THE CERVICOPLASTIC TREATMENT OF STERILITY.*

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In its clinical designation, the term sterility is purely relative, necessarily implying in a given case, the presence of approximately normal anatomic and physiologic essentials to conception, without the consummation of offspring. Physiologically, every woman who menstruates, ovulates. Biologically, ovulation predicates

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potential fecundity. The virgin ovary harbors from forty to sixty thousand ova. Ovulation, fertilization and nidation constitute the chronological cycle of conception and any perversion in their normal concurrence determines sterility. We cannot create a function, we can only attempt to activate one existing in dormant state, stimulate one deficient, or possibly mobilize one tentatively inhibited. The maturation of a Graafian follicle and liberation of its contained ovum, the evolution of the corpus luteum, the endometrial transmutation essential to deciduation, the subtle biotactic elements that dominate ovular fertilization and nidation are all susceptible to inhibiting influences, temporary or permanent, local or systemic, most of which involve problems far beyond our present diagnostic horizon and therapeutic scope.

Who can explain, why the conjugation of a perfectly normal female, with an equally normal male proves sterile, while the subsequent union of each with another, demonstrates the fecundity of both? To apply the serological hypothesis of "a selective ovular immunity to certain strains of sperma"—in explanation, is mere terminological juggling. Fecundity is a question of seed and soil, we cannot control the seed, we can only enhance its viability by correcting a deficient soil; we cannot control the ovule, we can only aim to correct certain endometrial abnormalities inimical to its fertilization and nidation.

The endometrium must provide a medium, conducive to the virility and progression of the spermatozoon in its fertilizing mission, and must respond normally to ovular fertilization with a concurrent activation of its decidual potentialities essential to normal nidation.

This functional complex, reveals the endometrium as a highly specialized tissue, to be assiduously conserved and not to be harrowed and scraped with impunity. It presents the clinical dominant in sterility of cervical origin and establishes the rational limits of our present therapeutic range.

Functionally as well as structurally, a sharp line of demarcation differentiates the corporeal endometrium from the cervical mucosa, but the one feature of greatest practical significance, is the remarkable disparity in pathological manifestations displayed above and below the internal os. According to established modern conceptions, chronic corporeal endometritis is extremely rare, our former acceptance of the condition was based upon misinterpretations of the normal endometrial changes characteristic of its menstrual cycle.

This relative immunity of the corporeal endometrium stands in

striking contrast to the extreme susceptibility of the cervical mucosa to infectious invasion, *but for this relative immunity of the corporeal endometrium*, the extreme prevalence of chronic endocervicitis, would render the largest majority of women sterile, for *a diseased cervical mucosa is capable of immobilizing and destroying spermatozoa.*

The marked spermatocidal effect of a diseased cervical mucosa is vividly depicted by Reynolds, who, utilizing Hühner's postcoital method for the direct observation of spermatozoa aspirated from the cervical cavity, states:

"It is extremely interesting to see how actively mobile spermatozoa progress across the field of the microscope in a cervical secretion of grossly normal appearance, until they come in contact with some clump of pus cells, with which the tail of the spermatozoon becomes entangled. The result then is, that it indulges in futile struggles to escape, by the violence of which, it becomes exhausted and in a few minutes gives up the struggle and lies still."

But beyond the direct devitalization of sperma by the diseased cervical mucosa, other factors, of equal and greater potency in the causation of sterility, may be incited through the influence of cervical disease upon the functions of the uterus as a whole, for, *while chronic endocervicitis never extends to the corporeal endometrium by direct continuity*, a sterility accompanying the former *is due in large part to the functional derangements induced in the latter.*

Chronic endocervicitis with derangement of uterine functions presents the most familiar symptom complex in gynecology, nevertheless the nature and sequence of the pathologic phases, that link the one to the other, constitute a gap in our knowledge, which is further obscured by standardized misconceptions productive of the hit and miss empiricism that characterizes prevailing corrective methods.

The correlation of these pathological links finds ample elucidation in my previous publications on tracheloplastic methods, hence the most salient features only will be reëmphasized here.

The specific functions of the uterus necessitate the widest possible range in the control of its blood supply and like the heart it automatically controls its fluctuating circulatory demands by intermittent cyclic contractions, not only during gestation, but throughout its entire functional existence.

The cervical cavity serves as atrium or reservoir to the uterine cavity proper and while its amplitude, as a portal of ingress for spermatozoa, has dominated the mechanical and surgical treatment

of sterility for ages, its efficiency as an excretory duct has not attracted consideration.

A cervix that offers sufficient egress for millions of blood cells during every menstruation, will readily afford ingress to a spermatozoon, whose diameter measures less than that of a single red corpuscle; moreover we constantly encounter fecundity in cases of so-called pin-hole os and sterility in widely gaping lacerated cervices.

The cervix is lined with racemose glands, whose secretions must find outlet in the normal and more especially in an abnormal state; under normal conditions, this drainage is effected, not *merely* by a simple outflow, through a passive os, but by the active rhythmic expression resulting from uterine contractions.

To comprehend this mechanism and realize the full significance of endocervical disease upon uterine function, we must discard our present conceptions of the myometrial arrangement and the fallacy of a cervical sphincter.

The architectural design of the cervical musculature precludes any concentric closure of its outlet, which dilates with every uterine contraction, because its muscle fibers, which are directly continuous with those of the corpus uteri, do not at any point completely encircle the cervix, but are disposed in serried successions of oblique circle segments, which, by contracting spirally upward, necessarily shorten every diameter of the uterus and by their uncoiling in the cervix widen the os like an iris diaphragm in a microscope.

Cervical dilatation thus becomes an integral part of uterine contraction instead of a passive dilatation in a hypothetical sphincter and incidentally explains the apparent obliteration of the cervix during parturition.

In the cervix, as elsewhere, every infection incites the greatest reaction in the lymphatic elements.

The cervical lymphatics may be traced from their lacunar origin in the cervical canal, through minute funnel-shaped ostia, *directly to the muscular coat*, where they expand into an extensive capillary net, which enmeshes every fascicle and bundle of the uterine musculature to the peritoneal surface, whence they drain into their main collecting channels at the base and top of the broad ligament (Leopold).

Thus the normal course of the lymphatics conveys an infection from the cervix—*not to the corporeal endometrium*—but along the intramuscular planes of the uterus, where it impairs uterine contractions by infiltrating the muscle sheaths resulting in nutritional

deficiency; progressing to the periadnexal network, it inhibits tubal peristalsis and agglutinates the fimbrial ostia by adhesions, finally reaching the ovaries, it impedes the normal rupture of Graafian follicles by thickening of the ovarian tunica albuginea, thus completing the chain of pathological events that link cervical disease and sterility.

Crystallized into a concrete postulate, chronic endocervicitis presents the key to the therapeutic problem in sterility of cervical origin, and the success of any curative attempt upon the cervix will be proportionate to its elimination of an existing endocervical infection.

The question as an abstract proposition, whether a tracheloplastic widening of the cervical canal cures sterility by facilitating the ingress of spermatozoa or the egress of deleterious secretions, would appear of more academic than practical interest, but applied to the individual case it acquires the significance that distinguishes between rational procedures and empirical practice.

Every curative attempt should be based upon a correct perception of indications and a definite realization of its aim and scope. We must know why we operate, in order to determine when and how to operate.

In general and special gynecological practice, dilatation and curettage still hold sway as the panacea for sterility, and while discriminate dilatation may prove of benefit in some very mild infections by establishing temporary drainage, curettage cannot be too emphatically condemned in any case.

The curet cannot reach the deeply situated racemose tufts of the muciparous glands in the cervical lining and it should not injure the utricular tubules of the corporeal endometrium, which are rarely if ever involved in the disease and whose specific functions in menstruation and gestation may be permanently vitiated by the lacerations and inoculations incidental to this time-honored traumatism.

Boldt states: "Next to curetting, dilatation of the cervical canal, principally to overcome sterility, is the operation most frequently done without proper indication. Those who have made observations will probably concede, that in 75 per cent. of patients so treated, the intervention is unwarranted."

The cervicoplastic operations in vogue for the cure of sterility comprise the so-called discission and amputation, that is—the cervix is either split or ablated.

Many women undoubtedly conceive after one or other of these operations, as they occasionally do after dilatation and curettage,

but to attribute an eventual fecundity to the curative effect of this or that procedure, is an obvious "*post hoc*' interpretation, for it must be conceded that, on the one hand, many women presenting operative indications, ultimately conceive without any intervention whatsoever, while on the other hand, a very considerable number of operations prove utterly futile of results.

Chronic endocervicitis involves the entire cervical mucosa from external to internal os and it follows that a partial ablation of the cervix can result only in a partial elimination of the diseased area while a high amputation for sterility is an unwarranted mutilation that defeats its ultimate object.

One hundred and twenty-eight complete postoperative histories from among four hundred recorded cases tabulated by Leonard in Howard Kelly's clinic, revealed 80 per cent. of sterility after low amputation for cervical lacerations.

In other words, eight out of ten women, of established fecundity, are sterilized by prevailing methods of cervix amputation and while we recognize an occasional postpuerperal sterility as a possible sequel of birth injuries its occurrence never attains to such proportions.

Just as long as endocervical disease persists, so long will its resulting sterility persist.

The normal cervical lining is not essential to conception or gestation, but a diseased lining is inimical to both.

All cases of chronic endocervicitis, characterized by erosions and mucopurulent secretions, are incurable by any means short of complete enucleation of the entire endocervical mucosa from external to internal os and the relining of the denuded canal by a cylindrical flap from the vaginal sheath of the cervix.

The preceding pathological indications and technical demands are fulfilled by the following procedure, which is essentially identical with that submitted by me for the tracheloplastic cure of infected cervical lacerations.

The main steps in the operation consist of:

1. Outlining and free liberation of an ample cuff from the vaginal sheath of the cervix.

2. Enucleation of the entire endocervical mucosa from its surrounding muscular bed, up to the internal os.

3. Sutural inversion of the vaginal cuff into the denuded cervical cavity.

To secure an ample cuff of vaginal mucosa, an outlining incision is made to encircle the eroded area around the external os.

The edge of the flap thus outlined is freely liberated as a cylin-

drical sheath completely around the entire cervix to the level of the internal os.

The eroded external os and the entire cervical lining, are now cored out of the surrounding muscular layer to the internal os, as a complete cone.

In a congenitally deformed cervix, its muscular framework thus exposed, may now be advantageously reshaped by appropriate incisions on the lines established by Sims, Pozzi, or Dudley, according to indications or predilection.

The tubular flap of vaginal mucosa is not included in any of these corrective incisions to which no individual stitches are applied, as the two main retentive sutures secure all necessary coaptations.

These retentive sutures are introduced as follows:

Beginning at the anterior edge of the cylindrical flap, a long heavy strand of silkworm gut is introduced on its vaginal surface, transversely through its center, like the first loop of a mattress suture, one-eighth of an inch from the free border and embracing one-eighth of an inch of tissue, this suture hangs free until a second suture is passed through the posterior edge of the flap in a correspondingly similar manner.

The right free end of the anterior suture, threaded in a specially bent Peaseley needle, is now carried into the cervical cavity to a level just above the internal os, whence piercing the cervical musculature in a direction forward, upward and slightly to the right, emerges on the vaginal surface at the base of the flap.

The left free suture end is directed in the same manner forward, upward and to the left, so that the two suture ends diverging slightly in their course, reappear in the center of the anterior vaginal fornix, about one quarter of an inch apart.

The free ends of the posterior suture are passed in a corresponding posterior direction and emerge in the center of the posterior vaginal fornix.

By tightening and tying each individual set of suture ends, the tubular vaginal flap is drawn into the denuded cervical cavity, thus relining its entire raw surface, at the same time it is automatically interposed between the edges of any supplemental incision or excision of the cervical musculature, preventing their reunion and is approximated to the circumference of the internal os, where it is firmly retained in close apposition until the sutures are removed.

Drawing too short a vaginal flap into the cervical cavity will foreshorten the anterior vaginal wall and tilt the uterus backward.

To lengthen a congenitally foreshortened anterior vaginal wall,

as suggested by Reynolds, it is only necessary to incise the anterior flap segment transversely and pull this transverse incision into a longitudinal slit before passing the main sutures which emerging at the sides of the slit, coapt and retain its edges in the longitudinal axis.

Additional sutures are usually unnecessary, the silkworm ends are left long to facilitate their removal and tucked into the vagina.

A narrow strip of iodoform gauze introduced into the cervix with the object of maintaining flat coaptation of all raw surfaces completes the operation.

This gauze is removed on the third or fourth day, when the patient is permitted to walk about.

The stitches are not removed until the end of the third week.

I would not dogmatically attribute the cure of sterility to this operation any more than to other procedures, for there is too much that is unknown and unknowable involved in the problem, but I may assert, after a very extensive and critical trial, that the method as outlined, radically eliminates chronic endocervicitis, the one established causative factor in the sterility of cervical origin.

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