FEMALE STERILITY

by

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Fashions change in human and veterinary medicine just as in everything else. Today endocrinology is the most popular avenue of physiological research in the laboratory and hormonal therapy the fashionable form of treatment in the hands of the practitioner.

Whilst I believe there is a big future for the therapeutic application of endocrinological research to a wide variety of hitherto more or less refractory disease problems. I do feel that at the moment the pendulum has swung too far in this particular direction and that enthusiasm for the newer knowledge has tended to obscure our outlook regarding the more fundamental causes which may happen to upset the hormonal harmony of the animal body, intricately interlocked and balanced as it normally is.

The present time is therefore particularly opportune for an impartial and dispassionate review of the available evidence regarding the basic causes of female reproductive disorders and one is forced to conclude that these conditions are in the main the result of either:-

(a) Specific infection, or
(b) Nutritional imbalance, or
(c) Genetic imbalance.

As regards the first mentioned, an American writer recently summed up the situation in this pithy sentence, -"engrossed in the wonders of endocrine therapeutics, cervicitis and endometritis, the most frequent causes of sterility in cows, are being forgotten."

Sterility may be either temporary or permanent and may be defined as any condition of the female genitalia which,

(a) inhibits the production of viable ova;
(b) renders the spermatozoa incapable of reaching and fertilising viable ova;
(c) prevents the successful implantation of viable fertilised ova;
(d) renders impossible the normal subsequent development of viable, fertilised and successfully implanted ova.

When female sterility is temporary in nature, either the recuperative powers of the organs themselves, or appropriate remedial treatment suffices sooner or later to restore the normal function. On the other hand permanent sterility results from either inherent and genetic defects in the organism or through subsequent deep seated and permanent damage to what formerly were physiologically normal organs.

Permanent sterility I intend to discuss quite briefly. Inherent defects, of which the bovine "free martin" is perhaps the best known example, are often prenatal and endocrine in origin. Such instances are sporadic in occurrence and it seems highly unlikely that research will ever find a solution to such "mistakes of Nature".

Genetic sterility as exemplified in "shy-breeding lines" has been reported from time to time. The hereditary causal defect may vary, but in every instance the condition, from its very nature, is self-limiting. Such problems are extremely interesting to the geneticist who may be able to offer valuable advice and guidance in overcoming them when they arise.

On the other hand, bacterial infections may become established in the physiologically normal female during the course of pregnancy or at the time of parturition and may produce gross pathological changes in the genitalia. Septic metritis may so injure the lining membrane of the uterus as to render subsequent implantation of the embryo an impossibility. The fallopian tubes may becaome permanently blocked as a result of inflammatory changes, or the ovaries themselves may be involved in the septic process and so injured as to be rendered incapable of again producing viable ova. My own post mortem records of some 600 cows culled from dairy herds for all reasons show that 9% were suffering from such lesions. This figure is much lower than those reported from Europe and America and reflects the hygienic advantages of the Dominion's open-air dairy system.

The predisposing cause of such septic sequelae of pregnancy is in most instances Br. abortus infection. Contagious abortion leads to intra-uterine death of the foetus and retention of the foetal membranes, thus paving the way for the entrance of secondary bacterial invaders. It is the latter, rather than Br. abortus itself which set up the septic conditions leading to permanent sterility.

Efficient co trol of contagious abortion plus an adequate veterinary coverage would go a long way to minimise such losses.

Temporary sterility is much more common than the permanent type and the reduced efficiency of late calvers probably represents a much greater economic loss to the dairy industry. Temporary sterility in its varied forms therefore offers a much more attractive and potentially fruitful subject for study and research into preventive and remedial measures.

Both infection and nutritional unbalance play a big part in temporary breeding failure. I believe that both together may play a part in some instances and am still not prepared to say which I consider the more important.

To consider infection first, - The major causes are in order of importance:

(a) Cervicitis or catarrhal inflammation of the uterine cervix. This condition is almost invariably streptococcal in origin.

(b) Low grade infections of the endometrium usually supervening on contagious abortion.

(c) Trichomonad infection, a protozoon parasite.

The bovine cervix with its tortuous passage and intricately folded mucous membrane seems, from its structure, particularly susceptible to the establishment of catarrhal inflammatory changes of bacterial origin.

It has been my experience that when an endemic form of temporary sterility occurs in a herd with a previously satisfactory breeding record, the cause is almost invariably a streptococcal cervicitis, if a change of bull brings no immediate improvement. The infection is transmitted from cow to cow by the herd sire. The latter not infrequently himself becomes infected and discharges large quantities of streptococci in his seminal fluid.

The condition has a tendency to spontaneous recovery after a period of two to three months when the symptons subside and the cows begin to settle to service. As a rule the majority of the cows in the herd are affected and very few settle in calf until after the turn of the year.

Satisfactory treatment presents difficulties owing to the intricate folding of the cervical mucous membrane and the most satisfactory method seems to be the introduction, under pressure, of bland bactericidal agents in paste form on the grease-gun principle. It is only by such a method that one can hope to penetrate into the numerous folds and corners of the infected mucous membrane.

My own post mortem records show that over 57% of some 300 barren cows showed evidence of cervicitis with or without other lesions. Similar records of some 270 pregnant dairy cows culled for slaughter during the months of April and May revealed that cervicitis was present in 32%. The average length of gestation in this group was 2.6 months as against an average of 4.2 months in the remainder. The cervix appeared normal in the latter and 4.2 months approximates the average expectation.

Apart from endemic streptococcal cervicitis sporadic cases of cervical infection due to other organisms are met with in most herds.

Catarrhal endometritis as a sequel to parturition occurs sporadically in every herd. The endometrium possesses remarkable recuperative powers and quite a number of cows which show evidence of leucorrhoea for some weeks after parturition ultimately clear up spontaneously.

Trichomonad infection occurs from time to time in endemic outbreaks. Infection is transmitted by the herd sire. Some cases respond well to appropriate treatment, others are resistant. It usually requires several successive seasons of unremitting attention and treatment to finally overcome an extensive outbreak.

The writings of many workers during the past 15 or 20 years have given us a fairly clear picture of the various parasitic forms of sterility but much research requires to be done before we can conscientiously say that we have in our hands the necessary means to effectively control and cure them. In particular I believe that extensive controlled trials should be carried out with the newer "sulpha" group of drugs, among others. Packing the uterus with powdered sulphanilamide has apparently produced spectacular results in my hands in one or two instances of septic metritis.

Better education of the farmer in breeding hygiene will help very considerably. Both this and skilled treatment of affected herds demand the services of the graduate veterinarian and progress must be slow until the Dominion possesses an adequate veterinary service.

Nutritional sterility is in practice almost invariably temporary in character. The classic example is of course Sir Arnold Theiler's work on acute aphosphorosis in South Africe where female cattle on a phosphorus deficient diet remained indefinitely in the anoestrus state. A similar state of affairs was not uncommon in New Zealand some 15 or 20 years ago on poorer country prior to the universal use of phosphatic fertilisers. In those days one would receive reports during January that half or more of the herd had still to come in season and take the bull for the first time. Such cases were associated with extremely low P205 content of the pastures. Analyses ranged from 0.25% upwards as against the optimum of about 1% P205 in the richest pastures. In such cases supplementary phosphates produced dramatic results.

The high producing dairy cow makes heavy demands upon her system for phosphorus for both production and reproduction and a survey carried out in Taranaki some years ago established a highly significant positive correlation (r = plus .5) between the P205 content of the pastures and the ratio of matings to conceptions in the 42 herds covered by the survey. The ratio of matings to conceptions varied from 1.1 to 2.1 and the P205 content of the pastures from 0.68% to 1.2%. Phosphorus values such as these would not normally be associated with mineral deficiency symptoms. The results of the foregoing analysis may be of particular importance at the present juncture since the P205 content of our dairy pas-

tures may be falling owing to the great reduction in the available phosphatic fertilisers. If such is in fact the case then we may expect a general increase in the number of returns to service. To counteract any such possibility it would seem sound practice to foster the widest possible use of phosphatic licks.

In ewes too an increased phosphorus intake at times produces a marked effect in improving the general efficiency of the flock. Natal mortality is reduced, the lambing percentage increased and the milking capacity of the ewes improved.

Of recent years the literature has contained a mass of data pointing to the profound influence of many dietary factors on the reproductive function. However interesting and valuable they may be one feels that these experiments on laboratory animals and using highly purified diets are still of academic interest rather than of practical value to the animal husbandman, since it seems fairly well established that pasture contains an abundance of the vitamins, amino acids, etc., requisite to normal reproduction. Whilst it would be unwise to dogmatise, it does seem on the balance of evidence that such experimentally demonstrated deficiency effects are unlikely to occur among New Zealand livestock.

Temporary sterility is also at times associated with a type of post-parturient dyspepsia of a chronic nature but which is as yet little understood. Affected animals have a poor appetite and there is evidence of a general endocrine upset as evidenced by low milk yield, low blood calcium and magnesium and a ketonaemia. Such cases are met with under the more intensive methods of dairying on heavy land and provide an interesting research problem which still awaits solution.

I have left to the last mention of cystic corpus luteum. This is one of the commonest causes of either temporary or permanent sterility but I do not know in what category to place it. It is apparent that any central cyst in the corpus luteum is incompatible with pregnancy. The cyst may be no larger than a mustard seed or it may occupy the greater portion of the corpus luteum. In my post mortem records this condition was very frequently associated with cervicitis in barren cows, but in an equal number the cystic condition was the only abnormality recorded. American workers have claimed that the cystic condition is the result of bacterial infection but cultural tests have entirely failed to substantiate this inso far as my own experience goes. When the luteal cyst is of any appreciable size its presence can be detected by a rectal examination. It can be readily ruptured and the corpus luteum expressed. An animal so treated will often conceive if mated at the next heat period.

So far as I am aware, no very detailed histological work has been done on their origin and the thought occurs to me that it is possible that some other tissue elements than thecal cells may at times become involved in the rapid proliferation of the developing corpus luteum. Whatever its source, it does certainly appear that the fluid filling the cyst may contain some substance of a hormonal nature which is definitely antagonistic to a receptive state of the endometrium. There I must leave it as my allotted time is up - an interesting and unsolved problem for some keen researcher.

DISCUSSION

Professor K.A. Wodzicki: How many cases of cystic follicle sterility are included in the cervicitis sterility cases?

Do the reported figures of cystic corpus luteum, reported in the paper, include also persistent follicles, and what is the percentage of the latter?

Reply: Some 50% of 315 barren cows examined showed evidence of cervicitis and fully one half of these also had cystic corpora lutea. On the other hand the corpus luteum of pregnancy never shows any sign of a cystic condition. I have no

information regarding the percentage of persistent follicles which cannot be differentiated on macroscopic examination.

Dr Filmer: Stated that it was doubtful if a variation of P 0 content from 0.68 to 1.2% in the pasture could per se effect any great increase in fertility. It was more likely to be a reflection of other qualitative changes. He asked what was intended by education in hygiene, and if there was anything in this line which could be promulgated through existing channels.

Reply: The apparent relationship between the P205 content of pasture and fertility was surprising in view of the much lower figures associated with sterility elsewhere. On the other hand New Zealand production from unsupplemented pasture is infinitely higher than that obtaining in other parts. The late Sir Arnold Theiler was most impressed with the data which he examined in great detail and satisfied himself that the relationship was statistically highly significant.

I was not referring to anything new or revolutionary in matters of herd hygiene, merely stressing the importance of such routine measures as irrigation of the bull's sheath after each service, hand service in preference to running the bull with the herd, withholding from service any cow showing evidence of vaginal discharge, better control of contagious abortion, avoidance of restocking with saleyard cows, etc.

Dr L.R. Richardson: Has there been any attempt to establish the colon as a reservoir of trichomonads parasitic in the genital tract?

Reply: Not so far as I am aware.

Dr I.L. Campbell: In children, infections of the reproductive tract are successfully treated with estrogens. Does Mr Webster know of any work along these lines in livestock?

Reply: I do not think so. Oestrogens seem to so stimulate the immature reproductive tract of the young as to materially assist it in throwing off certain infections, but I do not know that they have proved of any value in curing venereal infection in the sexually mature human female.

Mr R.E.R. Grimmett: asked if there were any correlation between the nitrogen content of the pastures and the degree of temporary sterility in the Taranaki herds investigated.

Reply: The data did not even suggest a possible relationship between the nitrogen content of the pastures and the incidence of temporary sterility.

Dr C.M.S. Hopkirk: Speaker agreed that a series of trials and further record work is required to gain more knowledge of female sterility in New Zealand.

The question as to whether cervicitis was generally responsibile for temporary sterility or was as common as was stated was doubted.

The fact that in outbreaks of mastitis in dairy herds in the spring of the year temporary sterility in many of the affected cows was also noted, suggested that temporary sterility was of nutritional origin rather than an infection.

Subacute trichomoniasis was a possible source of temporary sterility and much more observation should be carried out in affected herds with more recent methods of diagnosis to see whether that parasite were implicated.

Reply: I started my work with a perfectly open mind and was led to the conclusion that cervicitis was the commonest cause of endemic temporary sterility by the evidence I accumulated over a period of some six years work. The same view is held by leading workers in other parts of the world, and I prefer to maintain my opinions until concrete evidence to the contrary is produced.

Sporadic temporary sterility is certainly fundamentally nutritional in origin in many instances - e.g., the relationship between the P205 content of pasture and breeding efficiency. I also regard it as highly probable that other conditions such as mastitis, ketosis, etc., may be factors.

Trichomoniasis is another possibility which requires further investigation and which may prove commoner than is at present realised.

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