can tear away the uterine caruncles, or otherwise mutilate the uterus. He can remove the afterbirth in a large ratio of cases if he conservatively awaits the period when the chorionic tufts may be withdrawn from the caruncular crypts. He should so advise his client, as tactfully as the conditions will permit.

2. *The artificial stimulation of uterine and abdominal contractions.* Much has been written in favor of the artificial stimulation of the uterine and abdominal expulsive efforts as a remedy for retained afterbirth. Important questions immediately arise.

It has been stated above that, in those cases where retained pla-

centa is to occur, there is present during pregnancy some degree of placentitis, associated with uterine atony which causes dystocia fundamentally attributable to the metritis, although popularly ascribed to dorso-ilial position, to the retention of the head or a limb, etc. It is not clear that it is advisable to attempt to stimulate to action a seriously inflamed organ. On the contrary it is generally advised that an inflamed organ be permitted to rest. Mention is made above of a virulent outbreak of puerperal sepsis with retained fetal membranes. It would appear to be sheer folly, if not worse, to attempt to stimulate uterine contraction in such cases. Among the various agents which have been advised for this purpose, one of the oldest is *Secale cornutum*, or ergot, which was long esteemed for its ecbolic qualities, though experience has caused it to be generally abandoned in veterinary obstetrics. More recently pituitrin has been advocated, but its value has not been clearly established. It has largely been used early after the expulsion of the fetus, inevitably including those cases in which the membranes would spontaneously drop away if left alone. Other hormone preparations have been advised, but their value has not yet been authentically determined. Late in the course of the disease, when necrotic caruncles are being cast off and effective leucocytic barriers against further bacterial invasion have been thrown out, there is some reason for the hope that hormone preparations may possibly prove beneficial.

Closely allied to the use of hormones is the dislodgment of the corpus luteum of pregnancy in the cow. Here also it is extremely doubtful whether good may be accomplished, or even danger be avoided, if it be dislodged early. Later, when involution is well advanced or when pyometra has become established, the dislodgment of the corpus luteum usually causes the ripening of an ovisac and the advent of estrum, which effectively empties the uterus of abnormal contents. The dislodgment of the corpus luteum is generally superior to the use of hormones. Some advise massage of the inflamed uterus per rectum, but it remains to be shown that this is of actual benefit.

One important fact regarding the value of arousing expulsive contractions of the uterine and abdominal walls has generally been overlooked or ignored. The principal concern is with the cow, in which the placentae are multiple and caruncular. Histologists teach that the caruncles, standing up in mushroom-like columns, have no muscle fibers. Consequently, contraction of the uterine walls causes no contraction in the walls of the placental crypts and is powerless to exert any direct expulsive force upon the placental tufts of the chorion. Admittedly the detached afterbirth may be expelled by uterine con-

tractions, but this is superfluous in the mare and cow because the weight of the protruding portions suffices to pull the remainder out. In ideally healthy birth, involution of the uterus is prompt. The fetal placentae drop away from the uterine crypts and the healthy uterus expels, in so far as needed, the *detached chorion,* but it is not the office of the muscular wall of the uterus to *separate* the fetal, from the maternal placenta. The unknown force, which has held the fetal and maternal placentae in contact, ceases and the two structures fall apart. Upon the other hand there are sometimes violent expulsive efforts due to some painful disease of the vagina or cervix, or owing to the invagination of the ovarian pole of the gravid cornu. Such pathological expulsive efforts have no tendency to cause the expulsion of the fetal membranes.

3. *Douching the uterus.* So long as the membranes are retained, the uterus cannot be effectively or safely douched. During the era when douching was highly popular I caused rupture of the uterus in one or two cases by the mere weight of the fluid in the atonic uterus. The disaster occurred in cows already doomed, but the occurrence looked exceedingly bad. The atonic uterus may be unable to expel the water, or, should expulsive efforts occur, portions of the membranes may block the cervical canal and the severe contractions seriously injure or rupture the uterus. At best the fluid largely remains in the uterus, to constitute a highly favorable medium for bacterial growth.

After the membranes are out, the washing of the uterus appears to have definite value. There is, however, no need for the introduction of large volumes of water. I prefer to introduce, by gravity, not more than a quart of warm physiological salt solution with the long bull-douching catheter. The catheter is gently introduced to the ovarian pole of the gravid cornu, the solution permitted to flow in, the distal end compressed and lowered, and the water siphoned out. The process may be repeated immediately until the siphoned fluid returns clear. Some prefer large volumes of water to distend and reach every part of the uterus. As already indicated, the ovarian pole of the gravid horn is almost invariably the danger focus, and when it is emptied the entire uterus is comparatively clean. The plan offers no violence to the uterus, and the siphon removes any exudate more effectively and safely than large volumes of water.

4. *Disinfection of the uterine cavity.* So long as the afterbirth is retained, disinfectants cannot be accurately applied to the uterus itself. Some fluid may be introduced between the chorion and the uterine walls, but this does not reach the focus of the disease, the caruncles, to which the cotyledons are adherent. When the membranes have been partly or wholly removed, directly effective disin-

fection remains impossible of accomplishment. As Pomayer[158] and others insist, the superficial layers of the endometrium are far more readily killed than are bacteria, and the use of ordinary disinfectants can only be regarded as dangerous.

The use of feebly soluble disinfectants is apparently justifiable. My experience leads me to believe them valuable. While not technically a disinfectant, some have found animal charcoal, in gelatin capsules, of marked value. It may be introduced into the cavity of the retained chorion, into the utero-chorionic space, or into the uterine cavity after the removal of the membranes. It is insoluble, non-irritant, enmeshes bacteria and, some say, their toxines, absorbs gases, etc. A wholly harmless substance, it may be introduced in any volume.

Feebly soluble disinfectants, such as bismuth subnitrate, iodoform and others, may be introduced, suspended in mineral, olive or raw linseed oil. The oil serves as an emollient and the bismuth and iodoform, because of their high specific gravity, remain in the cornual apex and dissolve very slowly. The iodoform is impracticable in many dairies where the cows upon which it may be used cannot be effectively isolated. The milk is repulsively flavored for seven to ten days. Instead of iodoform, thymol iodide may be used, which does not impart a disagreeable flavor. The iodoform taint does not injure the milk as food for calves.

Retained afterbirth tends constantly to be continued by chronic endometritis or other types of disease interfering with reproduction. These should be handled along the general lines already advised.

If it is decided to attempt to re-breed the cow after retained placenta, it should be definitely borne in mind that the greatest remedy known for diseases of the reproductive system is sexual rest. The natural tendency of the owner is to strive to have the cow become pregnant again at the earliest possible date. Most retained afterbirths follow abortions, stillbirths, abbreviated pregnancies, dystocia and other phenomena which depress lactation, lowering the milk yield and abbreviating the duration of lactation. The retention of the afterbirth for a day or more constitutes definite proof that the cow has been unable to sustain safely the burden of reproduction. It is therefore imprudent to ask her to assume quickly the burden of a new pregnancy, when it has been shown that her prior gestation constituted an excessive load under which she faltered. According to my experience and the available data by others, the rest following retained placenta should be at least six months. Cows not economically worth that degree of rest should be discarded.

Chapter XXXII

PARTURIENT INJURIES

Rupture of the Uterus

RUPTURE of the uterus during labor may be variously caused. Human obstetrists describe a type of dystocia unknown to veterinary science, termed retraction, or ring dystocia, in the presence of which the uterus may rupture because of the violence of the contraction. There is some conflict of opinion among human obstetrists. According to Pride[160] and others there sometimes occurs a retraction or constriction ring, at the juncture between the upper and lower uterine segments, which so interferes with the expulsion of the fetus that the uterine walls rupture. The reason for the occurrence of this phenomenon in woman, and its absence in domestic mammals is not clear. It is probably due to the difference in posture. The upright posture of woman, with the fetal head resting against the lower uterine segment, compressing it against the pelvic walls, naturally tends to cause some degree of atony of the lower segment, or at least fails to stimulate its development. The quadrupedal position of domestic mammals results in constant tension, instead of compression of the vagina and cervix, and results in their extensive development. Another factor probably playing a part is the form of the uterus. Since that of woman is pyriform, contractions at the fundus may push the fetal head or other part against the side of the uterus, while the tubular organ of domestic animals, with the great length of the fetus compared with its transverse diameter, tends to keep the long axis of the fetus parallel with that of the uterus. I have not observed, and have found no accurate record of rupture of the uterus owing to direct uterine contraction, in any species of domestic animal. It may occur. Rupture is found in torsion of the gravid uterus and, not rarely, in dystocia when forcible traction is applied. I have ruptured the cervix and cervical end of the uterus when the cervix was sclerotic from disease. I have also ruptured the uterus when attempting to extract the fetus in the rotated bicornual pregnancy of the mare, as described in *Veterinary Obstetrics*.

Rupture of the Perineum

Rupture of the perineum, observed most frequently in the mare, is usually due to deviation of the head or the upward deviation of one or both fore feet. In the former the traumatism is due to the excessive volume of the presenting part, especially when forced ex-

traction is applied. The rent is usually enormous, with the anus, the vulva and an extensive area of the vaginal roof and the rectal floor torn asunder, leaving a vast cloaca, through which the feces fall into the vagina. In the second type, one or two of the feet of the foal become directed upward against the sacrum, puncturing the walls between the vagina and rectum, and protruding through the anus, while the head passes through the vulva, tearing asunder the intervening structures. If the accident is discovered early, the protruding foot may be repelled and the fetus extracted through the vulva, leaving behind a recto-vaginal fistula. Unfortunately the mare usually survives.

I have repeatedly observed a third type of perineal rupture in heifers. Owing to the frequency of genital hypoplasia due to imprudent feeding and handling of calves, the vulva and vagina may be absolutely too narrow to permit the passage of a normal fetus without tearing them asunder. So in the herd which I have described, in conjunction with Table X, the hypoplasia was so prevalent and extreme that the fetus could not be expelled. The application of severe traction caused such extensive rupture that several heifers perished from hemorrhage. Two survived, with complete rupture of the perineum and extensive involvement of the rectum and the vagina. One of these again conceived and calved. The injury is usually beyond repair. When due to genital hypoplasia, the breeding outlook for the heifer does not justify handling. In well selected cases of animals of high potential value, surgical handling may be advisable. This is described in the companion volume, *Veterinary Obstetrics*.

Lacerations and contusions of the vagina are inevitable in cases of genital hypoplasia in heifers; in the extraction of large mummies, or dry, emphysematous fetal cadavers; and from rough work by laymen in cases of dystocia. Later these may lead to atresia of the vagina. If the vaginitis is severe, cystitis may develop due to the invasion of the bladder through the urethra.

Traumatic hemorrhage sometimes occurs, but is only rarely dangerous. I saw one puerperal mare with contusion of the vulvar labiae followed by a large hematom. When the attending veterinarian opened the sac severe hemorrhage followed which was difficult to control.

Contusion of the gluteal and obturator nerves, as described in *Veterinary Obstetrics*, occurs not infrequently in the mare, causing paralysis and atrophy of the muscles supplied by them. The contusion is most probably caused by the impingement of the nerves between the sacrum or the ilium and the fetal cranium. The same injury possibly occurs in other species.

Chapter XXXIII

PUERPERAL DISPLACEMENTS OF THE URINO-GENITAL ORGANS

IN the companion volume, *Veterinary Obstetrics*, the various puerperal displacements of the uterus, vagina and urinary bladder have been described. It seems desirable to point out here that considerable confusion exists among writers regarding inversion and prolapse of the uterus. The phenomena of inversion and of prolapse, or eversion, are two stages of one displacement. The ovarian pole, usually of the pregnant horn, becomes inverted, as the tip of the finger of a glove. When the inversion so extends that the displaced organ protrudes through the vulva, it is designated as eversion or prolapse. Prominent authors state that as a rule only the pregnant cornu becomes inverted or prolapsed. They apparently ignore certain anatomical facts. The uterus of the cow, as shown clearly in Fig. 14, and yet more definitely in Fig. 187, is a uterus didelphys with a single cervix. The two cornua are firmly bound together throughout most of their length by the intercornual ligament, 8, Fig. 187. The free ovarian poles, 9, 9, may either—or theoretically both—become inverted as far as the ovarian border, 8, of the intercornual ligament. If the ovarian pole of the right horn becomes inverted, the inversion can extend independently only to the anterior border of the intercornual ligament. Its further inversion necessarily involves the left cornu, which must become telescoped into the right horn. It then follows, as an anatomical necessity, that in the cow (and other ruminants) extensive inversion of both cornua is impossible, and, when the one cornu becomes prolapsed through the vulva, the other cornu must accompany it, not inverted but enclosed within the everted cornu. The double uterus of multipara, Figs. 16 and 18, without intercornual ligament, permits one cornu to become prolapsed unaccompanied by the other.



FIG. 187. Horizontal Section Through the Genital Tube of the Cow to Show the Didelphic Type of Uterus.

1, The lips of the cervix; 2, the os uteri internum; 3, 3, narrow bands of the dorsal walls of the uterine cornua left uncut to indicate the breadth of the communication between the two cornua; 4, 4, a strip of black fabric extending from the base of one, into the base of the other cornu, between, 2, and, 7, showing the area commonly designated as "the body of the uterus"; 5, the cavity of the right cornu laid open along the mid-dorsal line and exposing the uterine caruncles in four rows; 6, the cavity of the left cornu; 7, the vaginal end of the intercornual ligament. *In order to retain the relations and to define the borders, the peritoneal and endometrial layers have been sutured together by overcasting with black thread;* 8, the anterior border of the intercornual ligament; 9, the free ovarian poles of the cornua.

Chapter XXXIV

PUERPERAL DISEASES

Intrauterine Hemorrhage

HEMORRHAGE into the puerperal uterus may occur in any domestic animal, due to trauma from parturition or from rough efforts at the removal of retained placenta. Such hemorrhages are usually of scant significance, except as they may suggest important wounds of the parts.

During the puerperium there occurs in the cow a far more significant profuse intrauterine hemorrhage, which is apparently identical in principle with the enormous hemorrhage from the caruncles constituting the basis for the hematic mummification already described. The hemorrhage is apparently due to the presence of a factor which destroys the equilibrium of the uterine vascular system.

The hemorrhage appears without warning soon after parturition. I have observed it at two to four days post-partum. My cases have occurred in cows in intensely infected herds. Each patient had given birth to a calf, but the parturition was tardy, testifying to the presence of uterine paresis due to the metritis of pregnancy. The fetal membranes were not long retained and the patients were apparently going along favorably, when suddenly without warning a very profuse uterine hemorrhage began. One of my cases had been left at night in a box stall, apparently well. When the caretaker arrived in the morning, the cow was weak from loss of blood and her stall looked like a shambles. Bright scarlet blood was spattered over the walls and bedding, and blood was still trickling from her vulva. A few hours later I found upon examination, as nearly as I could estimate, a blood clot in the uterus equal to two or three gallons. The blood was coagulated, odorless, and bright scarlet in color. Fearing recurrence, I proceeded to remove with great care a portion of the clot each day, until in three days I had completely evacuated the uterus. The blood maintained throughout its bright scarlet color, underwent no visible decomposition and acquired no specific odor. The cow recovered and was fertile. In a second case a colleague removed the blood from the uterine cavity, first controlling the hemorrhage by the use of adrenalin chloride hypodermically.

Later, in the herd shown in Table X, two cases were studied daily in heifers of low fertility associated with genital hypoplasia. It was observed that the milk of each of these was of a cherry-red color

until all the blood had been removed from the uterus. While putrid decomposition of the fetal membranes and purulent metritis were prevalent in the herd, the membranes came away promptly in each of these. There was no evidence, in any case observed, that the hemorrhage had been due to or associated with traumatic injury. I have not known of a fatality from the hemorrhage, which apparently ceases when the blood pressure falls.

It is highly significant that these immense hematoma frequently,

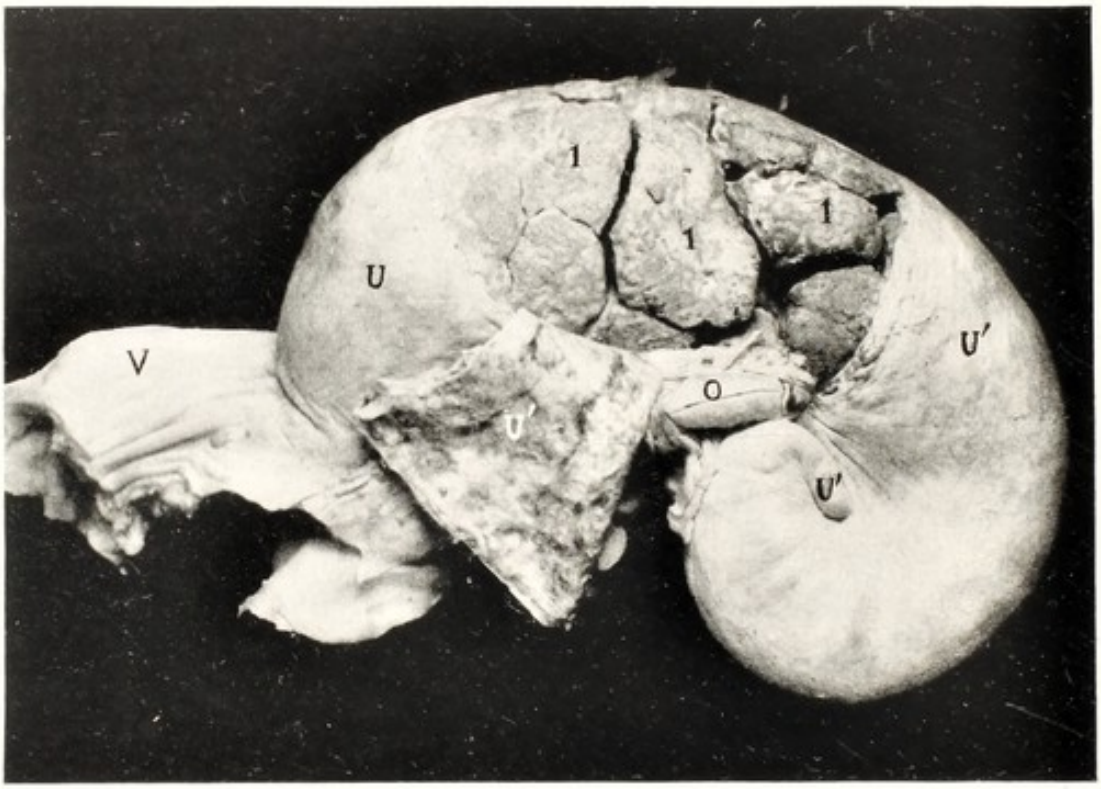


FIG. 188. Tesselated Intrauterine Hematoma of Cow.

(Compare Figures 177 to 180) V, Vagina; U, base of right uterine horn; *U'*, *U'*, ovarian pole of uterus; 1, 1, desiccated (mummified) blood coagula.

if not regularly, defy putrid decomposition, directly contradicting the oft-repeated hypothesis that in the *open* uterus putrefaction rages while in the *closed* organ the non-living tissues undergo mummification. In a series of 3000 uteri examined in the abattoir, I recognized three cases of desiccated (mummified) blood coagula in non-pregnant uteri, one of which is illustrated in Fig. 188. In all cases I have observed, the mummified blood clot has been in one cornu only, presumably that which had been pregnant. The hematoma, like the mummified fetus, remains indefinitely as an inert body, so far as known. Usually the desiccated blood-clot appears to exclude ovulation and estrum, like the mummy. In one of my abattoir cases, where the hematoma was quite small, and confined entirely to one horn,

an apparently healthy fetus three inches long was present in the other cornu. This was in conflict with the rule that in the presence of a foreign body—mummy, pus, etc.—ovulation and estrum are barred. When the desiccation of the hematoma is incomplete and soft, the black, tarry mass is extremely sticky. If an attempt is made to divide it, it is difficult to cut through the mass, because of the adhesiveness. Parts of the mass adhere to the knife and are very difficult to wash off. The blood clot is virtually insoluble in water, is without odor, and causes no visible irritation of the endometrium. Except for the one case in which pregnancy in the other cornu was present, the cervical canal was open.

The diagnosis is comparatively easy. In recent cases, after the fetus and afterbirth have been expelled for some hours to two or three days, bright scarlet blood is seen escaping from the vulva. The blood is brighter than that ordinarily following trauma, and the cow is not distressed. Vaginal exploration reveals an enormous mass of blood in the uterus. This may escape notice and the more or less desiccated hematoma may be discovered later while palpating the genital organs per rectum. The clot may then be doughy, if of recent origin, or hard, as in Fig. 188, if old. The hematoma bears no resemblance to pregnancy nor to pyometra. When the hemorrhage is active, it is presumably well to administer adrenalin, although, as stated above, fatal hemorrhage has not been observed. Within a day or two after the hemorrhage, my plan has been to introduce warm physiological salt solution and gently break up the clot manually, taking out only a third or half of the total, and repeating daily until all is out.

If the hemorrhage has passed unidentified and the coagulum has become desiccated, the logical method of handling would be the dislodgment of the corpus luteum, as in the mummified fetus.

Puerperal Tetanus

Puerperal tetanus is observed occasionally in the mare and cow, and is possible in any species. It most frequently follows uterine prolapse in the mare, because the endometrium comes in contact with infective bedding while the animal is recumbent. Tetanus sometimes follows retained placenta in the cow. When the cow is recumbent, the pressure upon the abdomen increases the protrusion of the membranes and causes contamination; when she rises, the membranes drop back into the birth canal and drag the infection with them. The disease offers nothing unusual in symptoms or handling compared with the ordinary tetanus resulting from wounds.

Pyometra

Pyometra may arise at any time in the genital life of the female. It has already been described as occurring during pregnancy, especially in bovine trichomoniasis. It also occurs in chronic metritis, as a continuation of puerperal pyometra, or it may arise in animals which have not been bred and in old-standing cases of sterility.

Economically, pyometra which becomes established during the puerperal period is of outstanding importance. Timely interference may prevent its becoming established and may avoid sterility. When pyometra becomes fully established and persists for a considerable time, fertility is generally closed.

Vague statements occur which tend to cause the inference that the pyometra of pregnancy, due to trichomonad invasion in the cow, may leave her fertile. The records are not clear. The exudate designated as pus in the trichomonad disease is unlike the usual purulent exudate associated with bacteria. Moreover, a careful study of reports fails to show clearly that cows which have suffered extensively from trichomonad pyometra retain their fertility. It seems that cows which have suffered from trichomonad infection, without notable pyometra, may sometimes retain their fertility.

Guarding against the establishment of pyometra during the puerperium is highly important. When dystocia or retained placenta is present, it indicates that utero-fetal disease existed during late pregnancy. Parturition almost certainly aggravates any metritis present. The dangers of the period may be largely evaded by careful attention for a few days post-partum. While discussing retained placenta it was noted that the non-gravid chorion and the cervical pole of the gravid membranes sometimes break off at the allantoic navel and drop away, leaving the ovarian pole of the gravid chorion retained. This is followed by necrosis of the uterine caruncles and the accumulation at the ovarian pole of pus and decomposing tissues. This, when neglected, may lead to pyometra and sterility.

Accordingly it is well to follow such cases closely, and empty the cornu of its putrid contents as early as possible. The presence of puerperal metritis and pyometra may be recognized early by siphoning out the contents from the ovarian pole of the cornu as already mentioned under "retained placenta." It is also identifiable by rectal palpation, which at the same time reveals the degree of uterine involution.

The establishment of pyometra should be opposed, especially in the cow, by repeated siphoning out of any accumulations of pus and tissue debris from the apex of the cornu where it collects. Later the corpus luteum of pregnancy may be profitably dislodged, causing

estrum and the emptying of the uterine cavity. Unless energetically handled, the disease passes into chronic pyometra or metritis and becomes an important cause of obstinate sterility.

It might be well to try, experimentally, in prudently selected cases, the removal of the corpus luteum of pregnancy, which in the cow usually becomes retained and quite firmly embedded. It would probably be impossible generally to remove by rectal compression. As a rule, it can not be reached per rectum, and were it reached, the dislodgment from the ovary would usually not be practicable by compression. It would not appear advisable to stab the ovary with the the ovarian scalpel, Fig. 43, lest infection occur through the vaginal incision. Apparently the safest method would be by laparotomy, after which the fibrous covering of the corpus luteum could be incised, and the body readily expressed. Or the containing ovary could be removed. If the cow has any breeding value remaining, it is quite certainly not on the involved side. Instead it would almost certainly improve the prospect of fertility by eliminating ovulation upon the involved side. This would confine the ovulation, and possible pregnancy to the better half of the uterus. This method of handling would be essentially experimental. The laparotomy in the cow is comparatively without danger.

Uterine Abscess

Uterine abscess occasionally follows puerperal disease, owing to atresia of the cervical canal from acute cervicitis. This may cause unimportant symptoms for a time, until the uterus becomes distended with pus to such degree as to cause pain and straining. A mare which I had attended in dystocia some weeks previously exhibited marked abdominal pain with expulsive efforts. Vaginal palpation revealed an enormously distended, fluctuant uterus with the cervix forced back almost to the vulva. The uterus was incised at the region of the cervix and several gallons of fetid pus were expelled. The mare made an uneventful physical recovery, but was inevitably sterile. I have observed uterine abscess in the cow only in slaughtered animals without clinical history. In one cow, general pelvic adhesions had occurred and the abscess had emptied into the rectum. Animals may recover their general health after the abscess ruptures or has been opened, but the breeding life is closed.

Puerperal Pyaemia. Pyaemic Arthritis. Pyaemic Abscesses. Puerperal Meningitis. Pyelo-nephritis

The pyaemic phenomena following puerperal uterine infection are without limit. Rarely, such pyaemic disease originates during gesta-

tion. While possible in all species, it is most frequent in dairy cows. The most frequent types are:

1. *Pyaemic arthritis* which is not rare in the cow and is observed in the mare. The most frequently affected articulation is the femoro-tibial or stifle. The lameness usually appears suddenly, weeks, or possibly months after retained placenta or other intra-uterine disease. The pain varies: the lameness may be moderate or may cause total disability of the affected limb. The diseased articulation is swollen and sensitive.

It is incurable, especially when involving the femoro-tibial articulation. It may be somewhat ameliorated by stimulating liniments, etc., and by freedom out of doors when the weather will permit.

2. *Pyaemic abscesses* occurring in various organs. They may appear in the articulations, in the liver, lungs or elsewhere. A mare which I had attended for dystocia developed a large abscess in the sublumbar lymph glands. It was readily palpable per rectum. When such abscesses are adherent to the rectum, they may readily be opened into that organ and a favorable prognosis given. If not adherent to the rectum, they may be opened from the exterior, but are difficult of approach because of their intimate relations with the iliac arteries.

3. *Puerperal meningitis*, which has come under my observation in a young cow in a herd reeking with genital infection. She calved without notable incident and appeared to be progressing favorably for about two days, when she suddenly developed symptoms of severe meningitis. The patient was exceedingly uneasy, with constant muscular twitchings. The head was held erect and the gait was rigid and uncertain. She was readily disturbed, as in tetanus. The course was stormy. Death occurred within one day. Autopsy showed intense injection of the meninges without notable uterine lesions.

There is little or nothing to be done, as the disease has usually passed beyond the realm of control before recognizable clinical warning has been given.

4. *Pyelo-nephritis* is growing in frequency in dairy cows under such environment as to suggest that it is pyaemic and largely the consequence of puerperal infection. It is characterized by general ill health and loss of condition, with frequent urination. The urine is sometimes bloody; at other times there is a discharge of fetid pus. It is incurable. In rare cases, when but one kidney is involved, its removal may be considered.

SECTION VII

THE DEFECTS AND DISEASES OF THE NEW-BORN

DISEASES OF THE NEW-BORN

WHEN the young mammal crosses the birthline from prenatal to postnatal life, it experiences abrupt changes in its environment. Some teratological young, such as acardiacus, *Schistosomus reflexus* and muscle contracture monsters, must immediately perish because the organs of respiration or circulation are devoid of function. Other defects, some of which have been mentioned in Section I, imperil the life or value of the young. Of far greater economic peril are those abnormalities arising during intra-uterine life which are carried across the birthline to invite or cause disease of the young. The new environment, into which the young is suddenly thrust, demands readjustment of virtually all body functions, and to this is added exposure to innumerable infections and other hazards.

Early post-natal life constitutes the most highly plastic era in the existence of the individual, during which the animal husbandman and veterinarian may profoundly affect its destiny: its probability of life may be raised or lowered; if it survives, its physical vigor and longevity may be strengthened or weakened; and its reproductive system may be normally developed or it may be arrested and its fertility depressed or destroyed. Ordinarily the health of the young is considered merely upon the basis of viability: if it survives, the handling is termed successful, although as an adult it may be worthless for reproduction and of low value for any other purpose.

Throughout this treatise it is attempted to view the diseases interfering with reproduction as an endless chain, stretching from the formation of the sex cells to the end of the life of the individual. Disease may be halted or created at any point, but in no era can any interference by animal husbandman or veterinarian operate retroactively to overcome arrests in development or destruction of parts by disease. The diseases and dangers surrounding the new-born are infinite. Only the more prevalent types may be considered in an effort to illustrate the principles involved. Teratological or congenital anatomical defects have been considered in Section I.

The transition of the young from intrauterine to extrauterine life demands abrupt and highly important changes in the physiological activities of systems and organs. Among the most outstanding of these are: the establishment of pulmonary respiration; the separation of the navel cord, with the discard of the fetal membranes and the readjustment of the circulation of the blood; the evacuation of the meconium from the alimentary system; and the oral intake of food and its digestion.

Chapter XXXV

ASPHYXIATION

THE asphyxiation of the fetus presents an extremely variable picture, according to the species of animal. In the mare, with diffuse placenta and remarkably stormy labor, parturition quickly ends in the birth or the death of the fetus. It has not fallen to my lot to deliver a living foal in cases of dystocia except in one instance. During twelve years of practice in a concentrated draft-horse-breeding district, where obstetrics constituted my major problem, I do not recall having delivered a living foal in case of dystocia. Since upon my arrival, I discovered no evidences of fetal life, in cases of dystocia, I cannot say that any fetus died during delivery. More recently, I served as resident stud veterinarian, where as a rule I had the mare under observation within fifteen to twenty minutes after the onset of labor. Under this environment, unfortunately for the interests of my client, I delivered the worthless living colt shown in Fig. 190. Physiological labor is usually completed by the dropping away of the fetal membranes within fifteen to thirty minutes after the expulsion of the fetus. In one case of dystocia the membranes had been expelled in toto before I was called to extract the fetal cadaver. The death of the equine fetus is generally attributed to asphyxiation. In the one unusual case in which the placenta was expelled and the fetus retained, asphyxiation might well be drawn upon to explain the death. The general hypothesis is that the placental dehiscence causes asphyxiation, but it is not known that the fetus was alive at the onset of labor.

The gravid cornua of the swine uterus, Fig. 18, attain a length of eight or ten feet each. According to Zietzschmann[278] the navel cord is about the same length as the fetus. When there are eight or ten fetuses in a cornu having a length of approximately one foot for each fetus, it is generally assumed by writers that the navel cord ruptures or the placenta separates before the fetus has travelled one foot. Hence the fetus at the ovarian pole, according to such hypothesis, must travel eight or ten feet in order to reach the vulva after the placental contact has been eliminated. This is merely a hypothesis. Apparently it is not known to what extent the uterine cornua shorten during labor. It is equally logical to assume that the cornua rapidly contract in length, and that the fetuses at the ovarian poles retain their placental contact until the amnion of the involved fetus is not far from the birth canal, through which it is expelled with

great rapidity. The healthy pig is presumed to be frequently expelled within its amnion, but the membrane is so frail that the young is rarely, if ever, asphyxiated. Hence the finding of a dead fetus enclosed within its amnion, suggests that the fetus was dead or mortally ill when expelled. The placental anatomy in the cow and other ruminants excludes the expulsion of the full-term fetus surrounded by the amnion alone, and it is rarely, if ever, expelled within the allantois and amnion. Aborts are frequently so expelled, especially in early pregnancy. Hence in domestic animals of all species, the fetus is rarely, if ever asphyxiated because of having been expelled within the fetal membranes.

Others invoke the inhalation of the amniotic fluid by the partially asphyxiated fetus as the immediate cause of its death. The healthy fetus rapidly swallows its amniotic fluid, and any lanugo, hair and other solids are filtered out, leaving the amniotic liquid a neutral fluid which may be rapidly absorbed before the fetus arrives at a point where its lungs might function. Pathologically the fetus may be born with large volumes of tough mucus in the pharynx and larynx, which may occlude the larynx and asphyxiate the young. This asphyxiation I have observed in pigs, deferred sometimes until several hours after birth.

Some writers have stated that, during fetal asphyxiation, the bowels are stimulated and expel the meconium into the amiotic sac. When one finds several gallons of diarrheic feces in the bovine amniotic cavity, the alimentary tract loaded with liquid meconium, and the fetal coat deeply stained so that the color is fixed in the hair, the cathartic power of asphyxiation appears amazing. If the fetus has not died within the uterus, diarrhea tends to continue unabated after birth, after respiration has become well established. It would also appear that, if fetal diarrhea is caused by asphyxiation, it should be far more common in the equine than in the bovine or ovine fetus. I have not observed diarrhea in the equine fetus, but have frequently seen it in bovine and ovine fetuses.

My experience has been largely limited to horses and cattle. The young are occasionally extracted, in dystocia, apparently lifeless except for heart beat. I have not observed the phenomenon in physiological birth. As a rule, they have died despite my efforts at artificial respiration. Sometimes the efforts at artificial respiration have been followed by recovery. Possibly most of them would have recovered spontaneously. The fact that careful study generally reveals a background of genital disease reduces the potential value of efforts at resuscitation.

Chapter XXXVI

SEPSIS OF THE NEW-BORN

WHEN serious placental disease has existed during gestation there not infrequently occurs a virulent septic condition of the newly-born young which largely results in its death. It is observed in all species of domestic animals and is usually recognized by the extreme weakness of the young, without notable symptoms of disease of a given organ or system. The phenomenon is most extreme in foals, chiefly in race horses, especially in concentrated breeding areas where, because of the sentimentality attached to certain mares and stallions, they are continued in forced breeding after they should have been withdrawn from the stud because of old age or genital disease. In my limited clinical experience with the phenomenon, it has occurred after prolonged gestation, usually exceeding 365 days, or 30 or more days beyond the optimum. All my observations have been upon colts, not fillies. So far as may be learned, such is the rule.

Although many, if not most foals suffering in this manner are carried far over time, they are usually very thin and under weight although they may be of normal length. Labor is usually prolonged unless prompt aid is available; the uterus is atonic and the fetus of very low vitality, which inevitably delays birth. So far as may be seen, the prolonged gestation and the atonic birth are due to one and the same cause.

In severe cases the foal is somnolent; when expelled it is virtually unconscious of surroundings, makes no effort to rise and, if helped up, neither tries to stand nor reveals any ability to stand. The mortality in such cases is fortunately very high. Some of them rally after one to several days, get up, and become apparently healthy and vigorous, as already related in one case.

There is no known remedy. So far as I have been able to determine, those which apparently recover cannot become useful animals. Since such foals are generally carried in utero far beyond the physiological limit, they fall under the dictum of von Oettingen,[153a] based upon a rich experience, that foals carried much over time never prove valuable as adults. The phenomenon is largely preventable by a respectful regard for the fundamental law of breeding hygiene, that only healthy animals, with adequate surplus of vigor beyond the needs for their physical life, be used for breeding purposes.

Chapter XXXVII

ANTENATAL AND EARLY POSTNATAL RUPTURES OF TENDONS AND LIGAMENTS

THERE occurs in foals an interesting series of ruptures of tendons, or rather of the spontaneous parting of the median end of the tendon from its muscle, and ruptures of important articular ligaments, or the separation of important tendons from their terminal insertions into bones. The lesions shown in Fig. 189 are not known except in foals. While tendons become ruptured from various causes in animals of any age, the distinctive type shown in this case is not known to occur except in the fetus or in the newly-born foal. It is not traceable to traumatism. In the foal shown in Fig. 190, where the rupture occurred prior to birth, the loss of control of the anterior feet led to dystocia. The most common lesion I have observed is the parting of the median end of the tendon of the extensor pedis longus muscle from the muscular body within its carpal sheath.

The lesion may exist at birth, or may occur or become obvious at one to ten days after birth.

The symptoms consist chiefly of a loss of power to extend the carpus. So far as I have observed, it is always bilateral. The foal may not be able to rise, but may stand after being helped to its feet. If unable to stand naturally, it may readily do so if the carpal joints be pressed backwards by the attendant's hands. Its movements are uncertain. The extensor pedis longus can exert no power upon the digits and the toe is advanced in a flail-like motion by the action of the chief extensor of the metacarpus. There is a baggy swelling upon the anterior surface of the carpus, due to distension of the sheath of the ruptured tendon. The rupture occurs, as shown in Fig. 189, at the superior portion of the carpus, and the ruptured end of the tendon, 2, drops down to the lower end of its carpal sheath, where it is readily palpated.

The lesions are not confined to the extensor pedis longus muscle and tendon. In Fig. 190, in addition to the extensor pedis longus rupture, the flexor pedis perforans of the posterior foot (deep flexor of the phalanges) was torn away from its insertion into the os pedis, or third phalanx. This caused the fetlock to drop to the ground and the toe to turn up as is sometimes seen in horses after neurectomy for navicular disease. A third phenomenon is shown in Fig. 191. Again the extensor pedis longus muscles have parted from their tendons at

the carpus. The extensive synovial capsule of the femoro-tibio-patellar articulation has become enormously distended in the three-day-old foal, and the patellae have been pushed away from the femoral trochlea. This has caused them to become dislocated far upward and the knee joint has been made functionless.

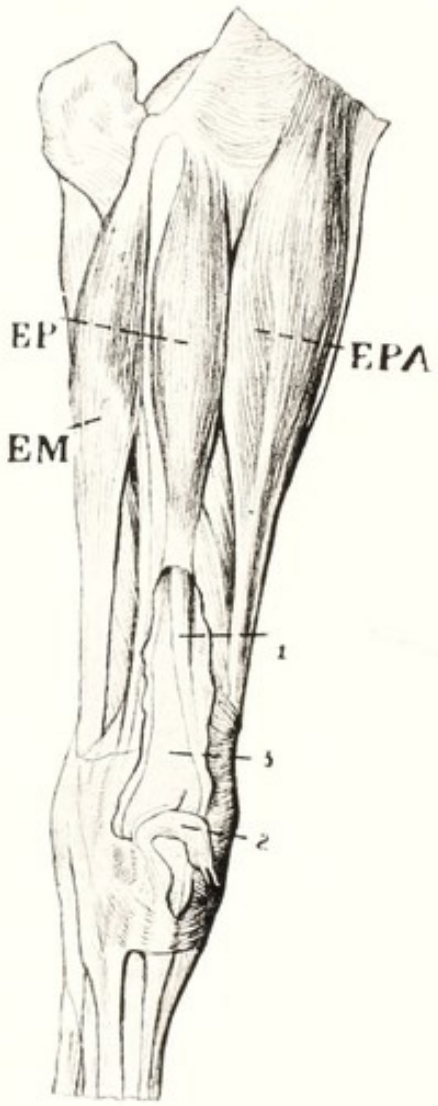


FIG. 189. Musculo-Tendinous Rupture of the *Extensor Pedis Longus* in Young Foal. Right anterior limb viewed from in front. *EM*, Extensor of the metacarpus; *EP*, extensor pedis longus; *EPA*, extensor pedis longus accessorius; 1, the distal end of the extensor pedis where tendon has parted from the muscle; 2, the upper or muscular end of the tendon which has been pulled out of the muscle and dropped down to the inferior end of the carpal sheath, 3.

The foal illustrated in Fig. 191 was born with posterior or ventral luxation of the metacarpo-phalangeal articulation of the right anterior foot. The phalanges were at right angles to the metacarpus. Under moderate pressure the dislocation was reduced, with an audible snap. The dislocation was readily renewed at will.

The cause of the disease is not known. It is possibly related to

the sepsis of the new-born already described, but there is no definite evidence. The foal shown in Fig. 191 appeared dull for several hours after birth, but did not clearly exhibit the somnolency so characteristic of acute sepsis of the new-born. The gestation was 380 days. As is the rule in such prolonged gestation, the fetal membranes were extensively diseased, showing engorged, edematous, calcified and necrotic areas in abundance. The navel cord was greatly enlarged, chiefly owing to an abnormally distended urachus, two inches or more in diameter at some places and filled with allantoic fluid.



FIG. 190. Foal born at 380 days with antenatal rupture of both *extensor pedis longus* muscles of the anterior limbs and the separation of the terminal attachment of the deep flexors of the posterior limbs from the third phalanges (Flexor pedis perforans. Flexor longis pollicis pedis of man). *Note the extreme emaciation of the foal, carried 45 days overtime. The carpal joints bend forward, being sustained only by the extensors of the metacarpus and the small accessory extensor of the third phalanx* (see Fig. 189).

Prevailing fashion might well ascribe it to nutritive deficiency, which may be made to cover the etiology of a vast assortment of diseases. A failure of nutrition was undeniable in Fig. 191; the foal, after a gestation period of 380 days was very thin, subnormal in size and its musculo-tendinous tissues frail. Its weight was at least 25 pounds below what it should have been after a healthy gestation period of 330 days. Without denying nutritive deficiency, the question arises concerning the cause of the deficiency. The imperfect clinical histories of my cases do not make the cause clear.

Fig. 189 is from a foal of a mare belonging to the Agricultural Col-

lege of Cornell University. Two ordinary grade work mares each dropped foals for two successive years, all four by a common grade stallion. All suffered alike from this disease. The mares were gently worked, and were well stabled and fed. They were managed by the professors of animal husbandry who taught students how to feed and handle horses and other domestic animals. Approximately forty years have elapsed since these four foals suffered from the disease. So far



FIG. 191. Rupture of the extensor pedis longus muscles of the anterior limbs with displacement of the patellae due to excessive distension of the femoro-patellar-tibial articular capsule. *a*, Patella; *b*, femoro-tibial articulation.

as I know, the phenomenon has not recurred in the greatly enlarged university stud, nor in the immediate vicinity. The foal shown in Fig. 190 was born and bred in the famous Blue Grass region of Kentucky, which, according to the breeders of fashionable thoroughbreds, supplies the most nutritious natural foods of any area in America or elsewhere. The mare was of famous pedigree and the sire commanded all the mares he could serve at $2,000 for the service. It is need-

less to state that the mare was well fed. There was no calcium deficiency in the soil, the limestone cropping out on the surface, heavily impregnated with phosphates. So, while the foal was badly undernourished, it was not because of any known deficiency in quality of quantity of food supplied to the mare. Finally the University of Kentucky, located in the Blue Grass area, has given special emphasis in both animal husbandry and veterinary science to the study of the breeding of race horses, and so far as known the feeding and handling of the mare were in harmony with the teaching of the university.

The prognosis is hopeless. One of the four foals so affected at Cornell University survived and could perform gentle work. It had defective control of its anterior limbs, and extended its anterior feet in a flail-like manner through the action of the extensor of the metacarpus. The animal was unsaleable except as a cripple. The foal shown in Fig. 190 survived and was sold for $100.00, which the buyer refused later to pay on the ground that he had been defrauded.

Chapter XXXVIII

IMPACTION OF THE LARGE INTESTINES WITH MECONIUM

THE large intestines of the fetus constitute a vast cesspool in which are stored up, for the duration of pregnancy, all those excreta from the fetal organs not readily evacuated by the placental route. If the fetus is physiological, no part of the meconium is known to be evacuated through the fetal anus. Ideally the meconium consists exclusively of non-toxic and non-irritant particles of tissue-waste, along with bile and possibly other excretions which presumably act to prevent decomposition.

Pathologically it is held by apparently reliable authorities that the uterine cavity of non-pregnant and pregnant females alike commonly harbor bacteria and other invaders, largely saprophytic in character, if the animal be in vigorous health. Presumably these parasites may penetrate the hurriedly formed fetal membranes, with their low powers of resistance, reach the amniotic cavity, be swallowed by the fetus and stored as a constituent of the meconium.

Such infections of the pregnant female as tuberculosis pass through the placentae and invade the fetal organs. It is claimed by most writers that *Bacillus abortus* is regularly swallowed by the pregnant female, reaches her blood stream, passes through the placentae, and finally reaches the fetal digestive tract and becomes a part of the meconium. According to the best available sources, the meconium of dairy calves commonly contains various bacteria. Apparently the meconium of foals has not been significantly studied.

When a new-born animal swallows milk, it also swallows bacteria. If the meconium is still in situ, any infections the milk may contain may multiply alike in both milk and meconium: any invaders which the meconium harbors may multiply dangerously in the milk and the meconium. The prompt and physiological evacuation of the meconium constitutes a highly essential guard for the health and life of the young.

The equine fetus and the new-born foal show a marked tendency to the impaction of the large intestines with meconium. One needs bear in mind the marked physiological difference in the meconium of equine and bovine fetuses. The meconium of the equine fetus is physiologically dry and firm as compared with that of the bovine young. Diarrhea is common in the bovine, but I have not observed

it in the equine fetus; in contrast, impaction is rare in the bovine, and common in the equine fetus. The incidence of impaction and of diarrhea in the new-born is essentially parallel with its frequency in the fetus. It is difficult, or impossible, to show that meconial impaction constitutes a definite disease in the equine fetus. It is suggested, however, in some cases. For example, the foal illustrated in Fig. 190, carried in utero 380 days, was born with an enormous volume of meconium impacted in the large intestine. The subject has not been studied from this viewpoint, but it is quite logical to surmise, until otherwise determined, that the impaction grows as the duration of pregnancy is prolonged, not fundamentally because of the element of time but because prolonged gestation is pathological and the behavior of the meconium is likewise abnormal. That is true of diarrhea in the bovine fetus: the diseased individual expels more meconium into the amniotic cavity than the healthy fetus stores in its alimentary canal, but that by no means empties the intestinal canal of the diseased fetus. The evidence indicates that the diseased fetus produces meconium more rapidly than the healthy one, regardless of the duration of pregnancy.

Stoss,[189] apparently taking a different view, considers the impaction as a post-natal disease, due to the failure of the colostrum to act as a cathartic, or to other causes which act post-partum. Stoss is generally correct when the diagnosis is based upon clinical evidences of colic. Generally the foal takes colostrum or milk before the clinical onset of obstipation colic. That fact is open to two different interpretations. My observations lead me to conclude that the impaction has become established during fetal life and that the post-natal colic is caused by the decomposition in the small intestine of the milk consumed, the discharge of which is barred by the impacted meconial mass which has long existed.

The symptoms are those of obstipation colic, not differing materially from those observed in adult horses. The colic is expressed by rolling, lying down and getting up, kicking at the belly, and, as the disease continues, by loss of appetite, rapid pulse and fever. A common, and somewhat pathognomonic sign is that the foal stands in an extremely extended position with the anterior limbs extended far forward and the posterior members far backward. This brings the body lower than normal, which is further accentuated by an attitude of lordosis.

Almost always the diagnosis may be verified if the impacted mass is palpated by inserting a finger into the rectum. Sometimes the meconium has been expelled from the anal end of the rectum and an

impacted mass persists far forward, beyond palpation. In rare cases, also, there occurs atresia of the colon, as described by Yamane[276] and others, and already discussed in Section I. This causes essentially the same symptoms, except that the rectum is necessarily empty.

The prevention of the clinical evidences of the disease is comparatively simple. The meconial colic is so common, among foals where breeding is forced and genital diseases prevail, that each foal should be regarded as potentially liable to colic, and the accumulated meconium artificially evacuated. This is usually easy of accomplishment with warm enemas of physiological salt solution. It may be safely carried out by an intelligent groom, under veterinary direction. It is best accomplished with an ordinary hospital irrigator, with a soft horse catheter substituted for the regularly supplied rubber tubing with hard nozzle. Only a small amount of the solution, eight to ten ounces, should be introduced at a time, the aim being that the enema should remain in the rectum for fifteen to thirty minutes, instead of being hurriedly expelled. It should begin as early as practicable after the foal is born and may be repeated hourly until the dry meconium is expelled and followed by soft yellow excreta. Olive, castor, or white mineral oil may be substituted for the saline solution. Some advocate the administration of four to eight ounces of castor oil per mouth. I am not aware that it is dangerous.

In neglected cases, when the meconial colic has become established, there is little to be done except to apply with greater persistency and frequency the remedies already mentioned. They will not always succeed. The rectum may be emptied for three to four feet with enemata, without great difficulty, but, if the case has been too long neglected, the impaction in the caecum and great colon may not be overcome by enemas. Cathartics given per mouth may not be competent to overcome the atony of the intestine.

Meconial impaction occurs rarely in the bovine fetus and is carried across the birthline into extra-uterine life. The meconium, as I have observed it, in these cases, is in small pellets united by a slender strand. The desiccation apparently occurs in the small intestine, and moving analwards, the meconial cord becomes doubled upon itself and closely packed in the rectum and colon. Presumably, such impaction may occur in any species, but becomes of material importance only in the foal.

Chapter XXXIX

THE PHYSIOLOGY, PATHOLOGY AND HYGIENE OF THE DIGESTIVE SYSTEM OF THE NEW-BORN YOUNG

The Artificial Feeding and Handling of the New-Born

Diarrhea of the Young. White Scours. Calf Scours, etc.

DIARRHEA of the new-born causes important losses in all species of herbivorous domestic animals; it runs somewhat parallel with the prevalence of sterility, abortion, retained afterbirth and related phenomena, and reaches its maximum virulence in dairy calves. Fetal diarrhea is prevalent in ruminants. The fetal disease is carried across the birthline in some individuals, but most fetuses which suffer from diarrhea succumb during intra-uterine life and are aborted or "born dead." I have described[225] enzootic diarrhea in lambs in a herd where abortion raged. The aborts, as well as the live lambs, were thickly and repulsively covered with diarrheaic feces. Many of the ewes refused to own their lambs and viciously repulsed them when they attempted to suck, suggesting that the lambs were disowned because of their repulsively filthy condition. In the past, the repulsion of the new-born by the mother has been largely attributed to some psychic disturbance of the dam. The above observations invite the hypothesis that perhaps in solitary cases, also, the repulsion of the young is dictated by repulsive disease of the latter, and may offer an explanation for cannibalisim in the sow. This animal regularly devours the fetal membranes of her young and completes the cleaning up of her bed by eating all fetal cadavers which she has expelled. It seems possible that she might also devour any repulsively diseased young. The herbivorous mother uniformly licks her physiological young, removing all fragments of fetal membranes and leaving it dry and clean. It is not at all surprising that the ewes repelled the dirty, diseased young, extremely repulsive alike to sight, smell or taste. Physiologically the dam identifies authentically her young, apparently by smell; when seriously diseasesd, the odor can no longer be authentic.

Diarrhea of the new-born is commonly ascribed to postnatal errors in food and care. In a way, this hypothesis is irrefutable. It is well, however, to bear in mind the dual causes generally operating—antenatal and postnatal. The disease prevails most abundantly and

virulently in those species and breeds kept under extremely artificial conditions in breeding, feeding and handling, all of which are most highly concentrated in dairy cattle. In highly bred dairy cattle, the diarrhea of new-born calves is an ever present and highly destructive peril; in commercial beef herds constantly in the open pasture, it is virtually unknown.

It is scarcely justifiable to consume space with a description of the symptoms of diarrhea in general. There is present more or less copious discharge of liquid feces, varying in color, somewhat according to species and environment. Severe diarrhea in calves is usually associated with diffuse gaseous decomposition of the excreta, the small gas bubbles giving to the discharge a pale color—*white scours*. The discharges are viscid and irritant. The tail and buttocks are smeared over with feces, followed presently by denudation of the hair. In severe cases there is rapid loss of flesh, ending frequently in loss of appetite and death.

As in many maladies, there is no fixed line of demarcation between health and disease. The milder cases pass by imperceptible gradations into such evidences of disease as adhesive feces which adhere to the tail and buttocks, dry rough coat and pot-belly.

As already stated, diarrhea in dairy calves occupies a position of great economic importance. The problem is a complex one into which many factors enter. Some of the principal of these are:

1. The weakness of the fetus and the invasion of its digestive tract by infections. Fetal diarrhea is generally present in abortion and the organisms associated therewith are most frequently identifiable in the alimentary contents. The presence of bacteria in the meconium is not confined to fetuses suffering from diarrhea or other identifiable disease. For example Hagan[76] recognized bacteria in the meconium of apparently healthy new-born calves and in seemingly physiological fetuses in the abattoir. It appears wholly justifiable to conclude that bacteria common to the uterine cavity of the non-pregnant cow, the utero-chorionic space of the gravid uterus, and the meconium of apparently healthy fetuses and new-born calves may become pathogenic under favorable environment, and cause disease.

The bacteria are swallowed by the fetus with the amniotic fluid. This may be regarded as parallel with the general view of the postnatal origin of diarrhea, that it is largely caused by the ingestion of contaminated, or otherwise unsuitable food. The swallowing of the amniotic fluid by the fetus is the means by which the fluid is kept free from disease invaders, as well as from hair, mucus and other epithelial debris. The bacteria, when imprisoned in the meconium,

are held in abeyance except when the numbers or virulence get out of control.

Any bacterial disease of the fetal membranes constantly imperils the asepsis of the meconium. Thus diarrhea is highly probable in cases of retained afterbirth and of abbreviated or prolonged gestation. Casual reading of the literature reveals statements that a cow gave birth to a healthy calf and had retained afterbirth: that a fetus was healthy despite the fact that its most essential organ was diseased! The hypothesis is indefensible from the standpoint of either logic or clinical observation.

2. The food supply to the dairy calf is abnormal. By means of selection, feeding, and handling, the quantity of milk has been multiplied many times, and in some breeds, such as the Jerseys and Guernseys, the ratio of butter-fat has been advanced. If the calf is left with the cow, it is free to over-eat and doubtless does so. This is not for long. Then it is taken from the cow and fed artificially, with an endless train of dangers.

The amount of milk available to the new-born equine or bovine young under primitive conditions is unknown. The ordinary mare certainly supplies a rather limited quantity of milk of very low solid content, as related to the body weight of the foal. The volume of milk supplied by an ordinary beef cow, living on the usual pasture, is not known, but since it is certainly quite limited, overfeeding of the new-born is largely excluded. The fetal digestive system has not been extensively studied. The principal known facts are that the fetus constantly and rapidly swallows its amniotic fluid with any solids which may be suspended in it, and that the liquid portions are promptly absorbed from the alimentary tract and thrown into the lymph and blood streams to go to the placenta for desirable changes: the amniotic fluid is an integral part of the fetal circulatory system.

When birth occurs this important part of the fetal circulation is abandoned, the digestive system expels the stored meconium, and the new function of taking food in the form of milk is initiated. The colostrum, or first milk, differs in its constituents from the milk secreted later. Great emphasis has been placed upon the desirability or necessity that the young calf shall get the colostrum because it acts as a purgative and causes the prompt expulsion of the meconium. No exception is made for the calf born with diarrhea. The purgative or laxative substance contained in the colostrum has not been identified and there is no proof that the colostrum possesses any purgative or laxative power. The foal, regularly getting all the colostrum from its

dam, suffers most severely of all animals from meconial impaction and colic.

Occasionally a calf becomes lost just after birth, presumably before it has sucked, and is not found for a number of days, when it has expelled its meconium without having tasted colostrum. Or, as in a number of my cases, a calf has been removed from a dying cow by cesarean section and the orphan does well although it receives no colostrum. Rarely a dairy cow is a persistent milker so that no opportunity is given for the formation of colostrum, but her calves do well. Sometimes the farmer errs in his records and milks his cow through pregnancy. If the cow, nevertheless, secretes colostrum, it does not physic those persons who drink it, and the calf does well. Naturally the calf obtains colostrum and, if physiological, thrives upon it, but it is not wholly clear why its value should be so emphasized over that of milk.

3. The artificial feeding of the calf subjects it to a multitude of dangers largely absent in other species of animals. The artificiality includes: the amount of milk; the intervals at which it is fed; the temperature of the milk at feeding; the cleanliness of the milk and milk vessel; the substitution of skimmed, for whole milk; the substitution of other foods for milk, etc.

4. Dairy cattle are more closely housed than other animals, although normally cattle are the least gregarious. Dairy cows are kept in the closest confinement, by the stanchion method, of any breed or species. Dairy calves, for commercial reasons, are largely born during winter and early spring, when the close confinement of the cows in the stanchions has lowered their vigor and intensified utero-fetal disease. Dairy calves are even more closely confined than their dams: they are stabled in winter to protect them from cold and during the summer to shield them from flies. The stables in many cases are colder in winter than out of doors and in the summer season the flies are often worse in the stable than outside. They are denied sunlight, open air, and physical exercise. The stabling deprives them usually of their most valuable food, next to milk, green grass.

5. The close confinement of cows and calves advances the virulence of bacteria, protozoa, and viruses which may regularly exist as nonpathogenic residents until they become destructive contagions, readily transmissible by close contact and through their food from common containers.

So dairy calves have calf scours. With the diarrhea are associated pneumonia, arthritis, and other phenomena. The symptoms of diarrhea of the new-born in general have been briefly described above.

It is well to add that, according to my observations in the one herd already cited,[257] there was one tell-tale symptom which forewarned of the approaching storm, before the calf showed any diarrhea, loss of appetite or distress. The calves were kept in individual pens upon clean, short-cropped grass and moved daily. Under these conditions there appeared, as a danger signal, droplets or streaks of blood in the feces. This cannot usually be observed in stable calves; and when two or more calves are kept in one enclosure, the identification of the calf from which the blood-stained feces came is difficult or impossible. The blood-stained feces may possibly have been owing to the type of infection prevailing. I find no record upon this point by other observers.

The infections involved are apparently legion. A long list of bacteria have been described by various writers, and to these one protozoon, coccidia, has been added.

Nowhere has high-pressure dairying outrun veterinary science more disastrously than in the field of calf diarrhea. The field is a complex one. Some of the outstanding causes have been briefly mentioned above. Each of these is difficult to adjust because remedies suggested are in conflict with what the dairyman regards as economic necessity; or long prevalent prejudices are too firmly rooted to make changes possible.

Each dairy calf should be regarded as a potential victim of diarrhea and the best available prophylactic measures employed. The peril is intensified by each and every abnormality involving its antenatal life. Bull 2 in Table II, with badly diseased genital organs, as shown in Fig. 138, showing a high abortion rate in his pregnancies, much metritis and a subnormal duration of pregnancies, inevitably begot only weak calves. The prevention of diarrhea in his calves dictated the killing of the bull and active preventive measures against calf scours immediately the calf was born. So with the bull illustrated in Fig. 139, with disastrously low duration of pregnancy and the high incidence of retained afterbirth, diarrhea was to be expected. The same law applies to the cow: if she has suffered from sterility, abortion, or retained afterbirth, diarrhea in her living calves is to be anticipated.

Here it would seem prudent to break with the universal idea that the calf should be left free and undisturbed with the cow for twelve, twenty-four, or more hours and be permitted to partake freely and abundantly of the colostrum. It is almost universally urged that, if the calf is weak (sick) and cannot get up, it be aided to rise, carried to the udder, and the teat pushed into its mouth. Or if yet more

seriously sick so that it cannot hold up its head, when its body is raised, colostrum must be hurriedly drawn and forced down the sick calf as a remedy for the infectious and toxic substances which it has carried across the birthline. It is idle to question the value of any food which may be swallowed, digested, and assimilated by the sick calf, but such a calf is not usually competent to digest and assimilate food. My view, based upon clinical experience, is that such calves should not be allowed to be with their dams without interference, but instead should be actively handled as diseased animals. I would not deprive them entirely of colostrum, but would limit the volume—one-half to one pound, mixed with an equal volume of limewater, to which I would add three (for Jersey or Guernsey calves) or four ounces (for Holstein calves) of castor oil. The castor oil will generally impress the dairyman as a violent cathartic, but I have not been able to see that it acted even as a laxative: its action appears to be antiseptic. The precise action of the limewater is not fully known. It doubles the water content of the milk and lowers the ratio of fat. The lime water emulsifies the fat, and presumably favors thereby its digestion and assimilation.

With the modification just mentioned, I would follow the usual custom of leaving the calf undisturbed with its dam for twelve to twenty-four hours and would then cause it to fast for an equal length of time. Then as the first feed—the calf being apparently healthy and hence not yet fed artificially—I would give one pound each of milk and limewater with four ounces of castor oil, already mentioned. Thereafter my experience teaches that one pound each of milk and limewater twice daily is ample food for the first week of life for a Holstein or other large calf—about one per cent of the body weight of the calf at each feed. This is in conflict with the general teaching. Most writers advise two to three times this amount. In my experience the above ration induces rapid growth, and the calf is very vigorous and playful. There is no pot-belly, nor is the calf gaunt. The abdomen is of even contour, as indicated in Fig. 35, and the hair retains the brilliant luster present at birth.

The stomach of the new-born is small. The calf when artificially fed and compelled to go without milk for ten, twelve, or more hours, becomes abnormally hungry and if offered a large volume of milk will naturally overeat. When four to six pounds of milk are given a hungry calf, it does not have room in its stomach in which the milk may be held pending the normal digestive action of the gastric juice. The rumen is extremely small and primitive. Fluid foods apparently pass by the rumen directly into the abomasum. If the volume of milk

swallowed exceeds the capacity of the true stomach, it must back up into the rumen or pass the pylorus into the small intestines. In the first case the gastric digestion is delayed; in the latter it is omitted. Opinions may differ regarding the importance of gastric digestion, but when the dairyman wishes to make cheese, he relies upon *rennet* from the calf.

Following significant clinical experience, I have been profoundly impressed by the results of the above plan of restricting the volume of milk, during the first week in the life of the dairy calf, to one pound each of milk (or colostrum) and limewater, twice daily. The calves grew more rapidly, and showed the highest degree of vigor that I have observed under any method of artificial feeding.

Although my observations[257] were published in 1935, and repeated since, the plan has apparently aroused no interest, and no public comment has appeared, either in approval or disapproval. It has for so long been held, virtually without dissent, that the new-born dairy calf requires a daily allowance of milk equal to ten percent of its body weight, that discussion seems out of order, regardless of the fact that the dairy calf suffers by far the highest mortality, during the first few weeks of its extrauterine life, of any breed or species of mammal.

The plan recommended above departs too radically from firmly established beliefs to justify its adoption without careful investigation. Dairymen complain of the cost of milk-feeding of calves. The proposed plan reduces this cost by 80 percent. Fortunately the method may be adequately tested, at trivial cost, by selecting for experiment a few bull calves of low value.

The milk should be clean. The milk is more digestible and less inclined to cause diarrhea if fed raw. If tuberculosis exists in the herd, the milk needs be pasteurized. There may be other diseases of the cow which render pasteurization advisable. Septic sore throat, typhoid fever, and other human diseases in which the milk serves as a carrier are not known to be transmitted to calves. It is stated that *Bacillus abortus* infection is transmitted to young calves by milk. Most writers hold that the infection is later eliminated and the calf arrives at breeding age in a healthy condition. This view is in harmony with the modern advocacy of calfhood vaccination. In researches, however, made in 1913-1914 in a herd saturated with *Bacillus abortus* I found[233] that the infection, according to the agglutination test, Table XIII, persisted from early calfhood until the heifers conceived and aborted. That does not settle the problem. The animals named were out of sick cows; they were imprudently

fed; they were reared under pesthouse conditions and had abundant reasons for breeding badly aside from the abortion bacilli consumed with their milk. There is no conclusive proof that an otherwise healthy calf, grown under prudent rules, is liable to suffer materially from abortion bacilli in the milk.

Some writers express the opinion that the milk from a cow suffering from mastitis may cause diarrhea in calves; they fail to submit data showing the peril. Logically it would appear to be dangerous.

The calves shown in Fig. 35 were in a herd where sterility, abortion, and retained afterbirth were raging at the time. The calving cows and heifers, with the calves, were kept distant from the dairy herd. The calves were fed the milk from those cows deemed unfit to go into the dairy—those suffering from retained afterbirth, metritis, vaginitis, etc. The milk drawn from these diseased cows, without special precautions of washing or disinfecting the udders was immediately carried to the near-by calves and fed without pasteurization. They were as healthy and vigorous calves as I have seen in any country or under any plan of feeding (see page 598).

The confinement of calves in the stable should be reduced to the available minimum. Next to adequate food, the greatest bulwarks of health are sunlight, pure air, and abundant exercise. These are not attainable in stables. Dairymen in temperate climates have come to believe that dairy calves must be kept in the stable in winter to avoid freezing and in summer to avoid flies. My observations, in regions where dairying was not a commercial objective and milk for family use was obtained from cows largely used for beef production, taught me that if a cow calved in the open with the temperature at or below zero F. the calf was liable to lose its tail and ears from freezing but retained its life. When a few weeks old, zero, or sub-zero weather had no perils for them, so long as they had plenty of food and some shelter against the force of the wind—a shed, a stack of straw, a wall, etc. Such extreme exposure to cold is not advocated, but the facts indicate that the general view that young calves *must* be constantly stabled in winter is entirely too radical and is injurious to their health.

As to the other claim, that calves must be kept in a *dark* stable during summer to protect them from flies, my experience most emphatically teaches that close confinement is infinitely worse than fly bites. The calves in Fig. 35 were in the tropics, with a perpetual fly season and an abundance of flies not seen in dreams by dairymen in temperate climates. The amazing observation was that the flies caused no readily recognizable harm. Especially the young calves, with their highly resilient, vigorous bodies, suffered no great annoy-

ance. They suffered far less from the flies than any group of calves I have observed in the best kept stables in temperate climates. The flies worked chiefly upon adult cattle with less flexible bodies, and especially concentrated their attacks upon the necks and shoulders of the bulls, with their thick, laterally immobile necks. If a calf were sick, however, it made a feasting center for the flies under the prevailing law that parasites love a defenseless host.

New-born dairy calves should be isolated. There is no more valuable method known for the control of disease due to contact, when the peril is limited to a comparatively brief period. The isolation is relatively inexpensive.

One of the grossest and most indefensible violations of established hygienic laws is the communal feeding vessels for young dairy calves. A sick calf, fed its milk ration from a pail, pollutes the vessel thoroughly from its mouth and nostrils. Some milk is left in the pail, more is added, and calf after calf is exposed to any and all infections carried by all the calves previously fed. The cost of feeding vessels is not great, and if one is feeding ten calves, the wear upon one pail for feeding the ten will be about ten times as great as if each calf were fed from an individual vessel. The pails should be sterilized after each feeding, should ordinarily be kept out of doors or in a thoroughly clean enclosure, and should be kept inverted, not nested.

Physical isolation is essentially impossible in the stable, but should be as closely approached as possible where the calves can not be kept out of doors. The isolation is especially desirable during the first month. The most effective method I have seen, when the calves can be kept out of doors, is by the light, portable pen. For this purpose I have used a pen 6 x 8 feet, 3.5 feet high. It consists of four 2 x 4 posts with 1 x 4-inch boards at top and bottom. The enclosure is completed by means of poultry wire filling the space between the top and bottom boards. Protection against hot sun and snow and rain may be secured by means of a 4 x 8-foot flat roof laid across one end of the pen, and the protection against cold winds may be secured by the use of a similar shield placed upon the windward side. The calf then has protection against wind, rain, or hot sun, and has adequate room for play.

Such a pen located upon a clean lawn, and moved daily to new ground, assures cleanliness. In the absence of any bedding, the feces expelled by the calf are in plain view and any blood streaks or other signal of impending diarrhea are at once discovered. The calf being alone, there is no question of the identity of the feces. Under this plan there is no rubbish or indigestible substances which the calf

may swallow, and hence no occasion for the use of a muzzle. The pens are inexpensive to make and distinctly economical in maintenance. The pens cost little more than a muzzle, which our best observers deem essential in the stabled calf. The moving of the pen for a distance of six feet daily requires less than a minute. The cost of bedding material and of labor involved in changing the bedding and of grooming or cleansing dirty calves is wholly eliminated. It is far more economical than keeping the calf in the stable, even though the change to the pens leaves the stable vacant. Finally, when inclement weather compels the placing of the calves in the stable, the latter, with proper care, is clean and in better condition for occupation.

The calf should not be permitted to eat or swallow filth or indigestible food. The early separation of the dairy calf from its mother throws its natural instincts out of balance. One of the first and most pernicious results of any disturbance in its digestive functions is the eating or swallowing of indigestible objects. When running free in a paddock, dairy calves will pick up and swallow dry grass, weeds, feces of horses, swine, cattle, or other animals, pieces of metal, wood, or fabrics of any kind. If in the stable, the least disturbance in digestion causes the calf to swallow bedding. Some dairymen offer hay to calves when but a few days old, before their rumen is ready to function or the digestive tract is ready to accept it with safety. Natural reproduction is so adjusted that cows calve in the springtime, when tender grass is available, which the calf may nibble with safety, when a few days old.

Udall[295] strongly advises the use of the muzzle in the stabled calf. Its value is undeniable in these, but, as stated above, is superfluous when calves are kept out of doors in portable pens on a clean lawn. As early as 1909 I[231] had advised the use of the muzzle on foals or calves discovered eating bedding or rubbish. In the stable, no calf of material value should be permitted to go without a muzzle during its first thirty days.

Artificial foods for dairy calves are recommended by influential writers. The dairy industry loudly proclaims that cow's milk is the best and cheapest food for the sustenance of children and for many other persons. The dairyman then performs an about-face and claims that calves may be grown more economically and quite as healthfully upon other food-stuffs than milk. Unfortunately he can partly defend this claim. Dairy calves, fed upon this highly lauded milk, suffer more alimentary disease and death than any other known new-born mammals. It is the abuse, not the use of cow's milk as calf food which brings the disaster. The actual value of artificial foods for

young calves can not be authentically determined by the rate of growth. The value of a dairy calf must be finally computed upon its longevity, fertility, and in case of a heifer, her dairying efficiency.

In the above plan for feeding calves, the ration of one pound each of milk and lime water twice daily is continued for a week. The milk ration may then be gradually advanced by four to eight ounces per day, until eight or ten per cent of the body weight per day has been reached. Close watch of the feces should be kept, and, should blood stains or other signs of disorder appear, the ration should be immediately returned to the original feed of one pound each of milk and lime water with four ounces castor oil. This is rarely needed if the recommended ration is closely followed, and the calf is prevented from eating rubbish.

When one month old, separated milk may be partly substituted for whole milk. If in the portable pens the calves will usually have grass available and will have become accustomed to eating it moderately. This should always precede any offering of hay or grain. These latter call for rumination, and the rumen develops slowly. During winter, when grass is not available, its best substitute is carrots, turnips, cabbage, or other succulent foods. Gradually small quantities of ground oats or other grain may be allowed, with the least woody type of hay, such as early cut clover or alfalfa.

The amount of food should be carefully limited until the calf has reached six to twelve months of age. Whenever the amount exceeds the needs, loss of vigor follows and disease is invited. The constant aim should be to maintain the degree of health illustrated in Figures 32, 34, and 35, and to escape the rough coat and pot belly of Figs. 31 and 33. Constant watch is to be kept upon the character of the feces. Should symptoms of diarrhea develop, it should be handled at once with vigor. More frequently, after the first few weeks, the chief signal of low vigor, due to errors in feeding, is the adhesiveness of the feces. Small fecal masses adhere to the tail and to the hairs about the anus and vulva. This generally passes unnoted, but it means much for the eventual value of the animal. It is to be borne in mind that, during the first six months, the reproductive system is undergoing its functional development and that, unless the health of the calf is maintained upon a high level during this period, it can not become a notably efficient adult.

Diarrhea is variously handled. I have employed a long list of remedies or alleged remedies. In my most recent experience I have adhered to the plan above described as prophylactic. The primary consideration is to first of all withdraw food to a sufficient degree

that it shall not constitute a burden. Few writers emphasize this necessity. It should be clear that the introduction of a large quantity of food, in addition to that already present in the alimentary tract, merely adds to the load which is already threatening the health or life of the calf. Unless prudent precautions are taken, the added food also decomposes. Hence the liberal allowance of food does not afford nutrition, but aggravates the malady. The commercial anti-calf scours serum acts beneficially as a prophylactic and as a curative agent, but in my later experience its use has appeared quite superfluous. Similarly good results are reported from the subcutaneous administration of 50 to 100 cc. of dam's blood.

Calf Pneumonia

Calf pneumonia. Pneumonia is a common complication of diarrhea in calves, especially in persistent cases of medium severity. A second type is observed and described, not notably associated with diarrhea and attacking the calves at three to six weeks or later. Writers do not deny, however, that diarrhea accompanies the pneumonia. Sanders[176] describes pneumonia as observed in dairy calves in Florida with a mortality of fifty to seventy per cent. He does not state that they were stabled, but does say that they were artificially fed. There were present the usual symptoms of pneumonia—accelerated breathing, cough, nasal discharge, etc.—and with these was persistent fetid diarrhea. Pneumonia recurs year after year in given herds, and various bacteria are found. It is not clear that the bacteria found differ notably from those normally occurring in the intestinal tract. In discussing Sander's contribution, Roberts states that in his observations, in Brazil, Puerto Rico, and the Dominican Republic, pneumonia is highly prevalent in purebred and grade dairy calves. It is not observed in native cattle nor in cattle running free in pasture. The cows in the affected herds were milked but once daily, and the calves were permitted to suck the dams before milking. The calves were confined in unsanitary buildings. Inevitably they were abnormally hungry and ate hastily and excessively.

Diarrhea and pneumonia occur but rarely in other than dairy calves. In dairy herds under my observation, where the calves were constantly at pasture and were permitted to suck their dams twice daily, neither diarrhea nor pneumonia has ever been seen. Udall[205] in describing calf pneumonia, does not directly state that the calves are free from diarrhea. He does say that it is virtually confined to stabled calves.

The stabling and artificial feeding of dairy calves are usually inseparably linked, but this is not always true. In the large Holstein herd

which I studied in the tropics[257] the calves were never stabled. Yet diarrhea and pneumonia prevailed under the common method of feeding. The avoidance of diarrhea and pneumonia calls for the recognition of at least three basic factors: the calf needs be healthy at birth (physiological duration of pregnancy, absence of dystocia, retained afterbirth, etc.); the evils of stabling should be eliminated; and the feeding must be effectively controlled.

Necrotic stomatitis is occasionally observed in association with diarrhea and pneumonia and is attributed to *Actinomyces necrophorus*. It is characterized by necrotic erodent ulcers in the mouth, which, along with diarrhea, rapidly exhaust the strength of the animal. The calf shows a pernicious tendency to swallow indigestible substances greatly aggravating the disease. A calf in my clinic died from the malady, and the autopsy revealed the presence in the alimentary tract of large volumes of bedding shavings. It was followed a few days later by a second calf from the same stable. Forewarned, I directed that no bedding be used. The students assigned to the case swabbed the ulcers in the mouth with absorbent cotton. Autopsy revealed that all the cotton used had been dropped on the floor and swallowed by the calf.

There is virtually no end to the complications of diarrhea and pneumonia. Skeletal disease, which may as well be called rachitis, not infrequently arises in chronic diarrhea and pneumonia.

Diarrhea in foals is frequent in areas where genital diseases prevail and horse breeding is strenuously pursued. Artificial feeding of the new-born, occurring rarely, exerts no material influence upon the incidence of the disease. As a rule, before diarrhea may set in, it is essential that the impacted meconium be expelled. In my rather limited observations among thoroughbreds, where I assured the expulsion of the meconium immediately following the birth of the foal, I observed no case of diarrhea. My impression is that the failure to see that the meconium is promptly expelled has an important relationship to the later diarrhea. The rules for age classification in thoroughbreds lead to the breeding of mares to foal in late winter or early spring, so that when the foal technically becomes a yearling on the following January 1, it shall be well grown. This serves to cause the foal to be born after a long period of stabling of the mare, before she has had a run at spring pasture. There is a marked contrast in the clinical history of diarrhea in calves and foals. Calf diarrhea is most common after abbreviated gestation, while in foals it is most frequent after prolonged pregnancy. In each, it is most common in the new-born of low vigor.

The symptoms are parallel in principle to those of calves. Ordi-

narily the onset is delayed because of the impacted meconium. If one examines critically the feces, it will be generally found that the discharges largely consist of straw or particles of rubbish of varying character. If the foal is watched, it will be observed that it is very fond of feces. The woody fragments in the diarrheal discharges are largely broken bits of hay or straw from the feces of adult horses which the foal has swallowed. The etiology of diarrhea consequently provides interesting comparisons with that in calves. Stoss[189] states that the onset of diarrhea in foals frequently coincides with the first estrual period (circa 9 days) of the mare, with the inference that the heat period causes the diarrhea. This I have not observed in common mares, but have seen it in thoroughbreds. However, in the thoroughbreds under my observation, genital disease was rampant, and at the ninth day the intra-uterine infection present fulminated, and a copious muco-purulent discharge ensued. This I have failed to observe in ordinary, healthy mares. Accordingly two hypotheses may arise: the estrum of the mare causes diarrhea in the foal; or the estrum causes the explosion of the endometritis and the latter causes the diarrhea.

Prophylaxis, so far as may now be seen, consists of assuring the early expulsion of the meconium, the withdrawal of some of the mare's milk if its volume so suggests, and the careful guarding against the swallowing of indigestible rubbish. If the foal is discovered in the act of swallowing rubbish, or the examination of its feces reveals its habit, the foal is to be muzzled, as I advocated as early as 1909.[231] When the diarrhea becomes established, the food supply should be reduced. Various plans for handling have been advocated. At first astringents (tannin, alum) were advised, then stimulants (aromatic spirits of ammonia, camphor, peppermint) and emollient enemas (elm bark, linseed decoction). The principles involved are essentially the same as for calves, and the same general plan is advised.

Diarrhea in lambs is sometimes highly destructive. Somewhat like calf diarrhea, it is virtually limited to stabled animals. Sheep, our most gregarious domestic animals, crowd closely together. When confined in stables or sheds they are generally crowded and the stable rapidly becomes polluted by excreta. Marsh and Tunnicliff[134] record extensive losses in Montana herds and cite numerous records of severe outbreaks in various parts of the world. Various bacteria were found in the diseased gastro-intestinal tract, usually of similar type to those found in healthy young. Invaders, which had existed as saprophytes, increased in virulence when the vigor of the ewes and of the new-born was depressed, and the invaders became pathogenic and transmissible.

Writers generally fail to state the reproductive health of the involved herds as expressed by the incidence of sterility or abortion. It has been stated above that, in an outbreak of enzootic abortion in sheep, intra-uterine diarrhea had been highly prevalent in aborts and living lambs alike.

The inference may be drawn from the literature that writers believe lamb diarrhea strictly postnatal in its origin. But it has not been shown that the lambs were actually vigorous when born.

It appears that the cause has not yet been clearly enough ascertained to provide effective means for prevention. The general principle applies that, if the ewes are bred to lamb at pasture in spring, the disease virtually ceases to appear. The shepherds, wishing early lambs for commercial reasons, violate the dictates of nature, with evil results. No satisfactory method has yet appeared by which the shepherd may ignore the natural law and at the same time evade the perils of disease. Marsh and Tunnicliff relate that one shepherd constructed a shelter in which the lambing ewes and their lambs were kept on elevated, slatted platforms above the floor. No bedding was supplied and the dung and urine dropped through the platform to the floor beneath. This is reported as successful in the one instance. The expense of the plan is not divulged.

Diarrhea of the young occurs in all species but its ravages are almost wholly confined to closely housed herds, and it attains its greatest virulence in the artificially fed dairy calves. Losses from diarrhea are reported common and important in closely confined swine. I have had no clinical experience with swine so handled. I grew up in a great swine-breeding area in the Mississippi valley. During my experience on the farm the swine were free, constantly out of doors, with a wide range, and supplied with abundant and varied food with maize as a basis. They had no garbage and did not serve as scavengers. I recall no instance of diarrhea in pigs among the thousands coming under my observation.

Omphalophlebitis. Navel Infection. Navel Ill. Pyaemic Infection of the Newborn

The pyaemic and septic phenomena of the new-born defy accurate classification. When the fetal placenta becomes materially damaged, a gateway has been opened to the invasion of the fetal blood stream, from which every organ and tissue in the fetal body and its membranes may be penetrated. The amniotic fluid, which must be regarded as an integral portion of the circulatory fluid of the fetus, becomes invaded and, being swallowed, contributes to the infection of the alimentary tract.

The genera and species of invaders are of unknown numbers. They are presumably as numerous, and largely identical with, the vast number of infections associated with sterility, abortion, retained afterbirth, and allied diseases.

When the fetus is born, new avenues of invasion are opened, through the alimentary tract, by improper feeding. The division of the navel cord at birth opens a highly important avenue through which pathogenic forces may invade the blood stream. Fortunately the natural precautions against invasion are highly effective. The natal wound involves the rupture of the umbilical arteries, vein and

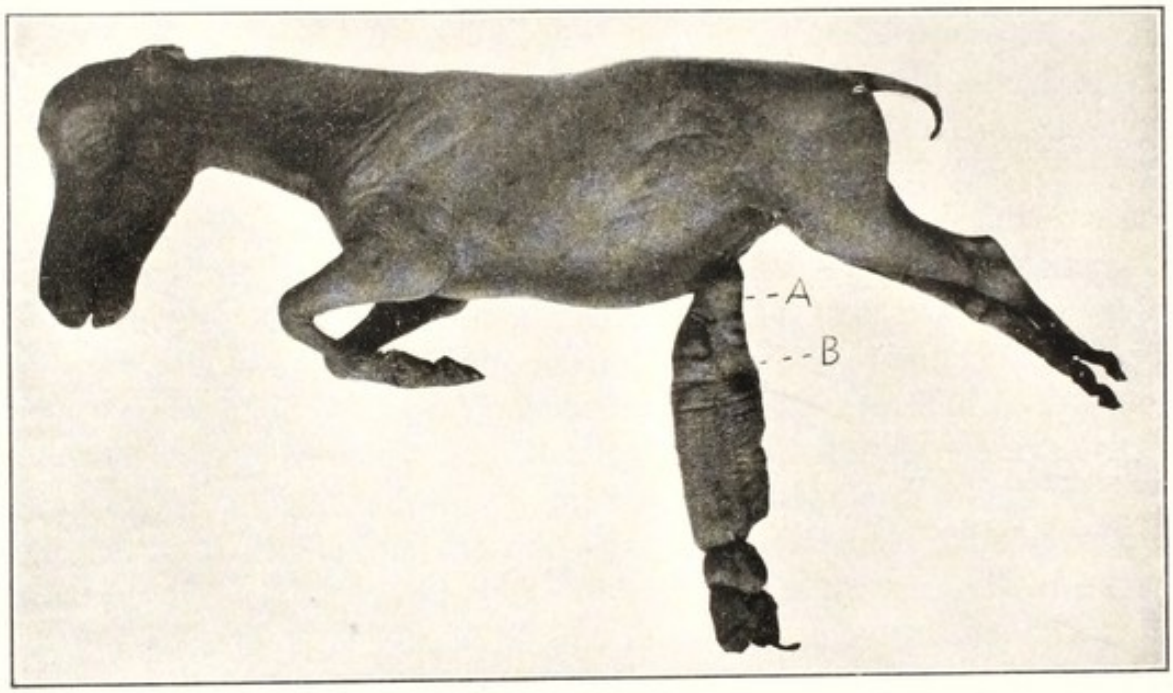


FIG. 192. Umbilical Cord of Foal.
A, Cutaneous portion; *B*, amniotic portion.

urachus, and the division of the enveloping amniotic sheath and connective tissues.

The umbilical cord is said to rupture near the umbilicus at the point of juncture between the cutaneous and amniotic sections, as indicated in Fig. 192, at which point the urachus and arteries are believed to be less resistant. The division of the cord is variously accomplished, according to the species. The bovine umbilical cord is less than one-half the length of the fetus. Its allantoic end is fixed in the ventral wall of the chorion and uterus, about the length of the fetus from the vulva. Regularly the cord ruptures before the fetus is completely expelled. The cow normally assumes the recumbent position when the expulsion is nearing completion. The fetus tends to be expelled in the arc of a circle, the ventral wall facing the inferior

commissure of the vulva. Sometimes the line of expulsion is extremely arciform, perhaps partly due to the resistance of the navel cord, and the buttocks and hind legs of the fetus are pushed out, so that the expelled fetus lies perpendicular to the long axis of the birth canal (vagina) with the navel cord intact. The cord is so small, as compared with that of the foal, that any important movement of calf or cow ruptures it. The amniotic sheath of the cord parts somewhat distal to the cutaneous navel. It is not accurately known at what point the urachus and the two umbilical arteries part. Immediately after the rupture of the cord, the broken end of the urachus, with the ruptured ends of the umbilical arteries, rests within

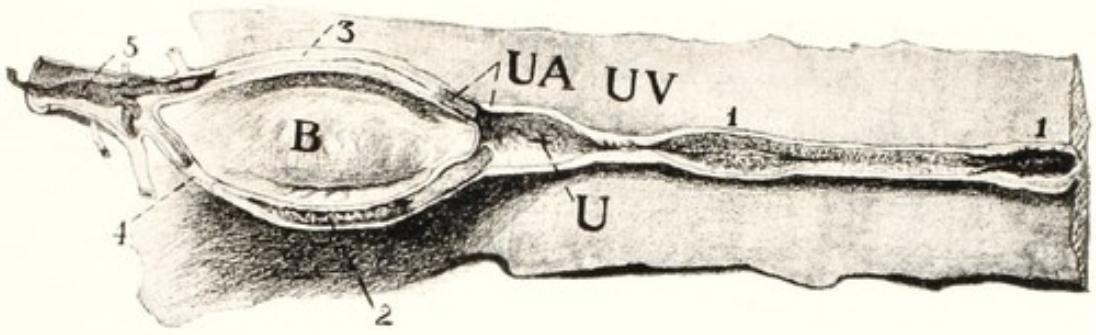


FIG. 193. Abscesses of Umbilical Artery and Vein of Foal.

B, Urinary bladder; *U*, umbilical vein, distended with pus at, *U*, 1, and 1; *UA*, ends of ruptured umbilical arteries, retracted from beyond, 1, at the right. Lying between the ruptured ends of the arteries is the stump of the urachus; 2, abscess in the stump of the right umbilical artery; 5, thrombus in the aorta and iliac arteries.

the abdominal cavity an inch or two from the umbilicus, as indicated in Fig. 193. All three tubes are bound together by connective tissue, which retains its continuity with the connective tissues of the umbilical ring. The connective tissue cord, extending from the broken ends of the urachus and umbilical arteries to the umbilical ring, retains its peritoneal covering, so that the broken ends of the vessels do not communicate with the peritoneal cavity. The remnant of the urachus and its surrounding connective tissue remains as the anterior ligament of the bladder, while the retracted ends of the umbilical arteries constitute the lateral ligaments of that organ. The retraction of the stumps of the arteries increases the thickness of their walls to the point of closing their lumen; their rupture by linear tension frays the broken ends; and the inverted connective tissue, drawn from the umbilical ring, effectively bars hemorrhage from the broken arteries. At the same time, the exalted blood pressure within the umbilical arteries destined to the placenta is transferred to the pulmonary system. Hence the hemostasis is secure

and the ruptured ends of the arteries are withdrawn a generous distance from the exterior. Similarly the stump of the urachus is withdrawn from the exterior and, physiologically, no urine may escape from the umbilical end (fundus) of the bladder. The umbilical vein ruptures within the ring or barely outside. Its thin walls do not markedly retract. The vein being without valves, all blood within the stump spontaneously drops away. This is further assured in natural birth by the licking of the navel by the mother, with her powerful, rasp-like tongue, which also removes the Whartonian gelatin remaining within the amniotic sheath of the cord. The asepsis of the navel wound is completed by the rapid desiccation of the exposed, non-functional tissues. This imprisons and renders harmless any chance bacteria which may be present under usual hygienic conditions. When the young animal is held in a highly polluted environment, as in an extremely filthy stable or yard, the infection may overcome the resistance.

Some influential writers have concluded that the rupture of the bovine navel cord within the vagina of the cow, provides the chief avenue for the invasion of the calf by the infection responsible for calf diarrhea. This hypothesis was possibly borrowed from human obstetrics, based upon the fact that the child, in passing through the vagina of an infected mother acquires gonorrheal ophthalmia, except the conjunctival sac is disinfected. The hypothesis of comparable infection of the bovine navel stump is not warranted by clinical observation. The vagina of no species of domestic animal is believed to carry any infection of such high virulence as gonorrhea. The fetus is further protected against vaginal infections by its membranes. The human amnion commonly ruptures at the cervix and does not materially protrude into the vagina, while the bovine amnion, regularly protruding, before it breaks, about one foot beyond the vulva, provides a comparatively efficient sheath. Hence the navel wound may not contact the vaginal walls.

Hemorrhage from the umbilical arteries is virtually unknown in domestic animals, when the cord is allowed to part in the natural manner, or if the natural process is imitated when the cord is divided artificially. I have observed no instance of navel hemorrhage in any species. A colleague related to me a case in a foal, in which the attendant had cut the cord in two and had ligated it. Apparently it was cut too far from the navel and was unable to retract properly. Stoss[189] refers to possible umbilical hemorrhage. Franck[64] relates a case of bleeding in a foal which he ascribed to the sucking of the navel cord by the dam. It is assumed by some that, if the cord is

divided before respiration has become established, hemorrhage is invited. If, as I learned in one case, the navel cord is ruptured while the fetus is yet entirely within the uterus, copious and fatal hemorrhage is immediate. In swine, however, the navel cord is ruptured or the fetal placenta is detached prior to the expulsion of the young, but umbilical hemorrhage does not occur. When navel hemorrhage occurs, it is to be controlled by compression of the arteries with ligature, suture or forceps.

The equine navel cord, much longer than that of ruminants, usually remains intact after the expulsion of the foal until ruptured by movements of the young or mother. The cord is far larger also than in other species, due chiefly to the very large urachus. The cord is not infrequently diseased, which increases its size. It is stated by numerous writers that the umbilical arteries are firmly bound in the navel ring, and hence cannot retract into the peritoneal cavity. That is in conflict with Fig. 193. I have not observed a foal in which, following spontaneous rupture, the vessels were not retracted as in the calf. Carnivorous young are so primitive in their development that they cannot rupture the cord by their own movements. The cord may rupture within the uterus if the placental contact is sufficiently resistant. Usually it is bitten in two by the mother and the arteries thus lacerated similarly to the parting of the cord by lineal tension. The umbilical cord of the pig is bitten off by the sow, unless ruptured within the uterus.

There is no occasion for ligating, compressing or bandaging the navel cord of normal new-born animals. Instead there is definite peril from ligation: it imprisons the Whartonian gelatin; if the allantoic arteries are caught, they are held out in the danger zone; and the ligation of the vein prevents the escape of any residual blood. All these prevent desiccation of the functionless tissues and invite infection.

It may sometimes be advisable to rupture the cord artificially, especially in the foal. The operation should be performed by linear tension, under aseptic precautions, after respiration has become established and pulsation of the allantoic arteries has ceased. There remains outside the abdomen, after the retraction of the umbilical arteries and urachus, the amniotic sheath of the cord, some Whartonian gelatin, and the collapsed umbilical vein. Normally the dam actively licks the umbilical stump, thereby removing almost all Whartonian gelatin, small blood clots, and water.

The dam may infect the navel during her licking of the fetus. In one case a tubercular cow evaded the watchman at calving time and

licked the navel before the calf had been isolated. Umbilical tuberculosis developed promptly in the otherwise apparently healthy calf, which I ascribed to the licking by the diseased mother. Aside from tuberculosis I am not aware of any peril to the new-born from the natural licking by the dam.

Umbilical infection may be either antenatal or postnatal. The antenatal infection is to be controlled by the application of the principles of sex hygiene already described.

The dangers from umbilical infection after birth are not important in the young of healthy parents born in natural environment. The foal suffers more frequently from umbilical infection than do other domestic young. This is attributed by some to the greater size of the urachus. The mare does not have the prehensile tongue of the cow and cannot so effectively lick the navel of the foal. Instead the mare nibbles at the navel with her lips, and does not so completely remove the large volume of Whartonian gelatin and other water from the stump. If the foal is born in a dirty stall or barnyard, navel infection is not improbable. In one intsance a client bred four valuable draft mares to a famous stallion. All four mares were kept in the barnyard, where they might be closely watched, and all four foals died from umbilical infection. No cases appeared among the foals of his other mares which had been bred to his own stallion and allowed to foal at pasture. I attributed the disaster to the barnyard infection. Later observations permit a second hypothesis—an overworked sire.

When foals are born under questionable environment it is highly essential that the navel wound be aseptically sealed as early as possible. The same principles apply to the navel of other species. The end may be attained directly by disinfection, or indirectly by hastening desiccation of the functionless tissues of the navel stump. I favor the combination of the two methods, and regard the desiccation as the permanent goal. The disinfection is preferably made by the application of tincture of iodine or alcoholic solution of corrosive sublimate. The liquid is placed in a wide-mouthed bottle, a small glass, or other suitable vessel. The foal (or calf) should preferably be held in the standing position and the mouth of the vessel held against the abdominal floor in a manner to submerge the navel stump completely in the disinfectant. The submersion should be continued for two or three minutes. Follow this disinfection with a desiccant powder such as powdered camphor and alum. The camphor is valuable as a fly repellent. Other drugs, such as powdered alum and iodoform, may be used. It is most conveniently applied with the

young animal held upon its back and the powder applied thickly by means of a convenient dusting apparatus. It should be repeated at frequent intervals until the stump of the cord is thoroughly desiccated, after which bacterial invasion is permanently excluded.

It has been stated above that infection may already exist in the navel tissues when the young is born. Most infections are attributed to post-natal invasion. One case of invasion of the navel by tubercle bacilli has been cited as caused by licking by the tuberculous cow. Young dairy calves, placed together and not muzzled, suck each other vigorously just after feeding and seize upon any protruding

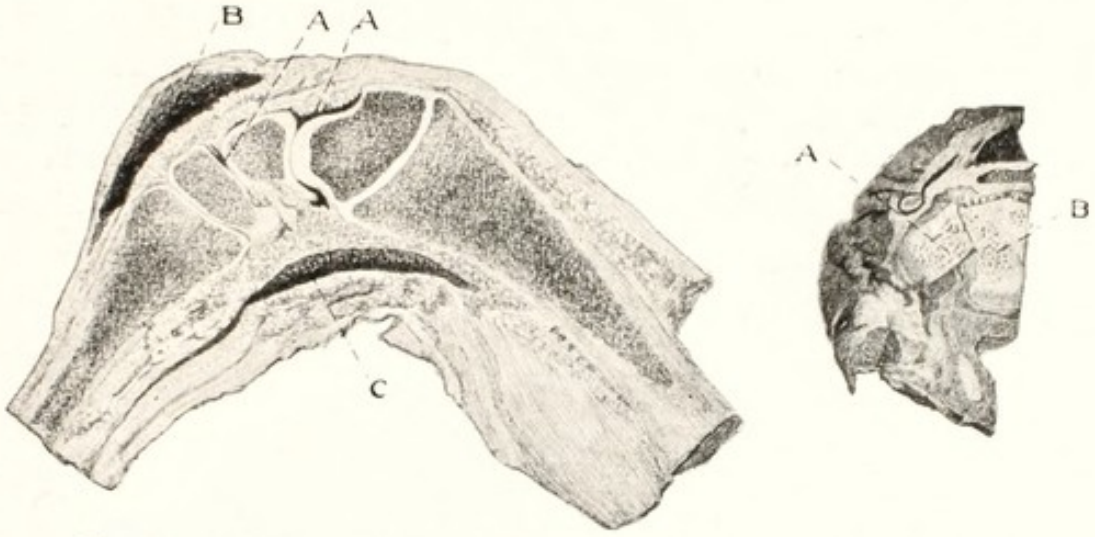


FIG. 194. Arthritis of Carpus of Foal Following Umbilical Infection.

Left, *A*, *A*, Intra-articular abscesses; *B*, precarpal abscess; *C*, postcarpal abscess; right, cross section of left, showing articular surfaces of carpal bones; *A*, *B*, purulent destruction of articular cartilage.

parts-scrotum, teats, or navel stump. Mastitis occasionally follows the sucking of the teats of the heifer calf, and navel infection at times follows the sucking of the navel stump. Most navel infections are ascribed to contact of the navel stump with filth, or to infection by flies.

The most primitive symptoms consist of slight swelling of the navel with purulent discharge, and not infrequently there is a dribbling of urine. Generally the dribbling of urine is vaguely defined as *persistent urachus,* but according to my observations, it is a *reopening* of the ruptured canal, resulting from infection. There are, admittedly, cases of persistent urachus, in which the broken end remains open. This I have not personally observed. As in other septic conditions of the young, diarrhea is common. There is fever, weakness, and inappetence. After it has existed for some days a purulent



FIG. 195. Abscess Within the Third Phalanx of Foal Following Umbilical Infection.

On the left: 1, Fistulous opening from abscess shown at 5, on the right. Right: sagittal section through phalanges; 1, 2, 3, First, second and third phalanges; 4, navicular bone; 5, necrotic area in third phalanx.

fistula may form in the stump of the umbilical vein, as indicated in Fig. 193. The dribbling of the urine keeps the tissues about the navel saturated, aggravating the disease.

Pyaemia becomes established in cases not rapidly fatal. Pyaemic arthritis is perhaps most common in foals. Any articulation may be involved. Thus in Fig. 194 the carpus is involved throughout, and in Fig. 195 pyaemic abscess has formed in the third phalanx. A third example of pyaemic abscess, involving the spinal cord and causing

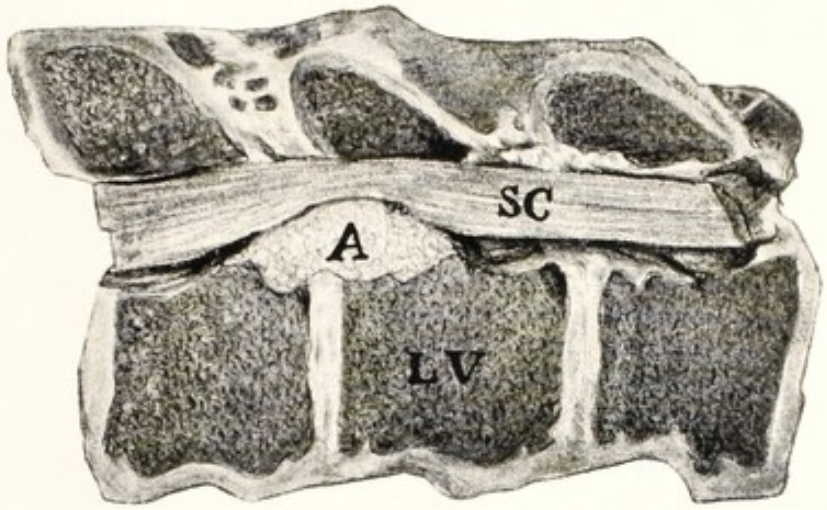


FIG. 196. Pyaemic Abscess in the Spinal Canal of Calf Following Umbilical Infection. *LV*, Lumbar vertebra; *A*, abscess; *SC*, spinal cord.

paralysis, is shown in Fig. 196. There is no limit to the location of such pyaemic lesions. Their symptoms are dependent upon the organ involved. The nature of the disease may be suggested by the age of the animal and the presence of other symptoms of navel infection.

The prognosis for infection of the navel is bad. Some recover in part, but they probably grow into very frail adults which prove valueless. In the earlier stages of the disease, apparent benefit has sometimes followed the use of the dam's blood injected hypodermically. Various antiseptics have been lauded, but their value is open to question.

BIBLIOGRAPHY

1. Abelein, Dr., "Die Trichomonadseuch des Rindes und das Scheidenkatarrh Problem," *Münchener tierärztliche Wochenschrift* (1932), p. 83.
2. Aberhalden, "Die Serodiagnostik der Schwangerschaft," *Deutsche medinische Wochenschrift* (1919), No. 46.
3. Adametz, L. and Schultze, R., "Untersuchungen über die wichtigsten Rassenmerkmalen, der Habitus und den Constitutionstypus der Tux-Zillertaler Rinder mit besondere Berücksichtigung ihrer Wirthschaftlichen Leistung dieser Rasse," *Zeitschrift für Züchtung*. Series B (1932), Vol. xxiii, p. 123.
4. Albrechtsen, Dr., "Die Sterilität des Rindes und ihre Beziehung zu den Ansteckenden Krankheiten der Geschlechtsorgane," Ninth International Veterinary Congress, 1909.
5. Andres, J. "369 Tage Trächtigkeit bei der Kuhe." *Deutsche tierärztliche Wochenschrift* (1931), B 39, S 67.
6. Ascheim and Zondek, "Hypiphysenvorderlappenhormon und Ovarialhormon im Harn von Schwangeren," *Klinische Wochenschrift* (1927), 1322.
7. Bamber, E. and Herdman, E. Catherine, "The incidence of sterility amongst tortoiseshell male cats," *Jour. Genetics* (1932), Vol. xxiv, p. 355.
8. Bang, B., "Aetiologie des seuchenhaften (infektiosen) Verwerfens," *Zeitschrift für Tiermedicin* (1897), Vol. i, p. 241.
9. Beebe, S. P. and James Ewing, "A study of the so-called infectious lymphosarcoma of dogs," *Jour. Med. Research* (1906), Vol. xv, p. 209.
10. Beller, Karl, "Anatomische, physiologische und bakteriologische Untersuchungen der Trächtigkeit und Geburt bei Haustiere," *Deutsche tierärztliche Wochenschrift* (1925), Vol. xxxiii.
11. Beller, Karl, "Künstliche Befruchtung in Turkei," *Münchener tierärztliche Wochenschrift* (1935), Vol. 86, p. 541.
12. Benesch, Franz, "Über Torsio Uteri bei Fleischfresseren und deren Heilung," *Monatshefte für praktische Tierheilkunde* (1922), Vol. xxxiii, p. 138.
13. Bengisch, Georg, "Ovarialzysten und Sterilität der Stuten," *Monatsheft für praktische Tierheilkunde* (1922), Vol. xxxiii, p. 97.
14. Blaha, E. A. "Ansteckenden Scheidenkatarrh der Rinder und 'Trachomkörperchen' bzw. Prowazeksche Körper (Chlamdozoa) bei denselben." *Berliner Tierärztlichen Wochenschrift* (1909) B XXV, S 879.
15. Bland, G. R., "Epizootic abortion experiments," *Oxfordshire County Council Education Committee, Second report* (1911-1916).
16. Bonnet, Robert, *Lehrbuch der Entwickelungsgeschichte.*
17. Boyd, Mossom M., "Crossing bison and cattle," *Jour. Heredity* (1914), Vol. 198.
18. Boyd, W. L., "Mummification of the fetus," *Jour. of Am. Vet. Med. Assoc.* (1924), Vol. lxv, p. 737.
19. Brauer, Carl, "Über das epizootische Verkalben nebst neuer durch viele versuchte erprobte Behandlungsweise," *Deutsche Zeitschrift für Tiermedicin* (1889), Vol. xiv, p. 95.
20. Bruce, E. A., "Nutritional abortion in sheep." *Report Veterinary Director General* (Canada) 1930.
21. Buck, J. M., Creech, G. G. and Ladson, H. H., "Bacterium abortus infection of bulls," *Jour. Agr. Research* (1919), Vol. xvii, p. 239.
22. Cadiot, P. J., *Bul. de la Soc. Centrale de Med. Veterinaire*, Vol. 50, p. 257.
23. Cameron, H. S., Fincher, M. G. and Gilman, H. L., "Trichomonas infection in cattle," *Cornell Veterinarian* (1933), Vol. xxiii, p. 297.
24. Cameron, H. S., "Bovine trichomoniasis," *Bull. 624 Univ. Calif.* (July 1938).
25. Carmichael, J., "Bulldog calf in African cattle," *Nature* (1933), Vol. cxxxi, p. 878.

26. CARPENTER, C. M., "Researches upon a spirillum associated with abortion in ewes," *Annual Report N.Y. State Veterinary College* (1919-1920).
27. CASLICK, E. A., "The sexual cycle and its relation to ovulation, with breeding records of the thoroughbred mare," *Cornell Veterinarian* (1937), Vol. xxvii, p. 187.
28. CATES, J. SIDNEY, "Revolution in the south." *Country Life* (July 1941), p. 18.
29. CESARI, E., "La prophylaxie veterinaire de la fievre mediterraneene," *Bul. de L'Academie Veterinaire de France* (1923), Vol. 76, p. 327. English translation in *Jour. Amer. Vet. Med. Assoc.* (1925), Vol. lxvii, p. 637.
30. CHAPMAN, A. B. and CASIDA, L. E., *Annual Rep. Agr. Expt. Station, Univ. Wis.* (1934-1935), p. 23.
31. CLAPP, HOWARD, "Cystic ovaries and twinning in Holsteins," *Cornell Veterinarian* (1934), Vol. xxiv, p. 309.
32. "Co-operative bull associations," *Farmers Bul. 993, U. S. Dept. Agr.* (1918).
33. CORNER, GEO. W., "Internal migration of the ovum," *Johns Hopkins Bulletin No. 361* (1921), Vol. xxxii.
34. CORNER, G. W., "A case of true lateral hermaphroditism in a pig with functional ovary." *Publication 274,* Carnegie Institution of Washington, pp. 137-142.
35. COTTON, W. E., "New researches and developments on contagious abortion," Twelfth International Veterinary Congress (1934), Vol. i, p. 283.
36. CREWE, A. E., "The significance of an achondroplasie-like condition met with in cattle," *Proceedings Royal Soc.* (1923), Vol. lixv, p. 228.
37. CUILLE, *Revue Veterinaire* (1905), p. 554.
38. CURSON, H. H., "A further note on freemartinism," *Eighteenth An. Rep. Director Vet. Services,* Pretoria, So. Africa (1932), p. 1067.
39. DE BRUIN, M. G. *Geburtshilfe beim Rind* (1897).
40. DELEE, JOSEPH B., *Principles and Practice of Obstetrics,* 2nd Ed. (1936), p. 743.
41. DIMOCK, W. W. and CASLICK, E. A., "Sterility in mares," *Jour. Amer. Vet. Med. Assoc.* (1924), Vol. lxv.
42. DIMOCK, W. W. and EDWARDS, P. R., "Infections of fetuses and foals," *Bul. 333, Kentucky Agr. Expt. Station* (1932).
43. DIMOCK, W. W. and EDWARDS, P. R., "The differential diagnosis of equine abortion with special reference to a hitherto undescribed form of epizootic abortion in mares," *Cornell Veterinarian* (1936), Vol. xxvi, p. 231.
44. DOYLE, T. M. and BECKETT, F., "The isolation of Brucella abortus from the milk of cows with negative reactions to the agglutination test," *Jour. Comparative Medicine and Therapeutics* (1936), Vol. xlix, p. 320.
45. DÜRING, T., "Do the spermatozoa influence the duration of pregnancy in cattle?" *Zeitschrift für Züchtung,* Series B, *Tierzüchtung und Züchtungsbiologie* (1937), Vol. xxxix, p. 25.
46. DUTOIT, J. P. and BISSCHOP, J. H. R., "The breeding of cattle on phosphorus-deficient veld," *Fifteenth Annual Rep. Dir. Vet. Serv., Union So. Africa* (1929), 1061.
46a. DUTOIT, J. P. and BISSCHOP, J. H. R., "The breeding of cattle on phosphorus-deficient veld." *Fifteenth Annual Report Dir. Vet. Service Union So. Africa* (1928), p. 1061.
47. ECKLES, C. H., "A study of breeding records of dairy herds," *Bul. 358, Univ. Minn. Agr. Expt. Station* (1929).
48. EGGIMAN, F., *Schweizer Archiv für Tierheilkunde* (1893), Vol. xxxv, p. 103.
49. ELDER, CECIL, "Control of Bang's disease in Missouri," *University Experiment Station, Bulletin 388* (1936).
50. EMMERSON, M. A., "Trichomoniasis in cattle," *Jour. Am. Vet. Med. Assoc.* (1932), Vol. lxxxi, p. 636.
51. ERRINGTON, J. B. and WILLIAMS, W. L., "The placentation of equine twins," *Cornell Veterinarian* (1933), Vol. xxiii, p. 354.
52. FAVILLE, GEORGE C., "Supposed Maladie du Coït among horses in Nebraska," *8th and 9th Annual Reports, Bureau Animal Industry* (1891-1892), p. 249.

53. FINCHER, M. G. and WILLIAMS, W. L., "Arrested development of the Muellerian ducts associated with inbreeding," *Cornell Veterinarian* (1926), Vol. xvi, p. 1.
54. FINCHER, M. G., "Double uterus; abortion," *Cornell Veterinarian* (1930), Vol. xx, p. 317.
55. FINCHER, M. G., FROST, J. N. and WILLIAMS, W. L., "Two severe cases of dropsy of the amnion and allantois, with a background of severe endometritis in a prior gestation," *Cornell Veterinarian* (1931), Vol. xxi, p. 212.
56. FINCHER, M. G., "Caesarean section, uterine torsion, double uterus," *Cornell Veterinarian* (1934), Vol. xxiv, p. 275.
57. FINCHER, M. G. and WILLIAMS, W. L., "Teratology of the bovine reproductive system," *Cornell Veterinarian* (1937), Vol. xxvii, p. 264.
58. FINCHER, M. G., OLAFSON, PETER, and FERGUSON, JEAN, "Sterility in bulls." *Cornell Veterinarian* (1942), Vol. xxxii, p. 407.
59. FITCH, C. P., DELEZ, A. L., and BOYD, W. L., "Preliminary report on the relation of *Bact. abortus*, Bang, to fistulae, poll evil and other suppurations in horses," *Jour. Am. Vet. Med. Association* (1930), Vol. 29, p. 17.
60. FITCH, C. P. and BISHOP, LUCILLE M., "Bacteriological study of the gravid and non-gravid uterus," *Cornell Veterinarian* (1932), Vol. xxii, p. 225.
61. FLEMING, GEORGE, Animal Plagues (1871).
62. FLEMING, GEORGE, *Veterinary Obstetrics* (1877).
63. FORSTER, J. P., "Extra-uterine pregnancy in a mare," *Cornell Veterinarian* (1918), Vol. viii, p. 127.
64. FRANCK, L., *Handbuch der tierärztlichen Geburtshilfe*, 2nd Ed. (1887).
65. FRIEDBERGER, FRANZ and FRÖHNER, EUGEN, *Lehrbuch der speciellen Pathologie und Therapie der Haustiere*, 12th Ed. (1889).
66. FUCHS, "Die Bedeutung der künstliche Besämung der Rinder für die Bekämpfung der Deckinfektion," *Deutsche tierärztliche Wochenschrift* (1935), p. 230.
67. GALLI, M., "La alterizationi degli organi genitalia esterni et interni dele feminine nate gemelli con un maschio," *Profilassi* (1936), Vol. lx, p. 72.
68. GILLEBEAU, ALFRED, "Über Verletzungen der Haustiere durch sexuell psycopathic Menschen," *Schweizer Archiv für Tierheilkunde* (1899), Vol. xli, p. 1.
69. GOETZE, R., "Über die neuen russischen Methoden der künstlichen Besämung bei Haustieren," *Deutsche tierärztliche Wochenschrift* (1933), vol. 41, p. 801.
70. GOOD, E. S. and SMITH, W. V., "Further investigations of the etiology and control of infectious abortion in mares," *Bul. 204, Kentucky Agr. Expt. Station* (1916).
71. GOOD, E. S., "Control of infectious abortion in mares," *Annual Report U. S. Live Stock Sanitary Assoc.* (1920), p. 85.
72. GOODFELLOW, WM. A., "Abdominal gestation." *Jour. Am. Vet. Med. Assn.* (1942), Vol. c, p. 150.
73. GREIL, ALFRED, "Die Krisis der Zwillingspathologie. Aetiologie der mongoliden Idiotie," *Wiener klinische Wochenschrift* (1935), Vol. xlviii, p. 868.
74. GUARD, W. F., "An unusual case of pregnancy in a cow," *Jour. Amer. Vet. Med. Assoc.* (July 1938), Vol. 93, p. 46.
75. GURLT, *Ueber tierische Missgeburten* (1877).
76. HAGAN, W. A., "The etiology and mode of infection in white scours in calves," *Report N.Y. State Veterinary College* (1916-1917), p. 140.
77. HAMMOND, JOHN A., "When cows do not breed," *The Farmer and Stock Breeder* (April 6, 1936).
78. HAMMOND, JOHN A., "Artificial insemination," *La Res*, Buenos Aires (1936), no. 65.
79. HAMMOND, JOHN A., "Breeding experiments," *Shorthorn Breeders' Review* (1936).
80. HARMS, *Lehrbuch der tierärztlichen Geburtshilfe*, 6th Ed. (1924).
81. HART, GEORGE H., "Genito-urinary infection in sheep," *Report California Agricultural Experiment Station* (1918-1919).
82. HART, GEORGE H. and CARPENTER, C. M., "Controlled vaccination experiments with Bacterium abortum," *Jour. Amer. Vet. Med. Assoc.* (1924), Vol. lxiv, p. 189.

83. HART, GEORGE H. and TRAUM, JACOB, "The relation of the subcutaneous administration of living *Bacterium abortum* to the immunity and carrier problem of bovine infectious abortion," *Technical paper No. 19, University of California Experiment Station* (1925).
84. HART, GEORGE H. and TRAUM, JACOB, "Abortion disease manifestations without the presence of *Bacillus abortus* (Bang)," *Annual Report Div. Vet. Sci., Univ. Calif.* (1922).
85. HELM, R. and ZÜNDI, M., "Die Feststellung der Trächtigkeit mittels der Alkoholreaktion nach Lüttge und von Mertz, und der Hormonmethode nach Dahmen-Wollersheim," *Archiv für wissenschaftliche und praktische Tierheilkunde* (1923), Vol. lviii, p. 503.
86. HENDERSON, J. A., "Observations on reproduction and associated conditions in a herd of dairy cattle," *Cornell Veterinarian* (1938), Vol. xxviii, p. 173.
87. HERTWIG, C. H. "Über die Beschälkrankheit der Pferde," *Magazin für die gesamte Tierheilkunde* (1842), Vol. viii, p. 269.
88. HERTWIG, C. H., "Über Beschälkrankheit," *Magazin für die gesamte Tiereilkunde* (1847), Vol. xiii, p. 373.
89. HESS, DR. E., "Die Sterilität des Rindes und ihre Beziehung zu den ansteckenden Krankheiten der Geschlechtsorgane," 9th International Veterinary Congress, The Hague (1909).
90. HIGGINS, C. H., "Anatomical abnormalities in a bitch." *North American Veterinarian* (1942), Vol. xxiii, p. 46.
91. HILTON, GEORGE, *Report of Veterinary Director General, Canadian Dept. Agr.* (1936-1937), p. 6.
92. HINDMARSH, W. L. and BLUMER, C. C., "Abortion in ewes," *Australian Veterinary Journal* (1932), Vol. viii, p. 149.
93. HOWARTH, J., "A B. coli-like organism causing abortion in sheep," *Cornell Veterinarian* (1932), Vol. xxii, p. 253.
94. HUGHES, E. H. and HART, HUGH, "Defective skulls inherited in swine," *Jour. Heredity* (1934), vol. xxv, p. 111.
95. HUGHES, WINNIFRED, "The freemartin condition in swine," *Anatomical Record* (1938), Vol. xlii, p. 213.
96. HUTT, F. B., "Bovine quadruplets," *Jour. Heredity* (1930), Vol. xxi, p. 339.
97. HUTT, F. B., "Inherited lethal characters in domestic animals," *Cornell Veterinarian* (1934), Vol. xxiv, p. 1.
98. HUTYRA and MAREK, *Pathologie und Therapie der Haustiere.*
99. ISEPPONI, "Beitrag zu den Ursachen der Unfruchtbarkeit der Kühe," *Schweizer Archiv für Tierheilkunde* (1887).
100. IWANOFF, E., *Die künstliche Befruchtung der Haustiere,* Hannover (1912).
101. JENNINGS, WM. E., "Some common problems in horse breeding," *Cornell Veterinarian* (1941), Vol. xxxi, p. 197.
102. JUNGHEER, ERWIN, "Mycotic infections of the bovine reproductive system," *Jour. Amer. Vet. Med. Assoc.* (1935), Vol. lxxxvi, p. 64.
103. KELLER, KARL and TANDLER, JULIUS, "Über das Verhalten der Eihaute bei der Zwillingsträchtigkeit des Rindes," *Wiener tierärztliche Wochenschrift* (1916), Vol. iii, p. 513.
104. KELLER, KARL, "Placentargefässanastomose bei Pferdezwillingen verschieden Geschlechts," *Zeitschrift für Züchtung,* Reihe B, *Tierzüchtung und Züchtungsbiologie* (1934), Vol. xxx, p. 147.
105. KELLER, KARL, "Biologie und Pathologie der Schwangerschaft und Geburt bei den Haussäugetieren," *Halban und Seitz, Biologie und Pathologie des Weibes,* p. 956.
106. KERN, FERDINAND, "Studien über das Wesen der Beschälseuche," *Zeitschrift für Tiermedicin* (1904), Vol. ix, p. 259.
107. KITT, T. H., *Lehrbuch der pathologischen Anatomie der Haustiere,* 5th Ed. (1927).
108. KITSELMAN, C. H.., "A study of Bang abortion live germ vaccines in a beef herd," *Cornell Veterinarian* (1933), Vol. xxiii, p. 309.

109. KOON, G. H. and KESLER, R. R., "The control of equine contagious abortion," *Jour. Amer. Vet. Med. Assoc.* (1922), Vol. lxii, p. 193.
110. KRILL, W. R., "Bicornual pregnancy in cattle," *Jour. Amer. Vet. Med. Assoc.* (1938), Vol. xcii, p. 41.
111. KÜNST, PROF. DR. and UHRIG, DR., "Klinische Beobachtungen über gehäuft auftretende Sterilität und seuchenhaft auftretendes Verkalben in Frühstadium der Trächtigkeit," *Tierärztliche Rundschau,* Nos. 6, 7, and 8 (1932).
112. KÜST, PROF. DR., "Beitrag zur Kentniss der Rachitis- und der Brüllerkrankheit des Rindes," *Tierärztliche Rundschau* (1930), Vol. 36, p. 741.
113. KÜST, PROF. DR., "Die Trichomonadsterilität und ihre Bekämpfung," *Deutsche tierärztliche Wochenschrift* (1936), Vol. xliv, p. 177.
114. LAGERLÖF, NILS, "Morphologische Untersuchungen über Veranderungen im Spermabild und in den Hoden bei Bullen mit verminderter, oder aufgehobenen Fertilität," *Acta pathologica et microbiologica Scandinavica, Supplementum* (1934), Vol. xix.
115. LAGERLÖF, NILS, "Ein Beitrag zur Sterilität in der Rinderzucht," *Zeitschrift für Züchtung* (1935), Vol. xxxii, N. 1.
116. LAGERLÖF, NILS, "Sterility in Bulls," *Veterinary Record* (1936), Vol. xlvii, p. 1158.
117. LEE, A. M. and SCRIVNER, L. H., "An investigation of severe losses from abortion in ewes," *Jour. Amer. Vet. Med. Assoc.* (1938), Vol. 92, p. 68.
118. LEHNDORF, GRAF VON, *Handbuch für Pferde Züchter* (1886).
119. LIEBETANZ, "Untersuchungen über den ansteckenden Scheidenkatarrh (Kolpitis granularis S. Follicularis infectiosa) der Rinder und die Gastroenteritis der Kälber," *Monatsheft für praktische Tierheilkunde* (1901), Vol. xii, p. 551.
120. LIENHART, H. F., KITSELMAN, C. H. and SAWYER, C. E., "Infectious abortion investigations," *Technical Bul. 14. Kansas Agr. Expt. Station* (1925).
121. LILLIE, FRANK R., "The freemartin: a study of the action of sex hormones in the foetal life of cattle," *Jour. Expt. Zoology* (1917), Vol. xxiii, p. 371.
122. LÖFMAN, O. W., "Schwergeburt (*Schistosoma reflexum*) bei einer Stute," *Wochenschrift für Tierheilkunde* (1889), Vol. xxxiii, p. 1333.
123. LÖJE, K., "Letaler Gener (Dodbringende Arvenanlaeg) hos husdyrene, speciely hos Kvag of rod dansk Malkrace," *Tidskrift for Landokonomie* (1930), Vol. x, p. 517.
124. LOTHE, H., "The breeding efficiency of a herd of cows negative to the agglutination test," *Jour. Amer. Vet. Med. Assoc.* (1929), Vol. lxxv, p. 143.
125. LUBBEHUSEN, R. E., FITCH, C. P. and BOYD, W. L., "A study of the value of the living vaccine in the control of bovine infectious abortion," *Cornell Veterinarian* (1926), Vol. xvi, p. 166.
126. LÜTJE, F., *Stang-Wirth,—Tierheilkunde und Tierzucht.*
127. MCCAMPBELL, C. W., *Beef cattle investigations for the year 1919-1920,* Fort Hays (Kansas) Branch Experiment Station.
128. MCFAYDEAN, J. and STOCKMAN, S., "Epizootic abortion in sheep," Board of Agriculture and Fisheries, Great Britain (1913).
129. MCINTOSH, R. A., "A prolific aborter," *Report of Ontario Vet. College* (1924), No. 18.
130. MCKENZIE, FRED W. and PHILLIPS, R. W., "Measuring fertility in the ram," *Jour. Amer. Vet. Med. Assoc.* (1934), Vol. lxxxiv, p. 189.
131. MCNUTT, S. H., WALSH, F. E., and MURRAY, CHARLES, "Trichomonads associated with breeding troubles in cattle," *Cornell Veterinarian* (1933), Vol. xxiii, p. 160.
132. MCPHEE, RUSSELL and ZELLER, "An inbreeding experiment with Poland-China swine," *Jour. Heredity* (1931), Vol. xxii, p. 295.
133. MALL, W. P., "The cause of tubal pregnancy and the fate of the enclosed ovum," *Surg. Gynecol. and Obst.* (1915), Vol. xxi, p. 289.
134. MARSH, H. and TUNNICLIFF, E. A., "Dysentery of new-born lambs," *Bulletin No. 361* (1938) *Montana Agr. Expt. Station.*
135. MEAD, S. W., "A diagrammatic method of presenting the history of reproduction in a dairy herd," *Jour. Dairy Science* (1938), Vol. xxi, p. 283.

136. MEYER, K. F. and EDDIE, B., "The problem of caprine Brucella infections in the United States," *Jour. Amer. Vet. Med. Assoc.* (1935), Vol. lxxxvi, p. 286.
137. MIESSNER, HERMANN, "Wissenschaft und Wirtschaft in der Sowjet-Union," *Deutsche tierärztliche Wochenschrift* (1933), Vol. 41, p. 225.
138. MIESSNER, H. and HARMS, FR., "Virusabort der Stuten," *Deutsche tierärztliche Wochenschrift* (1937), p. 685.
138a. MILKS, H. J., and WILLIAMS, W. L., "Persistence of the right, instead of the left primitive aorta in the dog, incarcerating the esophagus and causing its dilation," *Cornell Veterinarian* (1935), Vol. xxv, p. 365.
139. MILLER, FRED W. and GRAVES, R. R., "Reproduction and health records of the Beltsville herd of the Bureau of Animal Industry," *U. S. Dept. Agr. Technical Bul. 321* (1932), p. 10.
140. MILLER, FRED W., "A technic of artificial insemination of dairy cattle," *Cornell Veterinarian* (1937), Vol. xxvii, p. 103.
141. MILLS, A. M., "Producers' views of inspection and regulations," *Cornell Veterinarian* (1938), Vol. xxvii, p. 103.
142. MOHLER, JOHN R., "Dourine of horses," *U. S. Bureau of Animal Industry Bul. 142* (1911).
143. MOHLER, JOHN R., EICHORN, ADOLPH and BUCK, JOHN M., "The diagnosis of dourine by complement fixation," *Jour. Agr. Research* (1913), Vol. i, p. 99.
144. MOHLER, JOHN R., "Two great animal plagues: abortion disease and tuberculosis," *Cornell Veterinarian* (1938), Vol. xviii, p. 358.
145. MOHLER, JOHN R., E. A. WIGHT and ELMER LASH, "Benefits of eradicating Bang's Disease." *Miscellaneous publication No. 384,* U. S. Department of Agriculture (1940).
146. MOORE, V. A. and FITCH, C. P., "A study of infectious abortion in cattle," *Report N.Y. State Veterinary College* (1912-1913), p. 82.
147. MOUSSU, RAYMOND, "Avortement epizootique des bovides. Un traitement nouveau," *Rec. de Med. Veterinaire* (1935), Vol. cxi, p. 905.
148. MURPHY, DOUGLAS P., "The duplication of congenital malformations in brothers and sisters and among other relatives," *Surg. Gynecol. and Obst.* (1936), Vol. 63, p. 443.
149. NIEBERLE, N., "Die kongenitale Tuberkulose," *Archiv. f. Tierheilkunde,* Vol. 71, p. 347.
150. NOCARD, E., *Recuil de Med. Veterinaire* (1886), p. 675.
151. NORDBY, J. E., "An inherited skull defect in swine," *Jour. Heredity* (1929), Vol. xx, p. 229.
152. NUSSHAG, WILHELM, "Über die Sterblichkeit der Neugeborenen," *Tierärztlichen Rundschau* (1930), Vol. 36, p. 497.
153. NUSSHAG, W., "Über das gehäufte Auftreten einer Missbildung am Fohlen," *Berliner tierärztliche Wochenschrift* (1925), Vol. xli, p. 646.
153a. OETTINGEN, BERNHARD VON, Die Pferdesucht (1918).
154. OSTERTAG, R., "Der anstekende Scheidenkatarrh," *Monatsheft für prakt. Tierheilkunde* (1901), Vol. xii, p. 533.
155. PEARSON, KARL, NETTLESHIP, E. and USHER, C. H., *A monograph on albinism in man,* Dulau and Co., London (1911-1913).
156. PHILLIPS, RALPH W. and MCKENZIE, FRED. F., "The thermo-regulatory function and mechanism of the scrotum," *Research Bulletin 217* (1934), *College of Agriculture, University of Missouri.*
157. PLUM, N., "Tuberculous abortion disease in cattle," *Cornell Veterinarian* (1926), Vol. xvi, p. 237.
158. POMAYER, C., *Zurückhalten der Nachgeburt beim Rind,* Richard Schoetz, Berlin (1908).
159. PRICE, PRESTON A., "New light obtained by a study of primitive races on modern physical degenerations, including dental caries," *Dental Cosmos* (1936), p. 853.

160. PRIDE, W. T., "Retraction ring dystocia," *Surgery, Gynecology and Obstetrics* (1938), Vol. 66, p. 1047.
161. REICHEL, JOHN, "Contagious abortion bacillus vaccine," *Annual Report U. S. Live Stock Sanitary Assoc.* (1912), p. 105.
162. REINDL, W., "Notizen zum enzootische Abortus," *Berliner tierärztlichen Wochenschrift* (1909), B XXV, S 275.
163. REISINGER, L., "Untersuchungen über ein in Österreich gehäuft auftretendes, jedoch nicht durch den Bangischen Bacillus verursachtes Verwerfen der Rinder," *Wiener tierärztliche Monatsschrift* (1928), Vol. xv, p. 49.
164. RICH, R. A., "Methylene blue—a remedy for infectious abortion," *Bul. 174* (1913), *Vermont Agr. Expt. Station.*
165. RICHMOND, F. O., "Large abdominal cyst in a mare," *Amer. Vet. Rev.*, Vol. xxii, p. 770.
166. RICHTER, JOHANNES, *Die Unfruchtbarkeit der Ziegenböcken*, Richard Schoetz, Berlin, 1919.
167. RICHTER, JOHANNES, "Zwillings- und Mehrlingsgeburten bei unseren landwirthschaftlichen Haussäugetieren," *Arbeiten Deutschen Gesellschaft für Züchtungskunde* (1926), Vol. xxix.
168. RICHTER, JOHANNES, "Über Sterilität bei Bullen," *Berliner tierärztliche Wochenschrift* (1932), Vol. xlvii, No. 4.
169. RICHTER, JOHANNES, *Die Sterilität des Rindes*, 4th Ed., Richard Schoetz, Berl'n, 1938.
170. RIEDMÜLLER, L., *Zeitblatt für Bakt. Orig.* (1938), Vol. 128, p. 103.
171. ROBERTS, J. A. FRASER, "The inheritance of a lethal muscle contracture in sheep," *Jour. Genetics* (1929), Vol. xxi, p. 57.
172. ROBERTS, J. A. FRASER, and A. W. GREENWOOD, "An extreme freemartin and a free-martin-like sheep," *Jour. Anat.* (1928), Vol. lxiii, p. 87.
173. RODERICK, LEE M. and G. S. HARSHFIELD, "Pregnancy Disease of Sheep." *North Dakota Expt. Station Bul. 261* (1932).
174. SALMON, D. E., *Annual reports of the Bureau of Animal Industry, U. S. Department of Agriculture* (1886, 1887, 1888).
175. SANDERS, A. H., *Shorthorn Cattle* (1918).
176. SANDERS, D. A., "Some observations on the nature and transmission of enzootic bronchopneumonia (pneumo-enteritis) of dairy calves," *Jour. Amer. Vet. Med. Assoc.* (1939), Vol. xciv, p. 28.
177. SCHÄPER, W., "Todbringenden Erbanlagen (Letalefaktoren) bei Haustieren," *Tierärztlichen Rundschau* (1935), Vol. xli, Nos. 6 and 7.
178. SCHMALTZ, R., *Das Geschlechtsleben der Haussäugetiere* (1921).
179. SEYMOUR, FRANCIS J. and ALFRED KOERNER, "Artificial Insemination," *Jour. Am. Med. Association* (1941), Vol. 116, p. 2747.
180. SISSON, SEPTIMUS, *Veterinary Anatomy.*
181. SLATTER, ERWIN J., "Mummified fetuses in the bovine," *Veterinary Alumni Quarterly*, Ohio State Univ. (1934), Vol. xxii, p. 69.
182. SMITH, THEOBALD, "Spirilla associated with disease of the fetal membranes of cattle (infectious abortion)," *Jour. Expt. Med.* (1918), Vol. xxviii, p. 701.
183. SOHNLE, HUGO, "Untersuchungen über Fohlenlähme," *Monatsheft für prakt. Tierheilkunde* (1901), Vol. xii, p. 337.
184. STANG-WIRTH, *Tierheilkunde und Tierzucht* (1926).
185. STEPHENSON, H. C., SWEET, J. D. and WILLIAMS, W. L., "Torsion of the gravid uterus in a cat," *Cornell Veterinarian* (1931), Vol. xxi, p. 302.
186. STEVENS, J. D., "Report of the first year's progress of the Olympic Peninsula Dairyman's Association Bang's disease project in calfhood vaccination," *North American Veterinarian* (Feb. 1938), p. 35.
187. STEWARD, S. J., "The occurrence of *Onchocerca cervicales* in cases of poll evil and fistulous withers." University of Cambridge Institute of Animal Pathology, Third report (1933).

188. STOCKFLETH, *Deutsche Zeitschrift für Tiermedizin* (1878), p. 167.
189. STOSS, ANTON OTTO, *Tierärztliche Geburtskunde und Gynäkologie* (1928), Ferdinand Enke.
190. STREETER, GEORGE L., "Focal deficiencies in fetal tissues and their relation to intra-uterine amputation," *Publication 414, Carnegie Institution of Washington,* (1930), pp. 1-44.
191. SUNDARAM, S. K., "A brief description of the dairy farm at the Imperial Institute of Agricultural Research at New Delhi," *Indian Veterinary Journal* (1938), Vol. xiv, p. 357.
192. SZANTROCH, Z. and WODZICKI, "Zur Morphologie und Genese des Schistosoma reflexum," *Anatomische Anzeiger* (1930 and 1931), Vol. 71, p. 209.
193. TAUSSIG, J. FRED., *Abortion, Spontaneous and Induced* (1936).
194. THOMAS, J. H. F., "Prolificacy of sheep," *Jour. Ministry Agr.* (1932), p. 19.
195. THOMS, DR., "Der ansteckende Scheidenkatarrh der Rinder unter besonderer Berücksichtigung der pathologisch-histologischen Veranderungen der Scheidenschleimhaut," *Monatsheft für prakt. Tierheilkunde* (1905), Vol. xvii, p. 193.
196. THORP, FRANK JR. and GRAHAM, ROBERT, "The rapid spread of Bang's disease in an accredited herd," *Jour. Amer. Vet. Med. Assoc.* (1933), Vol. xxxii, p. 749.
197. TRAUM, J. and HART, G. H., "Abortion disease manifestations without the presence of *Bact. abortus* (Bang)," *Annual Rep. Division Vet. Sci., Univ. Calif.* (1922).
198. TRAUM, JACOB and HART, GEORGE H., "Manifestations of abortion disease without demonstrable etiological factor," *Calif. Agr. Expt. Station Bul. 353* (1923).
200. TUNNICLIFF, A. E. and MARSH, H., "Bang's disease in bison and elk in the Yellowstone National Park and on the National Bison Range," *Jour. Amer. Vet. Med. Assoc.* (1935), Vol. lxxxvi, p. 745.
201. UDALL, D. H., CUSHING, E. R. and FINCHER, M. G., "Vital statistics of diseases of the genital organs of cows," *Cornell Veterinarian* (1925), Vol. xv, p. 121.
202. UDALL, D. H., FINCHER, M. G. and GIBBONS, W. J., "Case reports," *Cornell Veterinarian* (1928), vol. xviii, p. 367.
203. UDALL, D. H. and WILLIAMS, W. L., "After effects from the dislodgment of the corpus luteum from the ovary of a cow," *Cornell Veterinarian* (1931), Vol. xxi, p. 295.
204. UDALL, D. H., "A peculiar cystic ovary," *Cornell Veterinarian* (1932), Vol. xxii, p. 286.
205. UDALL, D. H., *Veterinary Medicine* (1933), Ithaca, N.Y.
206. U. S. Bureau of Animal Industry, "Co-operative bull associations," *Farmers Bul. 993* (1918).
207. *Report United States Bureau Animal Industry, 1936.*
208. *Univ. Idaho Expt. Station Bul. 297* (1935), "Wrytail is a heritable defect in Jersey cattle."
209. WAGNER, H., "Untersuchungen über Gebärmutter- und Jungtierkrankheiten," *Deutsche tierärztliche Wochenschrift* (1927), Vol. xxxv, p. 655.
210. WAGNER, H., "Die Sterilität in ihrer Abhängigkeit des Organismus," *Archiv für wissenschaftliche und praktische Tierheilkunde* (1931), Vol. 63, No. 4.
211. WAGNER, H., "Sterilität und Vererbung," *Deutsche tierärztliche Wochenschrift* (1932), Vol. xl, p. 547.
212. WAGNER, H., "Untersuchungen über Trächtigkeitsdauer in eine Braunviehherde." *Deutsche tierärzliche Wochenschrift* (1932), Vol. xl, p. 547.
213. WAGNER, H., "Statistisch-Klinische Untersuchungen über die Brunst beim Rind," *Deutsche tierärztliche Wochenschrift* (1931), Vol. xxxix, p. 518.
214. WALLEY, THOMAS, *The four bovine scourges,* London (1879).
215. WALLRAFF, "Enzootische vorkommende Vereiterung der Hoden," *Repertorium der Tierheilkunde* (1846), p. 219.
216. WALSH, F. E., MCNUTT, S. H. and MURRAY, CHARLES, "*Trichomonas bovis* infection of cattle," *Cornell Veterinarian* (1934), Vol. xxiv, p. 60.

217. WALTON, ARTHUR, Ph.D., *The technique of artificial insemination*, Oliver and Boyd, London, 1933.
218. WALTON, ARTHUR, *Notes on artificial insemination of sheep, cattle and horses*, The Holborn Surgical Instrument Co., London.
219. WARWICK, B. L., "Breeding experiments with sheep and swine," *Bul. 480* (1931), *Ohio Agr. Expt. Station*.
220. WATSON, E. A., *Dourine in Canada*, Health of Animals Branch, Department of Agriculture (1920).
221. WAY, CASSIUS, "Functional disorders and their possible relation to breeding efficiency," *Cornell Veterinarian* (1924), Vol. xiv, p. 175.
222. WEBSTER, W. MAURICE, "Bovine sterility in New Zealand," *Australian Veterinary Journal* (1932), Vol. vii, p. 190.
223. WELLS, H. N., "Longevity," *Guernsey Breeders Journal* (1936), Vol. L, p. 445.
224. WELLS, J. G., JR. and RUSSELL E. HOWARD, "What should be the calving interval?" *Quarterly Bul., Mich. Agr. College*, Vol. xxiv (Aug. 1941).
225. WHITE, GEORGE C., RETTGER, LEO F., JOHNSON, ROBERT E. and MCALPINE, JAMES G., "Some economic phases of Bacterium abortum infection," *Bul. 135, Storrs* (Conn.) *Agr. Expt. Station* (1923).
226. WILLIAMS, J. WHITRIDGE, *Obstetrics*, 4th Ed. (1917).
227. WILLIAMS, W. L., "Transverse development of the fetus of the mare," *American Vet. Review* (1889), Vol. xiii, p. 298.
228. WILLIAMS, W. L., "Infectious abortion of mares," *Annual Report U. S. Bureau Animal Industry* (1889-1890), p. 449.
229. WILLIAMS, W. L., "A clinical study of odontomes," *Amer. Vet. Rev.* (1890-1891), Vol. xv, p. 1.
230. WILLIAMS, W. L., "Notes on odontomes," *Amer. Vet. Rev.* (1899-1900), Vol. xxiii, p. 82. Also in *Österreichische Monatschrift für Tierheilkunde* (1899-1900), Vol. xxiv, p. 122.
231. WILLIAMS, W. L., *Veterinary Obstetrics*, including Diseases of Breeding Animals and of the New-born, Ithaca, N.Y., 1909.
232. WILLIAMS, W. L., "Abortion and sterility in cattle," *An. Rep. N.Y. State Vet. Col.* (1911-1912), p. 79.
233. WILLIAMS, W. L., "Contagious abortion in cattle," *An. Rep. N.Y. State Vet. Col.* (1913-1914), p. 29.
234. WILLIAMS, W. L., "The granular venereal disease and abortion in cattle," *Bul. 106, Bureau Animal Industry, U. S. Dept. Agr.* (1914).
235. WILLIAMS, W. L., "Researches upon contagious abortion," *An. Rep. N.Y. State Vet. Col.* (1914-1915).
236. WILLIAMS, W. L., "Researches upon abortion in cattle," *An. Rep. N.Y. State Vet. Col.* (1915-1916), p. 192.
237. WILLIAMS, W. L. and CARPENTER, C. M., "Researches in the diseases of breeding cattle," *An. Rep. N.Y. State Vet. Col.* (1917-1918), p. 51.
238. WILLIAMS, W. L., "Diseases of the genital organs of sheep associated with abortion," *An. Rep. N.Y. State Vet. Col.* (1918-1919), p. 125.
239. WILLIAMS, W. L., "The 1922 report of the Amer. Vet. Med. Assoc. Abortion Committee," *Cornell Veterinarian* (1922), Vol. xii, p. 320.
240. WILLIAMS, W. L., "The male as a spreader of genital disease," *Cornell Veterinarian* (1923), Vol. xiii, p. 108.
241. WILLIAMS, W. L. and WILLIAMS, W. W., "Nymphomania of cows," *North American Veterinarian* (1923), Vol. iv, p. 305.
242. WILLIAMS, W. L., "A study of Hart's article entitled 'Controlled vaccination experiments on cattle with *Bacterium abortum*'," *Jour. Amer. Vet. Med. Assoc.* (1924), Vol. lxv, p. 189.
243. WILLIAMS, W. L., UDALL, D. H., FROST, J. N., CUSHING, E. R., FINCHER, M. G. and MABIE, M. H., "Studies in genital disease," *Cornell Veterinarian* (1924), Vol. xiv, p. 215-374.

244. WILLIAMS, W. L., "A technic for the study of genital disease by means of the fetal membranes," *Cornell Veterinarian* (1925), Vol. xv, p. 255.
245. WILLIAMS, W. L., "Genital diseases of horses," *Cornell Veterinarian* (1926), Vol. xvii, p. 107.
246. WILLIAMS, W. L., "A case of torsion of the bovine uterus with comments upon mummification and calcification of the fetus," *Cornell Veterinarian* (1926), Vol. xvi, p. 280.
247. WILLIAMS, W. L., "Strangulation of the great colon in a mare by its displacement beneath the pregnant uterus," *Cornell Veterinarian* (1927), Vol. xvii, p. 81.
248. WILLIAMS, W. L., "The significance of the duration of pregnancy," *Cornell Veterinarian* (1927), Vol. xvii, p. 331.
249. WILLIAMS, W. L. and GIBBONS, W. J., "The equine vascular allantois," *Cornell Veterinarian* (1929), Vol. xix, p. 3.
250. WILLIAMS, W. L. and SAVAGE, A., "A study of the head length variability of the equine spermatozoa," *Canadian Jour. Research* (1930), Vol. iii, p. 327.
251. WILLIAMS, W. L., *Veterinary Obstetrics*, 1931.
252. WILLIAMS, W. L., "Studies in teratology," *Cornell Veterinarian* (1931), Vol. xxi, p. 25.
253. WILLIAMS, W. L., "Equine and bovine twins," *Cornell Veterinarian* (1931), Vol. xxi, p. 368.
254. WILLIAMS, W. L., "The pathology of reproduction," *Veterinary Medicine* (1932), Vol. xxvii, p. 273.
255. WILLIAMS, W. L., "The trichomonad pest of cattle and the vaginal catarrh problem," *Cornell Veterinarian* (1933), Vol. xxiii, p. 47.
256. WILLIAMS, W. L., "A clinical study of bovine twins and free-martins," *Cornell Veterinarian* (1934), Vol. xxiv, p. 108.
257. WILLIAMS, W. L., "The influence of the ante-natal and early post-natal health of calves upon their vigor and fertility as adults," *Veterinary Record* (1935), Vol. xv, p. 49.
258. WILLIAMS, W. L. and FINCHER, M. G., "Uterus unicornis; retention of menstrual debris," *Cornell Veterinarian* (1935), Vol. xxv, p. 365.
259. WILLIAMS, W. L., "The problem of teratology in clinical veterinary medicine," *Cornell Veterinarian* (1936), Vol. xxvi, p. 1.
260. WILLIAMS, W. L., "Multiple pregnancies in cattle," *Cornell Veterinarian* (1936), Vol. xxvi, p. 207.
261. WILLIAMS, W. L., "Three cases of *Schistocormus reflexus*," *Cornell Veterinarian* (1936), Vol. xxvii, p. 297.
262. WILLIAMS, W. L., "Observations upon some aspects of horse breeding," *Vet. Jour.* (1936), Vol. xcii, p. 119.
263. WILLIAMS, W. L. and FINCHER, M. G., "An equine abdominal fission monster with uterus unicornis and single kidney," *Cornell Veterinarian* (July 1938), Vol. xxviii, p. 252.
264. WILLIAMS, W. L. and J. K. BUSHNELL, "Abscess of the left seminal vesicle in a bull with a fistulous opening into the rectum associated with dilatation of the left ureter and other significant lesions," *Cornell Veterinarian* (1941), Vol. xxxi, p. 63.
265. WILLIAMS, W. W., "Observations upon reproduction in a purebred dairy herd," *Cornell Veterinarian* (1922), Vol. xii, p. 19.
266. WILLIAMS, W. W. and SAVAGE, A., "Observations upon the seminal micropathology of bulls," *Cornell Veterinarian* (1925), Vol. xv, p. 353.
267. WILLIAMS, W. W., "The diagnosis and treatment of tubal disease," *Jour. Amer. Vet. Med. Assoc.* (1924), Vol. lxv.
268. WILLIAMS, W. W., "Tubal patency, cow," *North American Veterinarian* (Nov. 1925), p. 17.
269. WILLIAMS, WILLIAM, *The Principles and Practice of Veterinary Medicine*, 1874.
270. WING, H. H., "The period of gestation in cows," *Bul. 162, Cornell University Agr. Expt. Station* (1899).

271. WING, H. H., "The Cornell University dairy herd, 1889-1928," *Bul. 567, Cornell University Agr. Expt. Station* (1933).
272. WINTERS, L. M., KISER, O. M., JORDAN, P. S. and PETERS, W. H., "A six years study of cross-breeding swine," *Bul. 320, Univ. Minn. Agr. Expt. Station* (1935).
273. WINTERS, L. M., COMSTOCK, R. E., COLE, C. L., GREEN, W. W. and BULIK, J. J., "Artificial insemination of farm animals," *Univ. Minn. Agr. Expt. Station, Bul. 336.*
274. WOODWARD, T. E. and GRAVES, R. R., "Some results of inbreeding grade Guernsey and grade Holstein-Friesian cattle," *Tech. Bul. 339, U. S. Dept. Agri.* (1933).
275. WRIEDT, CHR., "Vererbungsfaktoren bei weissen Pferden im Gestüt Friedriksborg," *Zeitschrift für Tierzüchtung und Züchtungsbiologie* (1924), Vol. i, p. 231.
276. YAMANE, JINSHIN, "Über die Atresia Coli eine letale erbliche Darmmissbildung beim Pferde und ihre Kombination mit Gehirngliomen," *Zeitschrift für inductive Abstammungs- und Vererbungslehre* (1927-1928), Vol. xlvi, p. 188.
278. ZIETZSCHMANN, OTTO, *Lehrbuch der Entwickelungsgeschichte der Haustiere*, Richard Schoetz, Berlin, 1924.
279. ZSCHOKKE, E., *Die Unfruchtbarkeit des Rindes, ihre Ursachen und Bekämpfung*, Zürich, 1900.
280. ZWICK, PROF., and FISCHER, DR., "Untersuchungen über die Beschälseuche," *Arbeiten aus dem Kaiserlichen Gesundheitsamte* (1911), Vol. xxxvi, p. 1.

SUBJECT INDEX

ABBREVIATED gestation, 87
pregnancy, 87
Abbreviation of twin gestation, 88
Abdominal contractions in expulsion of fetal membranes, 565
floor, sloughing of fetus through, 544
Aberhalden test for pregnancy, 201
Abnormal pregnancy, 541
Wolffian ducts, 33
Abnormalities of corpus luteum, 456
Abortion, accidental, 312
and epizootic cellulitis, 225
and inbreeding, 18
as affected by age of female at first breeding, 110
bacillus, 255
bacteria, pathogenicity of, 307
bacterins, alleged immunization by, 315
carbolic acid in, 314
caused by moulds, 340
contagious, 253
contagious, exudate of, 289, 515
control by methylene blue, 314
control by quarantine, 326
control by slaughter, 326
diagnosis of bacterial, 294
diplococci in, 281
due to fungi, 340
endocrines in, 313
exudate, 289, 515
exudate of contagious, 289, 515
from sheep pox, 225
hormones in, 313
immunization against, 315
in foot-and-mouth disease, 225
in influenza, 225
in "pink eye," 225
in sheep, 109, 283
infectious, 253
lesions in tuberculous, 227
lesions of bacterial, 287
paratyphus bacilli in, 281
pyoseptic bacilli in, 281
spirilla in, 283
staphylococci in, 281
sterility, etc., control of, 310
streptococci in, 281
symptoms of bacterial, 286
vaccination of calves against, 321
vaccines, 316
varieties of bacilli in, 281
vibrios in, 283
virus, 281, 363
Abscess of navel, 609
of ovary and tube, 472
of seminal vesicles, 432
of testicle, 396
of umbilicus, 609
of uterus, 577
of vesiculae seminales, 432
tubo-ovarian, 472
Absence of palate in muscle contracture monsters, 16
Acardiac monsters, 61
twins, 61
Accessory male sex glands, diseases of, 432
Accidental abortion, 312
Actinomycosis, genital, 249
Acute orchitis, 395
Adenoma, cystic of ovary, 207
Adherent ovaries, 442
Advanced pregnancy, metritis of, 546
Adventitious placentae, 521
Afterbirth, etiology of retained, 558
handling of retained, 562
manual removal of retained, 562
retained, 549
as cause of vaginitis, 485
in cow, 555
in mare, 552
Age for breeding young animals, 110
Agglutination test for *Bacillus abortus*, 295
Albino eyelids subject to cancer, 18
horses, fertility of, 18
Alcaligines abortus (see *Bacillus abortus*)
Alimentary system of young, physiology and pathology of, 593
Allantochorion, edema of, 530
necrosis of bovine, 515
Allantois, dropsy of, 531
in buffalo-domestic cow hybridization, 11
in bulldog calves, 11
Amnio-allantoic dropsy, 531
in buffalo-domestic cow hybridization, 11
in bulldog calves, 11
in muscle contracture monsters, 15
Tux-Zillertaler cattle, 13
Amnion, dropsy of, 531
Amorphous twins, 58

Ampullae of the vasa differentia, inflammation of, 422
Amputation of fetal extremities by navel cord, 538
penis, 219
Anasarca, 537
Anastomosis of fetal sacs in ruminant twins and multiples, 51, 53
Anatomy of male genital organs, 383
seminal vesicles, 383
Anchylosis, 15
Angio-sarcoma of ovary, 206
Animal plagues and reproduction, 225
Ante-natal and early post-natal rupture of tendons, 585
Aorta, persistence of the right primitive, 18
Arrested development of Mueller's ducts, 26
Arteries, palpation of the uterine per rectum, 198
Arthritis, puerperal pyaemic, 577
pyaemic of new-born, 613
Artificial feeding of young, 593
induction of estrum, 162
insemination, 176
stimulation of uterine and abdominal contractions in retained placenta, 565
Ascheim and Zondek method of diagnosing pregnancy, 201
Asexual twins, 61
Asphyxiation of new-born, 582
of young, 582
Atresia of colon, hereditary, 17
of Graffian follicles, 439
of ovisacs, 439
of uterine cornua, 26
of vagina from irritant douches, 485
of vulva following dystocia, 485
Atretic Graffian follicles, 439
Avenue and date of invasion of abortion bacilli, 296
Avian tuberculosis of bovine genitalia, 245
Avirulent abortion bacillus vaccination, 321

BACILLI, species associated with abortion, 281
Bacillus abortivo-equinus, vaccination against, 324
Bacillus abortus, 255
absence of in chronic metritis of non-pregnant, 473
agglutination test for, 295
complement-fixation test in, 295
immunization against, 315
infection of heifer calves, 303
infection prevented by vitamin E, 149
losses from, Mohler, 5
pathogenicity of when injected into pregnant cows, 307
vaccination, twins following, 55
Bacteria, pathogenicity of in abortion, 307
Bacterial abortion, diagnosis of, 294
lesions of, 287
symptoms of, 286
Bacterial maceration of fetus, 513
Bacterins, alleged immunization in abortion, 315
use of in immunization against abortion, 315
Bacterium abortum (see *Bacillus abortus*)
Bacterium abortus-equi, 281
Bacterium paratyphi abortus equi, 281
Balanitis, 436
Bang's bacillus (see *Bacillus abortus*)
disease (see contagious abortion)
Basic causes of hereditary defects, 9
of interferences with reproduction, 9
of teratology, 9
Benign tumors of ovaries, 209
of oviducts, uterus and cervix, 216
of penis, prepuce and sheath, 219
of testicles, 209
of reproductive system, 209
Bent mummified fetus, 527
Beschälseuche, 341
Bicornual bovine twins, 53
pregnancy, 47
Birth injuries, 569
period, pathological phenomena at, 546
Bison americanus-Bos Taurus hybridization, 11
Bitch, non-pregnant genital organs of, 43
nymphomania in, 455
uterus of, 42
Black corpus luteum, 466
Bladder, rupture of in coitus, 173
Bläschenausschlag, 360
Bone-chewing from defective food, 147
Bovine genital organs, female, 41
hematic mummification, 523
nymphomania, 441
from central cyst, 451
twins, 53
Boyd, buffalo-domestic cow hybridization, 11
Breeding age for young animals, 110
dairy cows to calve each 11.5 months (Elder), 141
of old animals, 117

out congenital or teratological defects, 34
season, 107
Brucella abortus (see *Bacillus abortus*)
Brucellosis (see *Bacillus abortus* infection)
Buffalo-domestic cow hybrid, 11
Bull, sexual load for, 125
Bulldog calves, 11
in Nganda cattle, 13
"Bullers" (see nymphomania), 441
Bursatte, genital, 251

CALCIFICATION of cotyledons, 517
Calcium phosphate and sterility, 313
in food, 146
Calculi in Gartner's ducts, 33
Calf pneumonia, 604
scours, 593
Calves, immunization of against abortion, 321
persistence of *Bacillus abortus* infection in, 303
Calving, frequency of, 129
Cancer of albino eyelids, 18
vagina, 207
Canine uterus, 43, 44
Carbolic acid in abortion, 314
Carcinoma of Gartner's duct, 207
of penis, 208
of vagina, 207
of albino eyelids, 18
Carmichael, studies of bulldog calves, 13
Caruncles, defective uterine, 24
removal of necrotic, 564
uterine, necrosis of, 519
of heifer, 42
Caruncular hemorrhage, profuse, 523
Catheter, rupture of uterus by, 481
Cattle, reproduction in dairy, 4
trichomonad disease of, 352
Causes of retained afterbirth, 558
placenta, 558
Causes of uterine torsion, 492
Central cyst nymphomania, 451
Cerebral herniæ, 19
Cerebrum, herniæ of, 19
Cervical seal, pathological, 482
(see uterine seal)
tuberculosis, 247
Cervicitis, chronic, 482
Cervix, benign tumors of, 216
defective, with retention of estrual debris, 29
malignant tumors of, 207
sclerosis of, 479
Cessation of estrum in pregnancy, 190
Chapman and Casida, re-breeding dairy cows in 30 to 60 days, 134
Chorion, dropsy of, 530
edema of, 530
invaginated, necrotic tip of, 508
necrotic tip of, 507
Chorionitis of pregnancy, 513
of pregnant ruminants, 515
Chronic cervicitis, 482
diseases of the non-gravid uterus, cervix and vagina, 473
metritis, *Bacillus abortus* not incriminated, 473
of non-gravid uterus with pyometra, 474
of non-pregnant female, 473
orchitis, 404
vaginitis and vulvitis, 485
in retained afterbirth, 485
vulvitis, 485
Cleft palate from inbreeding, 18
Clinical diagnosis of pregnancy, 190
Coital exanthem, 360
Coitus, 167
dangers of, 169
false entry in (rectal), 171
frequency of, 125
rupture of vagina in, 173
Colic due to large ovarian cyst, 213
from par-ovarian cyst, 213
Colon bacillus in abortion, 281
hereditary atresia of, 17
impaction of with meconium, 590
strangulation of by gravid uterus, 501
Color and fertility, 18
Complement-fixation test for *Bacillus abortus*, 295
in dourine, 351
Confinement, 152
labor, and housing, 151
Congenital defects, basic causes of, 9
of seminal vesicles, 435
of testicles, uterine caruncles and teats, 24
of vesiculæ seminales, 435
penial, 23
surgical handling of, 34
tuberculosis, 227
Contact necrosis and necrotic tip of chorion, 512
Contagious abortion, 253
agglutination test for, 295
exudate of, 289, 515
sterility, 253
Contraction of tendons, 15
Contractions of uterus in retained placenta, 565

Control of abortion and sterility, 310
dourine, 351
trichomonad disease, 352
Contusion of gluteal and obturator nerves, 570
of vagina, parturient, 570
Copulation, 167
dangers of, 169
false entry in (rectal), 171
frequency of, 125
rupture of vagina in, 172
Corpora nigra, 466
Corpus luteum, abnormalities of, 456
black, 466
cystic degeneration of, 459
diseases of, 456
dislodgment of to cause estrum, 162
embedded, 456
enlarged, 465
hypertrophy of, 465
persistent, 456
retained, 456
Cotyledonitis, 517
Cotyledons, calcification of, 517
necrosis of, 517
Cow, genital organs of, 41
multiple pregnancy in, 53
nymphomania in, 441
profuse caruncular hemorrhage in, 523
retained afterbirth, 555
placenta, 555
twins in, 53
unicornual twins, 51
uterus of, 41
Cows, fertility of after twinning, 54
Crewe, studies of bulldog calves, 12
Cryptorchid testicle, dermoid cyst in, 209
Cryptorchidy, 23
Cyst-adenoma of ovary, 207
Cyst, dermoid of ovaries, 210
dermoid of testicle, 209
of ovary and colic, 213
Cystic degeneration of corpus luteum, 459
of ovisacs, 441
ovaries (nymphomaniac), 441
Cysts, multiple ovarian in fetus and adult, 158
par-ovarian, 213

DAIRY cattle, reproduction in, 4
Dangers of coitus, 169
copulation, 169
Death of fetus, 540
ovum, early, 505
due to defective sex cells, 505
Decomposition, putrid, of the fetus, 543
Defective cervix, retention of estrual debris, 29
descent of testicles, 24
nipples, 24
nutrition of fetus, 92
sex cells as cause of early death of ovum, 505
teats, 24
uterine caruncles, 24
Defects and diseases of new-born, 580
congenital, basic causes of, 9
hereditary, basic causes of, 9
of reproductive system, teratological, 20
Deformed cervix with retention of estrual debris, 29
Degeneration, cystic, of corpus luteum, 459
of tubuli seminiferi, 408
of testicle, 408
Degenerative orchitis, 408
Dermoid cysts of ovaries, 210
of testicle, 209
Destruction of endometrium in chronic metritis, 479
Detachment of tendons from bones in new-born, 585
Dexter cattle and bulldog calves, 11
Diagnosis of bacterial abortion, 294
dourine, 348
pregnancy, clinical, 190
by rectal palpation, 194
laboratory, 201
salpingitis, 467
trichomonad disease, 352
twin pregnancy, 200
Diarrhea in calves, 593
foals, 605
of new-born, 593
Digestive system of young, physiology and pathology of, 593
Diplococci in abortion, 281
Diseases, chronic, of non-pregnant uterus, cervix and vagina, 473
of accessory male sex glands, 422
corpus luteum, 456
Fallopian tubes, 466
female genital organs, 439
female reproductive organs, 439
fetus and uterus as cause of dystocia, 539
Gartner's ducts, 33
glans penis, 436
male genital organs, 383
new-born, 580
oviducts, 466
penial sheath, 436

prepuce, 436
the gravid uterus, 505
the intra-uterine ovum, 505
the ovum, 505
the testicles, 388
various genital organs, 379
Wolffian ducts, 33
puerperal, 573
tubal, 466
Disinfection of uterus in retained fetal membranes, 567
Dislocation of patella in new-born, 588
Dislodgment of corpus luteum after incision of its capsule, 165
followed by hemorrhage, 164
to cause estrum, 162
Displacements of gravid uterus and vagina, 487
pregnant uterus, 487
vagina in pregnancy, 487
Dogs, venereal tumors of, 375
Dorso-ilial position of fetus in uterine torsion, 499
-pubic position of fetus in uterine torsion, 499
Douching of uterus in retained fetal membranes, 567
vagina, atresia of, caused by irritants, 485
Double uterus, 26
Dourine, 341
complement-fixation test in, 351
control of, 351
diagnosis of, 348
prognosis of, 351
symptoms of, 343
Dropsy of allantochorion, 530
following necrosis of caruncles, 530
of allantois and amnion in bulldog calves, 11
amnion and allantois in muscle contracture monsters, 15
in Tux-Zillertaler cattle, 13
chorion, 530
Fallopian tube, 470
fetal membranes, 531
oviduct, 470
Duchess family of shorthorns, inbreeding in, 14
Ducts of Mueller, arrested development of, 26
Wolffian, abnormal, 33
Duration of estrum, 157
gestation, pathological, 86
physiological, 86
"heat," 158
pregnancy as affected by age at first breeding, 110
pathological, 86
Dystocia as result of utero-fetal disease, 539
followed by vulvar gangrene, 486
in *Bison americanus-Bos taurus* hybridization, 11
muscle contracture monsters, 15
Tux-Zillertaler cattle, 13

EARLY breeding of Hereford cows after calving, 134
death of ovum, 505
post-partum breeding and twins, 135
re-breeding after nymphomania, 138
Economic importance of genital tuberculosis, 227
Edema of allantochorion, 530
(Elder) breeding dairy cows to calve each 11.5 months, 141
Embedded corpus luteum, 456
Endocrines in abortion, 313
use of in retained fetal membranes, 566
Endometrium, destruction of, 479
Enlarged corpus luteum, 465
Enzootic orchitis, 402
Epididymitis, 401, 420
Epizootic cellulitus and abortion, 225
Equine abortion vaccination, 324
genital organs, female, 40
twins, 47
placentation of, 47
nymphomania, 451
placentitis, 513
Equus asinus-Equus caballus hybridization, 11
Essential mineral foods, 146
Estrual debris, retention of, 29
Estrum, 157
and hormones, 166
artificial induction of, 162
as affected by the suckling of young, 131
cessation of in pregnancy, 190
coitus and pregnancy, 156
duration of, 159
following calving, 131
hemorrhage from ovary, following, 456
symptoms of, 157
vitamins as cause of, 165
Ethics, the, of handing congenital or teratological defects, 34
Etiology of retained afterbirth, 558
retained placenta, 558
twins, 53

Eversion of uterus, 571
Exanthem, coital, 360
Excess of males with phosphorus-deficient food, 147
Excessive ratio of males in inbreeding, 18
Extensor pedis longus muscle, rupture of in new-born, 585
Extra-uterine fetuses, 498, 541
pregnancy, 541
probably due to uterine torsion, 498
Extreme outcrosses and monsters, 11
as cause of monsters, 11
Exudate of contagious abortion, 289, 515

FALLOPIAN tube, abscess of, 472
dropsy of, 470
inflammation of, 467
benign tumors of, 216
diseases of, 466
pus in, 471
tuberculosis of, 237
False entry in copulation, 171
Fatal hemorrhage from dislodgment of corpus luteum, 163
Feeding, 145
of new-born, artificial, 593
Female genital organs, diseases of, 439
inguinal hernia in, 22
reproductive organs, diseases of, 439
Fertility as affected by color, 18
as influenced by health of young, 96
of cows after twinning, 54
Fetal anasarca, 537
cadaver, sloughing of into rumen, 499
through abdominal floor, 499
static, 541
death, 540
extremities, amputation of by navel cord, 538
giantism, 92
membranes, dropsy of, 531
retained, 549
Fetus
palpation of the, per rectum, 198
bacterial maceration of, 543
defective nutrition of, 92
putrid decomposition of, 543
sloughing of into rumen, 499
trichomonad maceration of, 542
Fetuses, extra-uterine, 498, 541
Fibroma, uterine, 217
Foals, diarrhea in, 605
Foods and feeding, 145
Foot-and-mouth disease and abortion, 225
Free-martins, 61
Fungi as cause of abortion, 340
Fusion of fetal sacs in ruminant twins, 54

GANGRENE of vulva following dystocia, 486
Gartner's ducts, calculi in, 33
carcinoma of, 207
diseases of, 33
lymph in, 33
pus in, 33
General infections interfering with reproduction, 223
Generalized genital tuberculosis, 241
Generative organs, non-pregnant bitch, 43
of mare, 40
of woman, 39
Genital actinomycosis, 249
bursatte, 251
diseases, the problem of, 3
diseases, unclassified, 365
hypoplasia from ill health of new-born, 99
infections, unclassified, 365
organs, diseases female, 439.
of the male, 383
malignant tumors of, 205
of bitch, 44
of cow, 41
of heifer, 42
of mare, 40
specific infections of, 253
tumors of, 203
various diseases of, 379
virus diseases of, 360
pox, 360
system, benign tumors of, 209
hypoplasia of, due to bad health of young, 99
tuberculosis, 227
economic importance of, 227
Gestation, abbreviated, 87
pathological duration of, 86
prolonged, 88
Gigantic fetus, 92
teats, 24
Glans penis, diseases of, 436
tuberculosis of, 230
Glioma, hereditary cerebral, 17
Glista Ernestine, dairy record of, 7
Gluteal nerves, contusion of, 570
paralysis, 570
Gonads, undifferentiated, 70
Graffian follicle, hemorrhage following rupture of, 456

follicles, atretic, 439
pathological rupture of, in fetus, 159
ripening of in estrum, 157
Granular venereal disease, 365
Granulomata, venereal of dog, 375
Gravid uterus, diseases of the, 505
displacements of, 487
of bitch, 44
of sow, 45
strangulation of colon beneath, 501

HANDLING of new-born, 593
of retained afterbirth, 562
of retained placenta, 562
mare, 554
"Heat," 157
artificial induction of, 162
caused by hormones, 166
caused by vitamins, 165
cessation of in pregnancy, 190
hemorrhage from ovary following, 456
periods irregular after calving, 131
Health of new-born, its influence upon fertility, 96
Heifer, genital organs of, 42
reproductive organs of, 42
uterus of, 42
Hematic bovine mummy, 523
Hematoma, intra-uterine, 573
Hemorrhage after dislodgment of corpus luteum, 163
from ovary following estrum, 456
from uterine caruncles (fetal mummification), 523
into ovarian follicles, 455
into follicular crater after ovulation, 456
intra-follicular, 455
intra-uterine, 573
post-coital from urethra, 438
profuse caruncular, 523
Henderson, reproduction in dairy cattle, 4
Hereditary cerebral glioma, 17
defects, basic causes of, 9
hydrocephalus, 17
Hereford cows, early breeding after calving, 134
heifers, early breeding of, 115
Hernia, inguinal, in female, 22
in male, 21
scrotal, 21
umbilical, 20
uterine, 500
Herniæ of cerebrum, 19
Holstein-Friesian heifers, early breeding of, 115
Hormones as cause of estrum, 166
as remedy for retained fetal membranes, 565
in abortion, 313
Housing, confinement and labor, 151
Hybridization, 11
Hydrallantois, 531
Hydramnios, 531
Hydrocephalus, hereditary, 17
Hydrosalpinx, 470
Hymen, imperforate, 33
persistent, 33
Hypertrophy of corpus luteum, 465
Hypoplasia of genital system due to ill health of young, 99
testicular, 388
Hysterocele, 500

IDEAL duration of pregnancy, 86
Impaction of intestines with meconium, 590
meconial, 590
Imperforate hymen, 33
Immunization against abortion, 315
by bacterins, 315
by vaccines, 316
Inbreeding, 14
and abortion, 18
and excess of males, 18
Induction of estrum, artificial, 162
Infection of navel, 607
Infections, general, interfering with reproduction, 223
special of the reproductive system, 255
systemic, which may interfere with reproduction, 223
Infectious abortion, 253
sterility, 253
vaginitis, 365, 475
Infertility of albino horses, 19
of male tortoise-shell cats, 19
Inflammation of ampulla of vas deferens, 422
of Fallopian tube, 467
of ovaries, 441
of oviduct, 467
Influence of health of new-born upon its fertility, 96
Influenza and abortion, 225
Inguinal hernia, 21
Inherited lethal characters, 14
Insemination, artificial, 176
Inter-cornual migration of ova, 46
Interferences with reproduction, basic causes of, 9

Internal generative organs of mare, 40
of woman, 39
Intestines, impaction of with meconium, 590
Intra-follicular hemorrhage, 455
Intra-uterine hematoma, 573
hemorrhage, 573
Invaginated tip of chorion, 508
Invagination of twin chorions in mare, 49
Invasion of abortion bacilli, date of, 296
Inversion of uterus, 571
Irregularity of estrum after calving, 131
Irritating douches as cause of atresia of vagina, 485

KERRY cattle and bulldog calves, 11
Kidneys, puerperal inflammation of, 577

LABOR, 151
housing and confinement, 151
traumatic hemorrhage in, 570
Laboratory diagnosis of pregnancy, 201
Lacerations and rupture of vagina in coitus, 172
of vagina in coitus, 172
parturient, 570
Laminitis in retained placenta, 553
parturient, 553
Lesions of bacterial abortion, 287
of tuberculous abortion, 227
Location of ova in uterus, 45, 47
Lubbehusen, Fitch and Boyd, twins following vaccination against abortion, 55
Lympho-sarcoma, venereal of dog, 375

MACERATION of fetus in uterine torsion, 499
trichomonad, 358, 542
Maladie du Coit, 341
Male, anatomy of the genital organs of the, 383
diseases of accessory organs, 422
genital organs, anatomy of, 383
genital organs, diseases of, 383
inguinal hernia in, 21
number of females he may serve, 125
reproductive organs, diseases of, 383
scrotal hernia in, 21
Males, excessive ratio in inbreeding, 18
excessive ratio with phosphorus-deficient food, 147
Malignant ovarian tumors, 205
tumors of penis, 208
of reproductive organs, 203
of testicles, 205
of uterus, cervix and vagina, 207
Manual removal of retained afterbirth, 562
Mare, genital organs of, 40
nymphomania in, 451
placentation of twins in, 49
reproductive organs of, 40
retained afterbirth in, 552
placenta in, 552
twins in, 49
twin ovulation in, 49
twin pregnancy in, 49
Mares, breeding at 9 days post-partum, 129
uterus of, 40
McCampbell, studies in early breeding of Hereford heifers, 115
McPhee, Russell and Zeller, inbreeding of swine, 18
Meconium, impaction of, 590
Meningitis, puerperal, 577
Menstrual debris, retention of, 29
Methylene blue in control of abortion, 314
Metritis, chronic of non-pregnant female, 473
of advanced pregnancy, 546
of pregnancy, 513
Micrococcus melitensis (see *Bacillus abortus*)
Migration of ova, intercornual, 46
Mineral foods, 146
Minerals in control of abortion, 313
Molds and abortion, 340
Mohler, J. R. Bang-free cows to produce calves each 11½ months, 141
Mohler, losses caused by *Bacillus abortus*, 5
Moore and Fitch, injection of abortion bacilli into pregnant cows, 307
Moussu, claim that vitamin E prevents contagious abortion, 149
Muellerian ducts, persistence of median walls of, 26
arrested development of, 26
Mule, 11
rarity of cryptorchidy in, 23
Multipara, unicornual torsion of uterus in, 495
Multiple ovarian cysts in fetus and adult, 158
ovulation, 49
pregnancy in unipara, 53
Mummification, 15
hematic, 523
papyraceous, 541

Mummified fetus, 523
bent, 527
Mummy, bovine hematic, 523
Muscle contracture monsters, 15
Muscles, separation of from tendons in new-born, 585
Musculo-tendinous ruptures in new-born, 585

NAVEL, abscess of, 609
cord, amputation of fetal extremities by, 538
torsion of, 537
ill, 607
Necrosis, contact, in necrotic tip of chorion, 512
of bovine allantochorion, 515
of uterine caruncles, 519
Necrotic stomatitis, 605
tip of chorion, 507
and contact necrosis, 512
and *Schistosomus reflexus*, 509
invaginated, 508
uterine caruncles, removal of, 564
Nephritis, pyelo-, 577
Nerves, contusion of gluteal, 570
of obturator, 570
New-born, asphyxiation of, 582
defects of, 580
diseases of, 580
feeding and handling of, 593
handling of, 593
infection of navel of, 607
influence of its health upon its fertility, 96
pyæmic infection of, 607
sepsis of, 584
umbilical infection of, 607
Nganda cattle, bulldog calves and amnio-allantoic dropsy in, 13
Ninth-day breeding of mares, 129
Nodular vaginitis, 365
venereal disease, 365
Non-gravid uterus, cervix, and vagina, diseases of, 473
of bitch, 43
Non-pregnant uterus, cervix and vagina, diseases of, 473
of bitch, 43
Nursing and estrum, 131
Nutrition of fetus, defective, 92
Nymphomania, 441
cow, 441
due to small, central cyst, 451
early re-breeding after, 138
equine, 451
in bitch, 454
in mare, 451
in small animals, 455
in sow, 453

OBSTRUCTION colic from par-ovarian cyst, 213
Obturator nerves, contusion of, 570
paralysis, 570
Old animals, breeding of, 117
Omphalo-phlebitis, 607
Open urachus, 613
Optimum duration of pregnancy, 87
Orchitis, 394
acute, 395
chronic, 404
degenerative, 408
enzootic, 402
purulent, 400
tubercular, 229
Outcrosses, extreme, as causes of monsters, 11
causing bulldog calves, 11
Ova, location of in uterus, 45, 47
Ovarian cyst, small central causing nymphomania, 451
causing incarceration of rectum, 213
cysts, multiple in fetus and adult, 158
hemorrhage after ovulation, 456
scalpel, 165
Ovaries, adherent, 442
benign tumors of, 209
cystic degeneration of, 441
inflammation of, 441
malignant toumors of, 205
tuberculosis of, 237
Ovaritis, 441
Ovary, abscess of, 472
Oviduct, abscess of, 472
dropsy of, 470
inflammation of, 467
Oviducts, benign tumors of, 216
diseases of, 466
pus in, 471
tuberculosis of, 237
Ovisacs degeneration of, 439
pathological rupture of in fetus, 158
ripening of in "heat," 157
Ovulation during pregnancy, 47
multiple, 49
pathological, 37
teratological, 37
twin, 49
Ovum, early death of, 505

PALATE absent in muscle contracture monsters, 16
cleft, from inbreeding, 18
Palpation of the fetus per rectum, 198
per rectum in the diagnosis of pregnancy, 194
of uterine arteries per rectum, 198
Papyraceous mummification, 541
Paralysis, gluteal, 570
obturator, 570
Par-ovarian cysts, 213
as cause of obstruction colic, 213
Paratyphus bacillus in abortion, 281
Patella, dislocation of in new-born, 588
Parturient injuries, 569
and contusions of vagina, 570
laminitis, 553
Pathogenicity of bacteria associated with abortion, 307
Pathological cervical seal, 480
duration of gestation, 86
of pregnancy, 86
ovulation, 37
phenomena at birth period, 546
rupture of ovisacs in fetus, 158
spermatozoa, 76
spermatogenesis, 37, 76
uterine seal, 480
Pathology of digestive system of new-born, 593
of reproduction, basic causes of, 9
of semen, 411
of spermatozoa, 411
Pavilionitis, 467
Penial defects, 23
lymph nodes, tuberculosis of, 233
tuberculosis, 233
Penis, amputation of, 219
benign tumors of, 219
carcinoma of, 208
congenital defects of, 23
diseases of glans, 436
of sheath of, 436
entry of rectum by, in copulation, 171
malignant tumors of, 208
Pennsylvania plan of abortion control, 330
Permanent fetal *rigor mortis*, 494, 541
in uterine torsion, 494
Perineum, rupture of, 569
Peri-penial lymph glands, tuberculosis of, 233
Persistence of right primitive aorta, 18
Persistent corpus luteum, 456
hymen, 33
urachus, 613
Phosphates as food elements, 146
as remedy for sterility, 313
Physical injuries from coitus, 168
from copulation, 168
Physiology and pathology of digestive system of new-born, 593
"Pink-eye" and abortion, 225
Placenta, etiology of retention, 558
handling of retained, 562
manual memoval of retained, 562
retained, 549
as cause of vaginitis, 485
in cow, 555
in mare, 552
wearing out of, 117
Placentæ, adventitious, 521
anastomosis of in ruminant twins and multiples, 53
uterine of heifer, 42
Placental exhaustion, 117
Placentation in bovine twins, 53
equine twins, 49
twin, in mare, 49
Placentitis, equine, 513
of pregnancy, 513
of pregnant ruminants, 514
Pneumonia, calf, 604
Polyhydramnios, 531
Porcine uterus, 45
Post-coital urethral hemorrhage, 438
Post-ovulation hemorrhage into follicular crater, 456
Post-partum rest interval, 129
Pox, genital, 360
Pregnancy, 189
abbreviated, 87
advanced, metritis of, 546
bicornual, 47
cessation of estrum in, 190
chorionitis of, 513
clinical diagnosis of, 190
diagnosis of twin, 200
by rectal palpation, 194
disease of sheep, 109
duration of as affected by breeding primipara, 110
metritis of, 513
optimum duration of, 87
ovulation during, 47
pathological duration of 86
placentitis in, 513
prolapse of vagina in, 502
prolonged, 88
symptoms of, 190
tubal, 38, 46, 467
twin in mare, 49

Pregnant ruminant, chorionitis and placentitis of, 514
uterus causing strangulation of colon, 501
diseases of the, 505
displacements of, 487
of bitch, 44
of sow, 45
Prepubian tendon, rupture of, 500
Prevention of retained placenta, 560
Prepuce, diseases of, 436
benign tumors of, 219
tuberculosis of, 232
Primipara, influence of age at breeding, 110
in breeding Herefords, 115
in breeding Holstein-Friesians, 114
Primitive aorta, persistence of right, 18
Problem of genital disease, 3
Profuse caruncular hemorrhage, 523
Prognosis of dourine, 351
Prolapse of vagina in pregnant female, 502
of uterus, 571
Prolonged pregnancy, 88
Prophylaxis of retained placenta, 560
Prostate, tuberculosis of, 229
Prostatitis, 436
Protozoa interfering with reproduction, 341
Puerperal abscesses, pyæmic, 577
diseases, 573
displacements of urino-genital organs, 571
meningitis, 577
pyæmia, 577
pyæmic arthritis, 577
pyometra, 576
tetanus, 575
Purulent orchitis, 400
inflammation of Gartner's ducts, 33
Pus in Gartner's ducts, 33
in oviducts, 471
Putrid decomposition of fetus, 543
with sloughing through abdominal floor, 544
Pyæmia, puerperal, 577
Pyæmic arthritis of new-born, 613
infection of new-born, 607
Pyelo-nephritis, 577
Pyometra in metritis of non-pregnant female, 474
in pregnant uterus (see trichomonad disease), 474
of trichomonad disease, 358
puerperal, 576
Pyosalpinx, 471
Pyosepticum bacillus in abortion, 281

QUARANTINE and slaughter in abortion control, 326

RAINFALL, its influence on reproduction, 98
Rapidity of breeding, 125
Rectal palpation in the diagnosis of pregnancy, 194
Rectum, entry of penis in copulation, 171
incarceration of by par-ovarian cyst, 213
impaction of with meconium, 590
palpation of genital organs per, 194
strangulation of by par-ovarian cyst, 213
Removal of necrotic uterine caruncles, 564
Reproduction and animal plagues, 225
as influenced by rainfall, 98
basic causes of interferences with, 9
general infections interfering with, 223
infectious diseases interfering with, 223
influenced by health of new-born, 96
interferences by protozoa, 341
pathology of in perspective, 3
Reproductive cells, pathology of, 9
teratology of, 9
organs, cow, 41
diseases of female, 439
male, 383
heifer, 42
of mare, 40
of woman, 39
specific infections of, 253
senility of, 117
system, rectal palpation of, 194
special infections of the, in perspective, 255
teratological defects of, 20
tumors of, 203
virus diseases of, 360
Rest interval of female after parturition, 129
Retained afterbirth, 549
a cause of chronic vaginitis and vulvitis, 485
and laminitis, 553
cause of, 558
cow, 555
douching of uterus in, 567
handling of, 562
in mare, 552
incurable, 562
manual removal of, 562
prevention of, 560

stimulation of uterine contractions in, 565
use of endocrines in, 566
use of hormones in, 566
Retained corpus luteum, 456
fetal membranes, 549
douching of uterus in, 567
use of disinfectants in, 567
placenta, 549
and laminitis, 553
as cause of vaginitis, 485
cause of, 558
cow, 555
douching of uterus in, 567
handling of, 562
in mare, 554
incurable, 562
manual removal of, 562
prevention of, 560
use of endocrines in, 566
use of hormones in, 566
Right primitive aorta, persistence of, 18
Rubin apparatus, in determining patency of oviducts, 469
Rumen, sloughing of fetus into, 545
Ruminant twins and multiples, anastomosis of fetal sacs, 51, 53
Ruminants, chorionitis of pregnant, 514
placentitis of pregnant, 514
Rupture of bladder in coitus, 173
of perineum, 569
of rectum in coitus, 171
of the prepubian tendon, 500
of vagina in coitus, 172
of uterus by catheter, 481
by coitus, 173
from torsion, 494
in labor, 569
in multipara, 495
of vagina in uterine torsion, 494

SADISM and sodomy, 173
Salmonella abortus-equi (see *Bact. abortus-equi*), 281
Salpingitis, 467
diagnosis of, 469
"Sand," uterine, 530
Sanders, shorthorn cattle, 14
Sarcoma of genital tract, 208
of ovary, 206
Scalpel, ovarian, 165
Schistosomus reflexus and the necrotic tip of the chorion, 509
Sclerosis of cervix and uterus, 479
Scours, calf, 593
Seal, cervical (see uterine seal)
uterine in pregnancy, 191
Semen in testicular hypoplasia, 388
pathology of, 411
Seminal vesicles, abscess of, 432
anatomy of, 383
congenital defects in, 435
diseases of, 422
purulent inflammation of, 430
tuberculosis of, 229
Seminiferous tubules, degeneration of, 408
Semino-vesiculitis, 422
Senility in breeding animals, 117
Sepsis of new-born, 584
Sex cells, early death of ovum when defective, 505
undifferentiated, 70
Sexual glands, diseases of male accessory, 422
intercourse, dangers of, 168
load for male, 125
Sheath, benign tumors of, 219
of penis, diseases of, 436
Sheep, abortion in, 109
nutritional abortion in, 109
pox and abortion, 225
pregnancy disease of, 109
twins and multiples in, 53
Sire, number of females to be served, 125
"Sleeper" foals, 584
Slaughter in control of abortion, 326
Sloughing of fetal cadaver into rumen, 499, 545
through abdominal floor, 499
Small animals, nymphomania in, 455
Sodomy and sadism, 173
Sow, nymphomania in, 453
uterus of, 45
Special infections of the reproductive system in perspective, 255
Specific contagions of genital organs, 253
infections of genital organs, 253
of reproductive system, 253
Spermatogenesis, pathological, 76, 37
teratological, 37
Spermatozoa, defective, as cause of early death of ovum, 505
in testicular hypoplasia, 388
pathological, 76
pathology of, 411
staining of, 413
Spirilla in abortion, 283
Stabling (confinement), 151
Staining of spermatozoa, 413
Stallion, sexual load for, 125
Staphylococci in abortion, 281

Static fetal cadaver, 494, 541
and uterine torsion, 494
Steinkalb, 523
Sterility and abortion, control of, 310
contagious, 253
due to endocrine deficiency, 313
endocrines in, 313
from tubal disease, 467
hormones in, 313
infectious, 253
Stomatitis, necrotic, 605
Strangulation of colon beneath gravid uterus, 501
of rectum by par-ovarian cyst, 213
Streptococci in abortion, 281
Suckling of young and delay of estrum, 131
"Summer sores" (see genital bursatte)
Surgical interference with congenital defects, 34
Swine fetuses, extra-uterine, 498
Symptoms of bacterial abortion, 286
of dourine, 343
of pregnancy, 190
of trichomonad disease, 355
Systemic infections interfering with reproduction, 223

TENDONS, contraction of, 15
Teratological defects of reproductive system, 20
surgical handling of, 33
ovulation, 37
spermatogenesis, 37
Teratology in reproduction, basic causes of, 9
Testicles, abscess of, 396
benign tumors of, 209
defective descent of, 24
dermoid cyst of, 209
diseases of, 388
hypoplasia of, 388
malignant tumors of, 205
tuberculosis of, 229
Testicular degeneration, 408
hypoplasia, 388
Tetanus, puerperal, 575
Tip of chorion, necrotic, 507
Torsion, uterine, 488
of navel cord, 537
of umbilical cord, 537
of uterus, causes of, 492
and dorso-pubic position of fetus, 499
Tortoiseshell male cats, infertility of, 19
Traumatic hemorrhage in labor, 570
Trichomonad disease of cattle, 352
control of, 358
diagnosis of, 356
maceration of fetus in, 358, 542
disease, pyometra of, 358
symptoms of, 355
Trichomoniasis, bovine, 352
Tubal pregnancy, 38, 46, 467
disease, diagnosis of, 469
diseases, 466
disease, sterility from, 467
Tubercular orchitis, 229
Tuberculosis, avian of bovine genital system, 245
of cervix, 247
congenital, 227
of Fallopian tubes, 237
generalized of reproductive system, 241
genital, 227
of glans penis, 229
of the ovaries, 237
of oviducts, 237
of penis, 229
of prepuce, 233
of prostate, 229
of reproductive system, 227
of seminal vesicles, 229
of testicles, 229
of vas deferens, 229
Tuberculin test, failure of in genital tuberculosis, 227, 248
Tubo-ovarian abscess, 472
Tubuli seminiferi, degeneration of, 408
Tumors, benign of cervix, 216
of genital organs, 203
of oviducts, 216
of penis, 219
of prepuce, 219
of reproductive system, 203
malignant of ovaries, 205
of penis, 208
of uterus, cervix and vagina, 207
of the reproductive system, 203
of testicles, 205
venereal of dogs, 375
Tux-Zillertaler cattle and amnio-allantoic dropsy, 13
Twin ovulation, 49
in diseased females, 55
pregnancy, the diagnosis of, 200
Twinning, the causes of, 56
Twins, acardiac, 61
and free-martins, 61
amorphous, 61
asexual, 61
bicornual, 53

bovine, placentation in, 53
causes of, 56
equine, placentation of, 49
fertility of, 56
following abortion vaccination, 55
placentæ of in mare, 49
sex of, 56
unicornual, 53
Twisting of navel cord, 537

UMBILICAL cord, amputation of fetal extremities by, 538
torsion of, 537
infection, 607
veins, abscess of, 609
Unclassified genital diseases, 365
Unicornual twins, 53
uterine torsion in multipara, 495
Undependability of tuberculin test in advanced genital tuberculosis, 227, 248
Undifferentiated gonads, 70
sex glands, 70
Urachus, open, 613
persistent, 613
Urethral hemorrhage, post-coital, 438
Uterine abscess, 577
arteries, palpation of per rectum, 198
caruncles, necrosis of, as cause of dropsy of chorion, 530
defective, 24
heifer, 42
necrosis of, 519
removal of necrotic, 564
contractions, stimulation of in retained placenta, 565
cornua, atresia of, 26
fibroma, 217
hernia, 500
insufflation, Rubin method, 469
placentæ, heifer, 42
"sand," 530
seal in pregnancy, 191
pathological, 480
torsion, 488
and dorso-ilial position of fetus, 499
and dorso-pubic position of fetus, 499
causes of, 492
maceration of fetus in, 499
unicornual in multipara, 495
with static fetal cadaver, 494
Utero-fetal disease and dystocia, 539
Uterus, benign tumors of, 216
canine, 43, 44
chronic diseases of non-pregnant, 473
defective caruncles of, 24
didelphys, 26
diseases of the gravid, 505
of the pregnant, 505
double, 26
douching of in retained fetal membranes, 567
eversion of, 571
gravid, displacements of, 487
inversion of, 571
location of ova in, 47
malignant tumors of, 207
of bitch, non-pregnant, 43
pregnant, 44
of cow, 41
of heifer, 42
of mare, 40
of sow, 45
of woman, 39
one-horned, 26
pregnant, displacements of, 487
prolapse of, 571
rupture of by catheter, 481
in coitus (?), 173
in labor, 569
in parturition, 569
sclerosis of, 479
torsion of, 488
tuberculosis of, 241
unicornis, 26

VACCINATION against abortion, 316
contagious abortion, twins after, 55
in equine abortion, 324
of calves with avirulent abortion bacilli, 321
Vaccines in abortion, 316
Vagina, atresia of due to irritant douching, 485
carcinoma of, 207
displacement of in pregnancy, 487
malignant tumors of, 207
parturient contusions of, 570
lacerations of, 570
prolapse of in pregnancy, 502
rupture of in coitus, 173
Vaginal douching, atresia caused by irritants, 485
Vaginitis, atretic, from irritant douches, 485
chronic, 485
infectious, 365
nodular, 365
Vascular anastomosis of fetal sacs of bovine twins, 53
Vas deferens, anatomy of, 383
inflammation of ampulla of, 422
tuberculosis of, 229

Venereal disease, granular, 365
vesicular, 360
granulomata, dog, 375
lympho-sarcoma of dog, 375
tumors of dogs, 375
Vesiculæ seminales, anatomy of, 383
congenitals defects in, 435
diseases of, 422
purulent inflammation of, 430
abscess of, 432
Vesicular veneral disease, 360
Veins, navel, abscess of, 607
Vibrionic abortion, 283
Virus abortion, 281, 363
diseases of the reproductive system, 360
Vitamin E alleged to prevent contagious abortion, 149
in control of abortion, 313
Vitamins, 148
and estrum, 165
Vulvar gangrene following dystocia, 486
Vulva, constriction of the, 486
gangrene of, after dystocia, 486
hypoplasia of the, from defective nutrition in calves, 99
Vulvitis, chronic, 485

WATER calf," 537
Wing, inbreeding, 14
reproduction in dairy cattle, 4
Winters, Kiser and Jordan, outcrossing of swine, 18
Wolffian ducts, abnormal, 33
Woman, internal generative organs of, 39
Woodward and Graves, inbreeding cattle, 18
Wriedt on fertility in albino horses, 19

YAMANE, atresia of colon, 17
"Yellow body" diseases of, 456
retained, 456
Young animal, the influence of its health upon fertility, 96
animals, age at first breeding, 110
feeding of, 593
infection of navel of, 607

WELLCOME LIBRARY INSTITUTE