General Session

Dr. James Jarrett, Chairman

Genetics and Disease in Cattle

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Many different congenital defects, either of genetic, environmental, or unknown cause, or due to environmental-genetic interaction, have been identified in domestic, zoo and feral animals (16,37,90, 91,95,125, 154,156,157, 198,199). Congenital defects may be compatible with life but many are lethal and thus comprise an important part of neonatal mortality. The practicing veterinarian is the major source of further information for diagnosing and controlling these troublesome diseases. It is important that veterinarians be able to diagnose congenital defects which are of economic significance to the cattle breeding industry. Not only is diagnosis important but practicing veterinarians should be knowledgeable of methods for controlling genetically-induced defects in cattle. Congenital defects have been identified in all major cattle breeds; many more undoubtedly exist and await recognition and control. Most cattle breed associations and artificial breeding organizations have programs for controlling undesirable traits and genetic defects (151,155). This paper discusses definition, frequencies, causes, control and nature of congenital defects in cattle.

Definition

Congenital defects are defined as abnormalities of structure or function present at birth. This definition denotes time but not causes of congenital defects. Frequently, congenital is used synonymously with genetic, but this is not feasible as many, but not all, congenital defects are genetic. The definition excludes those structural and functional variants commonly identified by cattle breeders as undesirable in their breeding program. Distinction between abnormalities and normal variants may not always be obvious (209). Congenital defects are defined as being present at birth but many are not identifiable until later or are not identifiable without clinical, biochemical, or pathological examination. Many congenital defects affect a single structure or function; however, we are becoming increasingly aware of the diagnostic importance of syndromes consisting of a combination of various structural defects or the association of functional and structural defects.

Genetic variation is the means of evolution and the tool of animal breeders. Along with favorable variations there are many that are unfavorable and result in congenital defects. Congenital defects comprise a fair percentage of neonatal loss and may be of considerable economic importance. Genetic diseases may spread insidiously through a cattle breed until they are difficult to control. It is desirable to recognize such diseases early to prevent spread.

Frequency

Frequency of congenital defects in cattle varies according to breed. Within breeds, factors such as function or structure affected, level of nutrition, environment, age of parents, management and others modify the total number of congenital defects encountered and the frequency of individual defects diagnosed. In addition, congenital defects are either caused by genetic or by environmental factors or by their complex interaction. Thus, defects of each structure or function vary among breeds and families within breeds. Overall frequencies of congenital defects as well as those of structure or function affected are difficult to obtain as many defects are identified only by necropsy examination. Many defects go unnoticed and others are not reported for economic reasons. Others occur so rarely as to defy accurate accounting, and frequent reporting of other defects may reflect high interest of the observer rather than high incidence of the defect. However, several recent studies in various parts of the world indicate that congenital defects of the central nervous system, skeletal system and muscular system are the ones most frequently encountered. One-half to one percent of the calf crop has congenital defects (125,156,191-193,196). Although economic losses due to congenital defects

are less than losses due to diseases caused by nutritional or infectious agents, defects may cause considerable economic losses to individual cattle owners. Congenital defects due to genetic causes are particularly important because losses may be repeated generation after generation.

Losses of newborn dairy calves from stillbirths in DHIA herds in the United States amount to something over 6% (19). In 5,258 births reported from Kansas AI herds in 1954-1955, 2.1 calves in every 1,000 had congenital defects (125). In 4,980 births in Ohio herds surveyed in 1957 to 1960, 36 calves in every 1,000 had a congenital defect (83). In New Zealand, the incidence of defects reported by farmers in 1957 and 1960 ranged from 3.5/1,000 in 1957 to 2.4/1,000 births (6,7). In English dairy herds, perinatal mortality was 4.9% with 6.8% of the calves being defective (202). During bilateral dissection of 108 calves, and unilateral dissection of 31 calves, 103 different defects were identified (209).

A study of 1,000 German calves included 532 stillbirths; 84% had signs of neonatal asphyxia only, 6.9% were congenitally malformed, and the remaining 9.1% had evidence of infection (81). Of the malformed 468 calves that died within 3 months, 2.8% had congenital malformations that caused death within 3 days and 5.1% had nonlethal malformations and died from some other cause. In range herds of Angus and Herefords involving 6,406 calvings over a 10-year period, stillbirths amounted to 4.4%; 28% of the stillbirths were abnormal, or 1% of all calves (15).

Collection of congenitally defective calves in Hesse, Germany, for nine years totaled 2,293 from German Black Pied, German Red Pied, Simmental, Yellow cattle, Red cattle and crossbreeds. Frequency of congenital defects was estimated to be 0.25% (191-193). Relative frequency of the various body systems affected was: central nervous system (21.6%), musculature (13.7%), anomalous twins (10.0%), congenital systemic disturbances such as hydrops (9.5%), defects of large body cavities such as schistosomum reflexum (9.3%), facial skeleton (8.8%), bones of leg including joints (6.9%), digestive (4.3%), urogenital (4.3%), bone and cartilage (2.8%), heart and vessels (2.7%), skin (2.0%), and others (1.7%).

A total of 137,717 patients in veterinary college clinics in the United States and Canada contained 6,455 animals with congenital defects (188). Thus, the incidence of congenital malformation among all calves seems to range from 1/500 to 3/100 births, with 40-50% born dead and only a small fraction of the defects not being externally visible. For comparison, in man the frequency of congenital malformations is estimated to be 1-3%.

Causes

Although economic losses due to defects are less than losses due to diseases caused by nutritional or infectious agents, congenital defects may cause considerable economic problems. Besides economic importance, congenital defects have biomedical importance as models for studying various disease processes in man. Congenital defects are either genetically or environmentally induced or follow a complex pattern of environmental-genetic interaction.

Although causes of many congenital diseases in livestock are unknown, many follow simple Mendelian inheritance–mostly simple autosomal recessive inheritance. Other monofactorial inheritance patterns are described as overdominant, dominant, or incompletely dominant. Only a few reports describe sex-linkage. Congenital defects may also be inherited as a polygenic manner either with or without a threshold (16,46). There is an urgent need to find in cattle breeds the families which have least or none of the undesirable traits due to multiple gene inheritance. Examples are spastic paresis and abnormalities of the reproductive system.

Diagnosis of genetically-caused congenital defects is based on the rule that genetic diseases run in families. Thus, congenital defects occur in typical intergenerational and intragenerational patterns. That requires enumerating normal and abnormal offspring and identifying their familial relationships. Various statistical methods are used to analyze such data. Breeding trials are necessary to confirm inheritance patterns. Chromosomal defects and aberrations in chromosomal number are becoming increasingly important for studying the etiology of congenital defects in cattle (18,159, 160,163,194).

Genetically-caused defects require extensive and expensive adjustment of breeding programs whereas environmentally-caused defects may be eliminated by adjusting management practices. Environmental agents such as diet, toxic plants, other toxic substances, infections *in utero* and variations in ambient temperature, or a combination of environmental and genetic factors, cause congenital defects in cattle (16,76,125).

Crippled-calf disease may occur when pregnant dams ingest lupine *(L. sericeus, L. caudatus* and *L. nootkatensis)* during days 40 to 70 of gestation (105,150). Other plants incriminated or suspected of causing deformities in calves are *senecio* and related plants, *Indigofera spicata, cycadales, blighia,* loco plants, *papaveracea, colchicum, vinca,* tobacco and related plants, as reviewed recently (105,161).

Prenatal viral infections have been identified as teratogens in livestock (36). Cerebellar hypoplasia is caused by prenatal infection with bovine virus diarrhea virus (BVD) (22,23,101-104,201), hydranencephaly may be linked to prenatal infection by bluetongue virus (164,180,181), and prenatal akabana virus infection in Japan and Australia was shown to cause hydranencephaly and arthrogryposis (80,92,108,168,178,184).

Teratogenic agents involved in congenital defects are difficult to identify. In general, environmentally-caused defects follow seasonal patterns and known stressful conditions, may be

linked to maternal disease, and do not follow a familial pattern. Disease patterns are variable, calves from heifers are more frequently affected, fetal Ig is frequently detectable, abortion incidence increased, and morbidity in the herd or other associated animals may be observed (16).

Diagnosis and Control

The basic consideration for diagnosis and control is prevention. There are undesirable inherited traits that do not impair usefulness and such traits should be listed on the pedigree or in any advertisement. The remainder involve impairment of structure and/or function.

For diagnosing and controlling congenital defects, the following procedures should be followed. Continual effort should be made to have all affected calves reported. This requires cooperation of veterinarians, AI centers, animal breeders and cattle breed associations. Histories should be taken as completely as possible. Histories should include: breed, age of parents, parentage of affected and unaffected control calves, geographic region, season, type of pasture, soil type, exposure to or suspected exposure to teratogenic plants, feeding and management practices, breeding records, maternal medical and vaccination records, disease status of herd, periods of stress, drugs administered, congenital defects observed previously, and history of any similar congenital defects in neighboring herds.

All defective calves should be examined (clinical, biochemical, and necropsy). Defective calves should be subjected to a standardized necropsy. This should be done by the practitioner or the regional diagnostic laboratory and the defects classified by the body system primarily involved.

Serum samples should be taken whenever possible and checked for bovine virus diarrhea, bluetongue, and other viral antibodies. Sections of spleen, liver, and brain should be taken and examined for possible virus isolation if a viral teratogen is suspected. Serological and fluorescent antibody techniques may be more reliable when a viral teratogen is suspected.

Sections of brain, cerebellum, lungs, liver, kidneys and other appropriate tissues should be fixed in 10% buffered neutral formalin for histologic examination.

Chromosomal and genetic analysis should be done. Breeding records may be analyzed for evidence of inbreeding and for characteristic intergenerational and transmission and intragenerational frequencies of genetic defects. Etiologic diagnosis is made after the results of all the above criteria have been carefully considered.

Epidemiology of congenital defects has to be considered in three aspects: unknown, suspected and known. Many defects are of unknown etiology. We use the standardized approach above to clarify etiology. We recommend removal or testing of any AI bull suspected of siring three similar defective calves. 1) With all defects we check our files and the literature for any information of similar cases. 2) With suspected cases of hereditary origin we look for the following evidence of hereditary involvement: a) is the defect morphologically bilateral and symmetrical? b) can the defect be explained embryologically? c) is the calf inbred and has the defect occurred with the expected frequency? 3) Occurrence of defects with an established simple hereditary basis. One well-documented case is justifiable reason for announcement or removal of the sire from AI.

Testing Procedures for Suspected Sires

Major hereditary patterns commonly found in defective calves are: dominant, incomplete dominant, overdominant, recessive and polygenic. It should be emphasized that there are no minor defects in AI and that all defective calves should be recorded.

There is usually no need to test for dominance because it is easily recognized. Polygenic traits should be carefully recorded in the AI industry and we need to find families free of these traits, particularly those affecting reproduction.

It is desirable to test a bull for the presence of recessive genes prior to use as an AI sire. If the bull is of standard phenotype, he may be bred to unknown populations. Occurrence of defects would justify removal from AI service or at least labeling the bull. The suspected bull may be bred to known heterozygotes of a trait, to his own daughters or abnormal homozygous individuals if survival is possible. Table 1 summarizes these testing procedures. They are time-consuming and expensive; they are, however, much cheaper than trying to control a defect after it has spread insiduously through a cattle breed. Purchasers of semen should be informed of the presence of undesirable recessive genes that the sire carries.

Veterinarians can help their clients make decisions regarding their breeding programs. The practitioner should know genetic principles, methods of control of undesirable traits and should be able to diagnose those defects commonly of concern in the various breeds of cattle. In other words, cattle practitioners should have a good working knowledge of cattle teratology.

Table 1
Sire-Testing Procedures

Procedure	Offspring needed to reach probability level of: 0.05	0.01	0.001
Homozygous abnormals*	5	7	10
Heterozygotes*	10	16	24
Father-daughters**	22	35	52

*Checks for one trait only.
**Checks for all undesirable recessive traits.

Congenital Defects in Cattle

This section reviews congenital defects in cattle currently of concern in the United States. They are classified according to the major body system affected.

Skin. Developmental defects of the skin and adnexa may be generalized or localized. One of the more common skin defects is epitheliogenesis imperfecta, which affects calves of either sex. Epithelial defects are seen distal to carpal and tarsal joints, and calves have one or more defective claws. In addition, epithelial defects usually involve muzzle, nostril, tongue, hard palate, and cheeks. Calves affected with epitheliogenesis imperfecta are either born prematurely, or die shortly after birth due to septicemia. It is due to homozygosity of a simple autosomal recessive gene (133).

Fragility of skin similar to the Ehlers-Danlos syndrome in man has been identified in cattle. Two Hereford calves had hyperelasticity of skin and articular ligaments, cutaneous fragility, and delayed healing of skin wounds. The calves were inbred as a result of a father-daughter and a half-sib mating (169).

Lethal keratogenesis imperfecta appears a few months after birth, and is characterized by erosions in the region of the claws, tongue, oral cavity, and esophagus. It is a recessively inherited skin disease with lesions of exudative dermatitis of the legs, and erosions involving oral cavity, esophagus, and forestomachs has been described in Danish Black Pied calves (4). Some calves developed diarrhea; others, conjunctivitis, rhinitis, and bronchopneumonia, or central nervous system signs. The condition is a hereditary zinc deficiency syndrome, associated with immunodeficiency and skin disease and other signs (4).

A similar condition inherited as an autosomal recessive in Canadian Holstein-Friesian calves is referred to as "baldy calves." Calves are born normal but some develop scaly, thickened and folded skin over neck and shoulders. Affected calves are usually destroyed (125).

Congenital ichthyosis has been described in Pinzgauer calves (163) and Holstein-Friesians (100).

Defects of Hair. Congenital hypertrichosis has been described in European cattle. It is correlated with polypnea during hot weather (125). Abnormal curliness of hair transmitted as an autosomal dominant has been reported in Ayrshire calves (40).

Six different kinds of hypotrichosis are distinguished in cattle (92):

1. Hairless lethal, encountered in exotic breeds. Affected calves die shortly after birth due to this simple autosomal recessive.

2. Semi-hairlessness has been reported only in Polled Herefords and is characterized by thin coat at birth. Later, the hair coat is sparse, patchy, and the skin is wrinkled and scaly. It is inherited as a recessive trait.

3. Hypotrichosis associated with anodontia has been described in Maine-Anjou calves as a recessive trait.

4. Viable hypotrichosis, encountered in Guernseys and exotic breeds, characterized by partial to complete absence of hair at birth, is due to homozygosity of a simple autosomal recessive gene.

5. Hypotrichosis with missing incisor teeth has been reported in Holstein-Friesian calves. The trait is possibly dominant.

6. Streaked hairlessness in Holstein-Friesians is characterized by vertical hairless streaks over hip joints and sometimes over the body and legs and is a dominant sex-linked gene (92).

Albinism. Albinism may be classified as partial, incomplete, and complete. In partial albinism, the color of the iris is blue and white centrally and brown peripherally, and the coat color is usually characteristic of the breed or a dilute color (47,57,-100,113,114,115,176). A form of partial albinism is recessively inherited in Chediak-Higashi syndrome, which includes abnormally large, membrane-bound organelles in various cell types and increased susceptibility to infection (195).

Incomplete albinos, inherited as an autosomal dominant, usually have pure white hair and a few cattle may have small pigmented body areas. Iris color varies from blue to gray to white and may contain brown sectors. Incomplete albinos have colobomas of the nontapetal fundus and tapetal fibrosum hypoplasia. Complete albinism is a simple autosomal recessive trait and is characterized by pure white coats, white to pink irises, but a normal tapetum lucidum.

Cardiovascular System. The cause of cardiovascular defects has received little study and usually is in form of case reports (166). Ectopia cordis is a common cardiac malformation, but its cause is unknown. The heart may be located in the cervical region, outside the thoracic cavity through a sternal fissure, or in the abdominal cavity. Ventricular septal defects, considered common in cattle, may be single, isolated defects or may be combined with abnormalities of the large vessels which are referred to by the nomenclature of their human counterparts (45,125). Sixteen ventricular septal defects were reported in common Scottish breeds (45).

Lymphatic System. Dysplasia of the lymphatics is an autosomal recessive defect of the lymphatic system resulting in edema. It has been reported in Ayrshire calves (82).

Digestive System. Smooth tongue in Dutch Friesians is characterized by diminution of filiform papillae, fragility of the mucosa, velvety hair coat, soft horns, and microcytic hypochromic anemia (53). Smooth tongue in American Brown Swiss was considered to be due to an incompletely penetrant dominant gene (125). Defects of the intestine are usually localized and disrupt patency, such as atresia of the ileum, atresia of the colon and others (125,166,179,216). The cause of all these congenital bovine defects of the intestinal tract is not known but may be polygenic in nature. Recently, the defects and genetic predisposition to disease of the gastrointestinal system were discussed. Included in the review were discussions on atresia coli, ilei and ani (216).

Large Body Cavities. Hernias such as scrotal, inguinal, and in particular, umbilical, are common in cattle (194). Four of seven Holstein-Friesian bulls had offspring affected with umbilical hernias (49). The closer familial relationship of the four bulls having affected offsprings indicated recessive inheritance (40). Holstein-Friesian calves of U.S. origin are more frequently affected with umbilical hernias (188). Outbreaks of umbilical hernia in cattle have been attributed to an incomplete dominant gene, due to homozygosity of a simple autosomal recessive gene and to polygenic inheritance (5). Sporadic cases and environmentally-induced cases do occur.

Schizosomia, an extreme closure defect of the abdominal cavity, is a common lethal defect of cattle. Studies on its cause are scarce. It is considered to be a polygenically-caused defect being part of a complex of unbilical hernia, abdominal fissure and finally, schizosomia (191-193).

Male Reproductive System. Various deviations of the penis and prepuce have been described; all are rare with unknown causes. Penile agenesis is rare. Duplication of the penis was described (20). Persistent penile frenulum, considered common in Shorthorn and Angus, is thought to be inherited (25).

Since the study of Lagerloef and Settergren (109) in Swedish Highland cattle, numerous other investigators have reported testicular hypoplasia, which is bilateral or unilateral, and partial or complete.

Cryptorchidism, or incomplete descent of the testicle may be unilateral or bilateral. The available evidence indicated possible genetic transmission (213).

Segmental aplasia of the Wolffian duct, characterized by aplasia of segments mostly located in the head of the epidymidis, is usually unilateral (34).

Multiple abnormalities of the male genital tract, including segmental aplasia of the Wolffian duct, gonadal hypoplasia, and intermittent cryptorchidism have been found to result from chromosomal aberration in number such as XXY (200).

Intersex. An intersex is an individual with congenital anatomical variations that may have some reproductive organs of both sexes, or be genetically one sex and phenotypically the other (17,70). Hermaphrodites have both sexes by definition, gonads of both sexes, either as an ovary and testis, or combined into an ovotestis. A pseudohermaphrodite has the gonads of one sex and reproductive organs with some characteristics of the opposite sex (17).

The tubular reproductive organs of a hermaphrodite vary. The karyotype of a cow with testes, uterus, and male ductal structures, revealed in the peripheral blood predominantly XY and XX cells (38,163). Karyotype of a hermaphrodite, externally a male with empty scrotum, a small vagina, one seminal vesicle, uterus, right oviduct and ovary, left spermatic cord, and ovotestis was predominantly XX. Lung, muscle, and uterus had XX; blood, bone marrow, kidney, testis, and ovotestis had mainly XX but also XY cells (38).

Freemartins are heifer calves born cotwin to a male. About 93% are affected with variable hypoplasia or agenesis of organs developing from the Mullerian ducts and stimulated development of the Wolffian duct system and are usually sterile. Bovine freemartinism may not be caused by humoral factors but may be a function of sex chromosome mosaicism (42). Blood cell chimerism, defined as an individual with cell populations of more than one genotype arising through a mixture of different zygotic genotypes, was not considered to be the cause for the sterilizing effect in the female cotwin (15,211). Recent investigations of freemartin embryos and fetuses revealed two successive phases in the development of a freemartin. The initial phase of inhibition from day 50 to 75 of gestation is characterized by arrest of gonadal development in the female and regression of Mullerian duct development. The fetuses were considered to be under the influence of an inhibiting factor. The phase of masculinization follows at day 75 (217).

Female Reproductive System. Ovarian aplasia has been reported with and without associated anomalies of the tubular reproductive structures. Ovarian hypoplasia in the Swedish Highland breed may be total or partial, and unilateral or bilateral (109). Zemjanis and coworker (221) diagnosed an incidence of 1.9% ovarian hypoplasia in 20,913 clinical examinations in 14 Minnesota and Wisconsin herds.

Defects of oviducts, uterus, cervix, and vagina have been described in several breeds. In many, fusion of the Mullerian ducts is either lacking or exaggerated (203). A high incidence of duplication of the cervix in Hereford cattle was reported to be due to a sex-limited, simple autosomal recessive gene with low penetrance and varying expressivity (204). Segmental aplasia of the Mullerian duct system has been described by many workers (187,221). White-heifer disease falls into two distinct classes of morphological aberrations, both with partial or total persistence of the hymen and one with additional defects cranial to the hymen. Common to both types are functional ovaries and accumulation of secretion products (50). It is considered to be inherited in a polygenic pattern (75,219).

Rectovaginal constriction has been described in Jersey cattle and appears to be inherited as an autosomal recessive or may be followed by a more complex pattern (143). No gross chromosomal abnormalities have been found. The defect is characterized by inelastic constrictions at the junction of the anus and rectum and the vestibule and vulva.

Prolonged gestation has been recorded for most dairy breeds as hereditary and two distinct pathological conditions have been distinguished (106). In the first type, due to a single autosomal recessive gene, the calf continues to grow *in utero* and is carried up to 100 days past normal term (66).

Calves born or taken by cesarean section are weak and die in hypoglycemic crisis (87). In the second type, also caused by homozygosity of a recessive gene, the fetus ceases to grow beyond 7 months and may be affected with cranio-facial defects (206). In severely defective, as well as nearly normal calves, the adenohypophysis did not develop (106).

Muscular System. Congenital defects of muscle are common in cattle and are economically important (54,56,59,125,132,177).

Congenital flexure of the pasterns occurs in Jersey and other breeds due to homozygosity of a simple autosomal recessive gene. Calves knuckle over in the front pasterns; only occasionally are the hind legs also affected. The condition is reversible and affected calves usually recover in 1 to 8 weeks (165).

Arthrogryposis, defined as permanent abnormal joint fixation present at birth, is one of the most frequent congenital defects observed in calves. The arthrogrypotic syndrome includes more than one etiologic or pathologic entity, is worldwide in distribution and has been described in all major breeds of cattle (54-56, 69,71-74,79,84,85, 105,119-121,131,136, 138,144,150,156,183). Spina bifida caused bilateral arthrogryposis of the hind legs (84). Hydranencephaly and spinal cord lesions were described in calves affected with arthrogryposis (215).

Arthrogryposis and associated defects in Charolais calves consisted of tetramelic arthrogryposis and cleft palate. The contractures of the legs were symmetrical. Metacarpophalangeal and metatarsophalangeal joint surfaces were incongruent and the distal trochlea of the femur and the patella were hypoplastic. Some calves also had kyphoscoliotic deformities of the vertebral column. The muscular system was affected by wasting and replacement of muscle fibers by fat cells. Arthrogryposis in Charolais calves is inherited as a simple autosomal recessive trait with incomplete penetrance (51). Prenatal viral infection with akabane virus can cause arthrogryposis (80).

Muscular hypertrophy (Doppellender, double muscling, muscular hyperplasia) is encountered in all major beef breeds of the United States and most European breeds (21,125). Muscular hypertrophy varies widely. Characteristic is the rounded outline of the hindquarters frequently referred to as "ham-like." The tail is attached more anteriorly than normal. The muscles of the shoulder, back, rump, and hindquarter are separated by deep creases, with those between the semitendinosus and biceps femoris and between the longissimus dorsi muscles of either side particularly noticeable. Necks of the double-muscled animals are shorter and thicker; their heads smaller and lighter. Many double-muscled animals stand in a stretched stanced. The diaphysis of the long bones tend to be shorter. Variable degrees of macroglossia may be present. Additional signs are infantile genital tracts, impaired reproduction, slower sexual maturity, lengthened gestation, and increased birth weight combined with dystocia. The double-muscled calves are less viable and are particularly susceptible to rickets and joint problems (21,177).

Oliver and Cartwright (177) classified cattle populations into three groups: the majority with normal phenotypes, those with most or all characteristics of muscular hypertrophy, and those with some of the typical characteristics of double-muscling. Furthermore, they pointed out that double-muscling is mediated by a pair of incompletely recessive genes. Ashmore and Robinson (8) demonstrated a significant decrease in succinic dehydrogenase activity in muscular hypertrophy. Fewer reacting fibers were observed and the activity was less in fibers from double-muscled calves.

Joint Defects. Congenital disorders of joints may be generalized or restricted to a single joint. Ankylosis, abnormal union of the ends of bones forming the joint, may involve one or more joints. Bilateral spontaneous osteoarthritis of the stifle joint in Holstein-Friesian and Jersey cattle has been described as an autosomal recessive trait (204). The occurrence of hip dysplasia in Hereford cattle seems to be hereditary (24). Although joint diseases are of considerable importance, the shortness of the section here indicates the scarcity of work on such diseases. Hereditary components and other etiologic factors remain largely unstudied.

Skeletal System. The entire skeletal system may be affected such as in dwarfism and osteopetrosis, or single regional defects may be encountered (54,63,-118,142,173,175). Dwarfism is a universal problem in all cattle breeds and was an economic problem to the American beef cattle industry. Dwarfism is a defect of interstitial growth of epiphyseal, articular, and basocranial cartilages resulting in variable shortness of legs, cranial base, and vertebral column. Various types of dwarfism are distinguished such as short-headed, long-headed, and Telemark, all considered to result from recessive genes. In addition, the Dexter, comprest, and compact mutants (generally considered to be dominants) are part of a complex of conditions from more than one locus and seem to be related to the recessive types (65,67).

Osteopetrosis has been encountered in black and red Angus, Hereford, and Simmental calves (60,63,-122,126, 145,147,172). Osteopetrosis is characterized by small size and birth weight, brachygnathia inferior with impaction of molar teeth, misshapen coronoid and condyloid processes, open fontanelle, thickened cranial bones, agenesis or hypoplasia of major foramina of the skull, and complete lack of bone marrow cavities. Radiographically, the bone density is increased. The basic microscopic feature is lack of remodeling of the primary spongiosa which persists throughout the metaphyseal and diaphyseal areas. Calves affected with osteopetrosis are born prematurely at 251 to 272 days (mean 262) gestation. The condition is frequently mistaken for an abortion problem.

Acroteriasis congenita is a simple recessive syndrome consisting of low birth weight, amputation of

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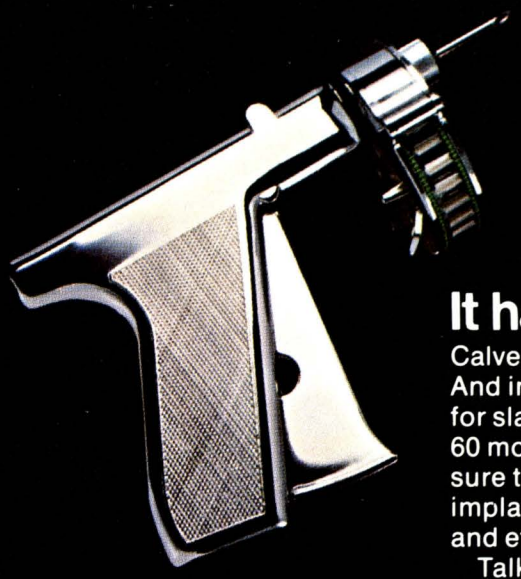

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all four legs, facial skeletal defects, cleft palate, brachygnathia inferior, microtia, and hydrocephalus (190).

Osteogenesis imperfecta was recently reported in Charolais calves, the main features being reduction in bone mass leading to spontaneous fractures. It is inherited as a simple autosomal recessive trait (95).

Arochnomelia was described in German Simmental calves. The syndrome is characterized by dolichostenomelia with extreme fragility of long bones, deviation of vertebral axis, tetramelic arthrogryposis, brachygnathia inferior, and cardiac defects. The disease is due to homozygosity of a simple autosomal recessive gene (195).

A number of regional skeletal defects are of concern in cattle. Cleft palate occurs frequently with other defects, particularly arthrogryposis. Cheilognathoschisis in Shorthorn calves has been reported as a simple autosomal recessive trait (218).

The jaws may be affected with campylognathia or lateral deviation of the face with normal development of the mandible. Abnormal length of the upper or lower jaw is referred to as superior or inferior prognathia, respectively. Short upper and lower jaw are termed superior or inferior brachygnathia. Inferior brachygnathia (short lower jaw, parrot beak, parrot mouth) may be a single, isolated congenital defect in cattle. It may vary considerably in degree. It may be accompanied by cerebellar hypoplasia in Angus calves, a lethal believed to be due to homozygosity of a simple autosomal recessive gene (39). Inferior brachygnathia in calves combined with other defects may result from an autosomal trisomy chromosomal defect (86). Austrian Simmental calves affected with short lower jaw were described and considered to be due to recessive genes (213). Craniofacial dysplasia *(tete a mouton)* described in the Limousin breed of France, involves deficient ossification of the frontal sutures, convex profile of the nose, inferior brachygnathia, bilateral exopthalmus, scoliosis of the upper jaw, macroglossia, and defects of omasum and heart (125).

Short-spine lethal, a recessive trait, is characterized by reduction and fusion of spines and ribs from the normal 13 to 6 or 7 (129). Fusion of the occipital area with the atlas is seen in horses and calves (130,140).

Perosumus elumbis or agenesis of the caudal segments of the vertebral column is rare. Other defects of the spinal column include kyphosis (dorsal deviation), lordosis (ventral deviation), scoliosis (lateral deviation), and their combinations. Kyphoscoliotic deformities are commonly associated with arthrogryposis (56,136). Spina bifida is usually associated with abnormal development of the spinal cord (29). Lateral deviation of the neck is referred to as torticollis or wryneck. Total agenesis (anury) and partial agenesis (brachyury) of the caudal part of the spinal column are found commonly in cattle and are often associated with defects in such other organs as eye and heart. Taillessness is most likely not an inherited condition since breeding tailless cows to a tailless bull resulted in normal calves (58).

Tibial hemimelia, a recessive lethal, is common in Galloway cattle (149,152,170). Adactyly, a recessive lethal, was described in Shorthorn calves (123). Development of additional digits, polydactyly, has been described occasionally (128,170).

One of the commonest skeletal defects in Holstein-Friesian cattle in the United States is syndactyly. It is defined as fusion or non-division of functional digits (116,127,134, 135,141,148,151). Other breeds reported being affected with syndactyly are Angus (117,151), Chianina crossbred cattle (171), and Simmental in Austria (107).

Syndactyly in Holstein-Friesian cattle is a recessive trait with incomplete penetrance and varying degrees of expressivity (134). Syndactyly follows a particular expressivity pattern; the right front foot is most frequently affected, followed, in order of frequency, by the left front, right rear, and left rear foot. The degree of fusion or non-division is always more advanced in the right front foot when more than one foot is affected. Osteologic defects in syndactylous feet have followed certain sequences; the second phalanges are frequently horizontally fused, followed by the third and then the first phalanges. Osteologic anomalies paralleled the asymmetrical external pattern. Muscles, blood vessels, and nerves accommodated the syndactylous deformities (1-3). Hereditary bovine syndactyly is accompanied by a functional defect expressed as hyperthermia that is triggered by higher outside temperature. When syndactylous cattle and control cattle were subjected to controlled conditions in a climatic chamber, all five syndactylous Holstein-Friesian cattle developed signs of malignant hyperthermia. This complemented experience with syndactylous cattle (kept at Kansas State University) which had succumbed to environmental stresses such as higher ambient temperatures and calving. Gruneberg and Huston (68) studied the development of syndactylous-carrier and normal Holstein-Friesian embryos at 31 to 45 days of gestation and found that the anlagen of the metacarpal bones were close together in the 37-day syndactylous embryo and the distal ends had started to fuse. The normal 30-day embryo had widely-separated blastemata of the future phalanges, whereas the syndactylous embryo had a single mass of blastema. The use of preterminal cesarean section and recovery of fetuses can accelerate breeding trials considerably (167).

Central Nervous System. Congenital defects of the central nervous system are common, and structural change involving both skeletal structures and central nervous system, or only the latter. Functional defects also occur. CNS defects are of economic significance and most are of comparative interest (26,33,37,41,78).

Anencephaly involves nonclosure of the cranial portion of the neural tube and failure of the cranium to develop. Its cause in cattle is not known. Only a few cases have been described in cattle (30).

Arhinencephaly, defined as the absence of rhinencephalon, is a rare deformity in cattle and is characterized by unilateral or bilateral absence of the olfactory bulbs, tract and nerves (173).

Agenesis of the corpus callosum is defined as absence of all or part of the corpus callosum. This defect has rarely been described in cattle and its cause is unknown (31).

Hydranencephaly is defined as complete or almost complete absence of the cerebral hemispheres in a cranium of normal conformation, with the space filled with cerebrospinal fluid surrounded by a thin membranous cerebral tissue. A congenital syndrome consisting of hydranencephaly or arthrogryposis, or both, occurs sporadically or as epizootics in calves. Associated pathologic changes included cerebellar hypoplasia, muscular atrophy, cleft palate, scoliosis, spina bifida, abortion, stillbirths, and premature births (71-74,80). Seasonal occurrence in unrelated herds of different breeds, epidemiological, serological, and pathological findings indicated an environmental factor (221). Several causes of the defect in cattle have been identified, including ephemeral fever virus, hyperthermia, Japanese encephalitis virus, bluetongue virus, and akabane virus (71,80,164).

Hydrocephalus is common in cattle (26,33,54,61). It is defined as accumulation of excessive fluid within the ventricular system. Calves affected with internal hydrocephalus are born dead or die within a few days of birth. Congenital internal hydrocephalus appears to be inherited in many breeds as a simple autosomal recessive trait (11,62,64). A breeding trial with Hereford cattle heterozygous for internal hydrocephalus demonstrated that the condition is due to homozygosity of a simple autosomal recessive gene. The homozygous-affected calves were dead at birth and had internal hydrocephalus, myopathy and bilateral microphthalmial. Congenital hydrocephalus in Hereford and Shorthorn calves was accompanied by stenotic aqueduct, cerebellar hypoplasia, myopathy, multiple ocular anomalies, retinal detachment and dysplasia, cataract, microphthalmia, and persistent pupillary membranes. The anatomic expression of the hydrocephalus varies considerably (61,64).

Meningoencephalocele is a protrusion of meninges and brain tissue through a cranial cleft. The herniated portion sometimes forms a large liquid-filled sac. Meningoencephalocele usually occurs in the frontal region (porencephaly), but some are midfrontal, parietal, or occipital. Whether this defect is inherited in cattle is unknown. However, meningoencephalocele in Galloway cattle associated with tibial hemimelia is due to homozygosity of a simple recessive autosomal gene.

The Arnold-Chiari malformation consists of a herniation of tongue-like processes of cerebellar tissue through the foramen magnum into the anterior cervical spinal canal, plus caudal displacement and elongation of the medulla oblongata, pons, and the 4th ventricle (27). The malformation is often accompanied by spina bifida, hydrocephalus and meningomyelocele. The Arnold-Chiari defect is rare in cattle and its cause is not known (27).

The clinical signs of cerebellar hypoplasia are recumbency with extended limbs, intermittent opisthotomus and ataxia. Cerebellar hypoplasia was first described as a genetic defect in calves with an autosomal recessive mode of inheritance (26,39,41, 44,182). Cerebellar agenesis, hypoplasia and atrophy are being observed with increasing frequency in domestic animals. Agents responsible for these defects include mutant genes, viruses, and toxic agents (76).

Bovine viral diarrhea virus causes cerebellar hypoplasia and degeneration in calves when susceptible pregnant heifers are inoculated at 79, 107, 116, 146, and 150 days of gestation with BVD-MD virus. Cerebellar hypoplasia and degeneration were also observed in calves carried to term (22,23,101-104,201). Pathologic changes in the genetic form of cerebellar aplasia or hypoplasia differed from those of BVD viral-induced cerebellar defect. The ocular lesions and large irregular cavities in the folial white matter, and inflammatory process observed in the BVD-induced cases were not described in those of genetic form, while cerebellar aplasia was not seen with the viral cerebellar hypoplasia and degeneration.

Cerebellar ataxia (hereditary hypomyelinogenesis congenita) has been reported in Jersey (206), Shorthorn (88), Angus-Shorthorn (220) and other breeds (125). The clinical signs were incoordination and failure of synergic muscle groups to act harmoniously. Tremor may be present persistently or occasionally and some calves appear normal until 2 to 3 weeks of age. The condition is considered to be an autosomal recessive trait (88,200).

Progressive ataxia was described in Charolais cattle (168,186). Clinical signs, first noticed in cows 8 to 24 months of age, progressed in 1 to 2 years from slight ataxia involving all four limbs to recumbency.

Congenital cerebellar atrophy with familial convulsions and ataxia was described in Holstein and Angus calves (12,214). Clinical signs usually appeared during the first few hours of life and were characterized by single or multiple, sudden tetaniform seizures of variable intensity that lasted 3 to 12 hours or longer. The brain was grossly normal but histopathologically selective degeneration of Purkinje cells with variable intensity was evident. The disease appeared to be inherited in Angus with a dominant mode of inheritance with incomplete penetrance (12). We observed the condition in a Charolais calf with clinical and pathological changes almost identical to those of familial convulsions and ataxia (28).

Hereditary neuraxial edema was first reported in neonatal Polled Hereford calves (35). Affected calves were unable to rise or lift their heads and lay quietly without struggling, were incoordinated and had coarse muscular tonic contractions. A sudden touch

or loud noise elicited vigorous extension of the legs and neck (32,35).

Spastic and Paralytic Diseases. Included are those with clinical evidence implicating CNS involvement. Pathogenesis and neuropathologic lesions have not been described, or not well defined. Some of the diseases are hereditary. Spastic paresis is characterized by spastic contracture of the muscles and extensions of the stifle and tarsal joints of one or both hindlimb(s), and thus has been referred to as "contraction of the Achilles tendon," "straight hock," and "elsoheel" (10,13,111,197). Spasticity characteristically affects the gastrocnemius and superficial flexor muscles and tendons, and in some cases, the biceps femoris, semitendinosus, semimembranosus, quadriceps, and abductor muscles. Bilateral involvement is not common.

Affected breeds are Friesian, Ayrshire, Angus, Beef Shorthorn, Shorthorn-Ayrshire cross, Charolais, Meuse-Rhine-Yssel, Groningen, Brown Swiss, Red Danish, and Kankrej Indian cattle, as reviewed recently (26,125).

The radiographic findings on affected hock joints are fairly consistent and are characterized by the increased angle of the joint, osteoporosis and exostosis around the distal epiphyseal line of the tibia, curvature and exostosis of the dorsal side of the calcaneus, and widening of the epiphyseal line of the calcaneous (125). Central nervous system lesions have not been reported (41). Although spastic paresis was thought to be inherited as a simple recessive, recent breeding experiments indicated that spastic paresis is not inherited as a simple recessive (205,217). Polygenic influence(s) as well as environmental factors may interact to express spastic paresis.

True epilepsy (idiopathic epilepsy) is a convulsive state without discoverable etiologic factors or definite underlying lesions. Epileptic convulsions were recorded in Brown Swiss cattle as an autosomal dominant (9).

Mannosidosis in Angus cattle of New Zealand and Australia is associated with a deficiency of mannosidase and occurs in Angus and Murray Grey cattle as a simple autosomal recessive (96-99). The deficiency of mannosidase results in storage of an oligsaccharide containing glucosamine and mannose (99). Mannosidosis is characterized by ataxia, incoordination, head tremor, aggression, and failure to thrive. Most affected cattle die within the first 12 months of life. Neonatal mortality also occurs. The primary pathologic lesion is vacuolation of neurons with vacuoles formed from saccular dilations of the Golgi apparatus. Vacuoles also occur in macrophages and reticuloendothelial cells of the lymph nodes (96-99). Since affected calves have an absolute deficiency of α-mannosidase and heterozygotes have a partial deficiency of this enzyme, mannosidosis can be controlled by identifying and eliminating heterozygotes (208,218). Mannosidosis was recently diagnosed in two Angus herds of the United States (153).

Spinal cord defects of calves have been described under spina bifida and spinal dysraphism. Spina bifida implies a defect of the vertebrae with or without spinal cord anomalies. Spinal dysraphism is a malformation of the spinal cord. Spina bifida has been described in many cattle breeds (29). The morphologic changes in 18 calves (4 Holstein-Friesian, 3 Herefords, 3 Galloways, 3 Angus, 1 Simmental, 1 Charolais, 1 Brown Swiss, 1 Shorthorn, and 1 Angus-Hereford) were described. Twelve cases had both vertebral and spinal cord defects, five had only spinal cord anomalies, and one calf had a vertebral defect without spinal cord involvement. Arnold-Chiari malformation is frequently associated with spinal cord defects. The most common lesions of the spinal cord were hydromyelia and syringomyelia (29).

Congenital Defects of the Eye. Relatively few ocular defects have been described in cattle either as single or multiple defects restricted to the eye, or observed in conjunction with defects in other organs, or associated with pigment deficiencies (48). Ocular defects in cattle frequently include abnormalities in other body organs, especially the central nervous system. Anophthalmia may be associated with taillessness (112). Hereditary encephalomyopathy and internal hydrocephalus in Hereford calves were combined with retinal dysplasia (210). Multiple ocular defects in grade Shorthorn calves, such as retinal detachment, cataract, microphthalmia, persistent pupillary membrane, and retinal dysplasia, were associated with internal hydrocephalus (124). Though internal hydrocephalus in cattle has received considerable attention, little information is available about the associated ocular lesions. Congenital defects of the caudal segments of the vertebral column and high ventricular septal defects occurred together with anophthalmia and microphthalmia (112). The frequency of anophthalmia and microphthalmia was estimated in six U.S. breeds to range from 1 in 7,500 to 1 in 50,000 births (112). Prenatal infections with virus of bovine virus diarrhea causes cerebellar hypoplasia combined with ocular defects (retinal atrophy, acute and chronic neuritis, cataract, and microphthalmia with retinal dysplasia) (207).

Conclusion

There are many bovine congenital defects of unknown etiology, suspected etiology and known etiology. Congenital defects of known environmental etiology such as bovine virus diarrhea-induced cerebellar atrophy, or plant-induced deformity such as crooked-calf diseases caused by lupine, require changes in management procedures. Defects caused by genetic factors are of considerable concern to the AI stations, cattle breeders, and cattle breed associations. Initial diagnosis of congenital defects is frequently required of practicing veterinarians. Some of the genetic diseases of current concern to the cattle industry of the United States include congenital defects of the skin such as epitheliogenesis imperfec-

ta, hypotrichosis, fragile skin, and protoporphyria. Of the internal organs, the reproductive system is of particular concern. Defects of bone are numerous and may affect single structures such as adactyly, tibial hemimelia, and syndactyly. Generalized skeletal defects of importance include dwarfism and osteopetrosis. Muscular defects of significance are double-muscling and arthrogryposis. Furthermore, genetically-caused defects of the central nervous system need further attention, particularly internal hydrocephalus and mannosidosis.

Many defects encountered of suspected etiology need close attention. In this category, as well as those of unknown etiology, practicing veterinarians remain the main source of information to guide further study.

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