naive but at the same time have the same grazing pattern and this is often difficult to achieve.

The persistent activity of a drug can also be determined in field trials. In this case, the appearance of worm eggs or larvae in the faeces of treated animals grazing continuously on contaminated pastures can be evaluated. Faecal samples are collected regularly and, after faecal culture, the genera of nematodes present are identified.

For interpretation of the duration of persistence, the prepatent period must be taken into account. If parasite naive animals are used, the difference in time of appearance of nematode eggs or larvae in the faeces can be taken as an approximation of the duration of persistent activity. In the case of cattle that are already shedding nematode eggs or larvae at the time of treatment, it is even more difficult to interpret the data. This method has the same limitations indicated for the second and third types of controlled trial resulting in considerable uncertainty in the estimates of duration of persistence. The control animals provide little or no information on the timing or amount of challenge during the trial. Any factors that influence egg or larvae output would not be separated from treatment effects in any statistical analysis. Even if animals were not exposed to effective challenge, this would still appear to suggest persistent activity of a drug. Because of the many uncontrolled assumptions, the duration of persistent activity determined by this method cannot be compared with data gained from so-called controlled trials based on worm counts.

Each of the four methods described can be justified; however, the results are not directly comparable. Therefore, whenever persistence of a product is claimed it is important that the method on which the calculation is passed is adequately described. This is particularly important when a persistence claim is under review by a regulatory authority and when periods of protection of different products are compared scientifically for promotional reasons.

Data generated in different studies using the same methods should be cautiously compared. If methodology differs, comparisons are misleading and cannot be made.

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Intersexuality in a Charolais heifer

J. Cole, M. Broadwell, G. Rogers

Veterinary Record (1997) 141, 656-657

A TWO-YEAR-OLD Charolais heifer, presented for a routine pregnancy examination, was found to have abnormal vulvar conformation (Figs 1, 2). The heifer was heavily muscled, weighed 519 kg and appeared masculine in conformation. The recto-urethral distance was 30 cm. A prominent raphe extended 15 cm beneath the rectum in the area normally occupied by the vulva. A 4 cm vulvar orifice surrounded by a tuft of long coarse hair was located just caudal to the mammary tissue. A 15 cm pouch resembling a prepuce extended cranially from the orifice. No palpable evidence of other structures was found in this area. During urination, urine projected caudally from the vulvar orifice. On rectal examination, a rudimentary uterus and seminal vesicles were palpable, and smooth, round gonads were noted. Results of karyotyping revealed chromosome spreads of 60 XY and 60 XX, indicative of a chimeraism. Serum was assayed for testosterone (28 ng/dl) and oestradiol (<50 pg/ml).

On postmortem examination of the reproductive tract, a 4 cm diameter testicle-like structure was found in the area normally occupied by the left ovary; the right gonad was not retrieved at necropsy. The epididymis and spermatic cord were intact and proportional to the testicle-like structure. The uterine horns were approximately 0.5 cm in diameter and joined the uterine body at the level of the trigone of the bladder. Seminal vesicles were located alongside the uterine body just caudal to the bladder, and prostatic tissue was caudal to the seminal vesicles. On further dissection, a rudimentary testis was found at the cranioventral most aspect of the reproductive tract. No cervical tissue was noted and the vaginal/vulvar tissue appeared morphologically normal.

The structures identified grossly were confirmed by histological analysis. The testis was underdeveloped and non-functional. Some lobules were composed of very few seminiferous tubules, and others consisted of many tubules lined with pseudostratified columnar epithelium. Sertoli-like cells predominated in the epithelium. These cells were dying and sloughing off, filling the lumens of the tubules with cytoplasmic fragments. Intestinal cells surrounded some of the seminiferous tubules. These cells were eutrophicated and virtually devoid of cytoplasm, indicative of a non-functional gland. The musculature of the epididymis was hyperplastic. The prostate was poorly developed. The collagen and smooth muscle of the prostate was not as prominent as were the other glandular tissues; however, secretory material was present in some prostatic ducts (Fig 3). The histology of the other structures was unremarkable.

Intersexuality has been reported in all domestic species. Intersexuality includes conditions of true hermaphroditism and pseudohermaphroditism, abnormalities of accessory genital organs, gonadal dysgenesis and freemartinism (McEntee 1990). In true hermaphrodites, the genetic sex is found to be either a chimera, an extra chromosomal combination (XXY, XXXY, XXXY), or a normal female (XX) (Roberts 1986). In freemartins, the development of female reproductive organs is suppressed by Müllerian inhibiting substance (MIS), a factor produced by the Sertoli cells of the testes, and the development of male reproductive organs is enhanced due to testosterone production by the Leydig cells of the testes. The degree to which the development of the female reproductive organs is suppressed depends on how early in fetal development the blood vessels of the twins’ fetal membranes fuse, allowing the MIS and testosterone to exert their effects. Recently, a sex coding region (sry) has been identified on the Y chromosome (Berta and others 1990). The H-Y antigen, which was previously believed to be the testis determin-

Corrections

Experimental reproduction of iodine deficiency in cattle M. A. McCoy, J. A. Symth, W. A. Ellis, J. R. Arthur, D. G. Kennedy (VR, November 22, p 544). It is regretted that, as a result of a production error, Dr Arthur’s address was omitted from this paper. His address is:

J. R. Arthur, BSc, PhD, Rowett Research Institute, Bucksburn, Aberdeen AB2 9SB

Study of the heart rate and energy expenditure of ponies during transport O. Doherty, M. E. Booth, N. Waran, C. Salthouse, D. Cuddeford (VR, December 6, p 589). The authors wish to clarify the formula used to calculate the energy expenditure in the ‘Analysis of results’ section on p 590. Energy expenditure was calculated as:

J/kgs = [(1/(V0/60) x 20-92) x 1000]/liveweight (kg)

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Corrections

J. R. Arthur

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