laudable enterprise—but as a description of what is now intended, misleading when applied to readjusting adults or old men. The processes of orthopedics involve motion, prompt or graduated, applied by the hand or by apparatus or by cutting such structures as are permanently obstructive. It is a process of remedying through encouraging motion in a living mechanism or organism.

The process is one of kinesthesia or kinesis, or kinesiatrics, or of akinesis, the life element is indicated by the root term "bion." An even better one, the term orthobiotic, is also used by the French to embrace a larger group of subjects, the straightening out of life, the whole of life processes, including the mind—psycho-orthobiotics.

I would submit that we might do worse than to adopt and recommend this comprehensive term orthobiotics and the special one bio-kinetics as descriptive of that entire enterprise of readjustment, restitution, rearrangement, rather than orthopedics as now conceived and employed.

Finally, the great domain of physical hygiene, regulation of conduct (chiefly physical) and of behavior, chiefly of the affective faculties,—feelings, emotions, etc.,—which needs a better presentation through improved terminology. The aim is harmony throughout all actions and interactions, all manifestations of energy, and even here the one phase most comprehensive is bio-kinetic regulation.

ENDOMETRITIS: ITS PATHOLOGY AND DIFFERENTIATION FROM MALIGNANCY.*

BY W. CLAUDE USHER, M.D.

PERHAPS no other medical term is used with less precision than endometritis. Many authors, as Dudley, Hirst, etc., speak of it as an independent disease, but many, like Ashton, include under endometritis, all uterine inflammations. A short paragraph from Ashton's "Practice of Gynecology" will make this clear: "The starting point of inflammatory affections of the uterus is, generally, the endometrium, and the disease may, eventually, extend to the muscular walls of the uterus and even to the

peritoneum. Metritis, or inflammation of the uterine parenchyma is, therefore, not a distinct or separate disease, but a condition that is secondary to an infection of the endometrium. Sometimes the peritoneal coat of the uterus is primarily affected, and the disease subsequently involves the parenchyma of the organ, and finally the endometrium. These cases are, however, comparatively rare, and are due to septic inflammation in one of the pelvic organs causing an adhesion between it and the uterus. Again, a laceration of the lower segment of the uterus may be followed by septic infection which may extend into the parenchyma before involving the endometrium."

"In the light of modern pathology, inflammation of the uterine mucosa becomes a subject of vital importance, since it is the starting point of nearly all the inflammatory lesions of the pelvic organs."

Such a statement coming from this well-known authority brings us to a fuller realization of the importance of the endometrium in pelvic disease. While Dudley states that "the most significant factor in metritis is the endometrium," his anatomical classification of metritis into endometritis, myometritis, and perimetritis, lessens the student's appreciation of the importance of the endometrium.

Bewildering, is indeed a mild term to express the variety of classification for endometritis. Bases for such have been etiological, pathological, anatomical, and clinical. Etiologically, endometritis may be said to be: (1) Bacterial (gonococcus), tubercle bacillus, streptococcus, staphylococcus, or saprophytes; (2) non-bacterial (secondary to uterine tumors, cervical lacerations, uterine displacements, etc.). Pathological classification will be considered later; anatomical has already been illustrated (Dudley), and it remains only to speak of the clinical classification, viz., (1) acute, (2) chronic. In routine gynecological work, these are the two forms most often differentiated; further subdivision of the chronic form into glandular and interstitial types sometimes occurs.

The acute or septic form of endometritis is uncommon, but may follow injury, or more usually pyogenic, saprophytic, or gonorrheal infection of the mucosa. Pyogenic infection with streptococcus or staphylococcus occurs usually after labor, or abortion. However, an acute endometritis may be associated with cer-

* This paper was written by Dr. Usher, not long prior to his death in 1910, as a part of his work in fourth-year gynecology in the Harvard Medical School. It is printed now as a tribute to the memory of this young hospital interne of much promise.

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tain infectious diseases, as typhoid, diphtheria, etc. Pathologically, the mucous membrane is swollen, congested, loosened in attachment, hemorrhagic, and covered with mucopurulent exudate. Necrosis of superficial membrane usually occurs with formation of granulation cell tissue, in its deeper layers walling off the process, and preventing absorption. In severer grades, the organisms may penetrate the lymph channels, blood spaces and myometrium, leading to suppuration in the uterine wall, peritonitis, salpingitis, oöphoritis, and probably septicaemia.

A clinical or etiological classification of chronic endometritis must be very wide. Thus we might speak of the simple chronic forms including the congestive and constitutional types or of the septic chronic forms including the tubercular, gonorrheal, etc. The chronic endometritis is the usual course in a gonorrheal infection, and leads to the pathological pictures soon to be presented. Tubercular endometritis is, of course, usually secondary to tubercular tubes or cervix, and presents a swollen mucosa with characteristic elements of tubercular tissue, *viz.*, caseous material, tubercles, giant cells, etc., and may be of the miliary, interstitial, or ulcerative types.

Prolonged inflammatory disturbances, whether the result of infection, or of some uterine condition, as displacement, subinvolution, etc., causing congestion, results eventually in increase of both adenoid and of interstitial tissue, *viz.*, hyperplasia. Pope describes it as beginning with a glandular proliferation following microbe infection, or the above causes, with extension to the surface of the underlying parenchyma, and resulting in the formation of embryonal cells. The epithelial surface of the endometrium is raised, and proliferation of the epithelial cells occurs with enlargement, and twisting of the gland tubules. Clinically, this stage might be regarded as a simple endometritis with leucorrhea, but no interstitial hypertrophy. Round celled infiltration of the interglandular connective tissue next occurs, so that the glands become more widely separated by the increased stroma. The round cells may in turn become spindle, and epithelioid in type, and by pressure on the ducts cause swelling of the glands, and retention cysts. The vessels increase in number and size, so the uterine wall becomes congested and we now get the second clinical stage, with leucorrhea and interstitial

hypertrophy plus engorgement. Thus, the whole process is really one of young vascular, connective tissue formation, which by successive transformation into spindle cells, fibrils, etc., becomes scar tissue. This continuous pressure on the glands results in their atrophy and disappearance, at least from the surface layers of the mucous membranes. Thus we have the atrophic stage (sometimes termed senile endometritis). To sum up, therefore, the hyperplasia described by Pope and Hirst may be either glandular, interstitial, or of mixed type, *viz.*, both glandular and interstitial. Dudley's histological classification of chronic endometritis into (a) glandular or parenchymatous with hypertrophic and hyperplastic types, and (b) interstitial, agrees well with the above outline.

In Abel's Gynecological Pathology, endometritis is viewed as inflammatory, and non-inflammatory in origin. Those changes resulting in certain forms of tissue proliferation due to inflammatory disturbances, he speaks of as being either atrophic (interstitial) or hypertrophic in type. This interstitial type agrees with the changes already outlined, *viz.*, round celled infiltration of the stroma, the cells becoming spindle and epithelioid, and proceeding to the organization of scar tissue with subsequent atrophic changes. The hypertrophic type is characterized by simultaneous affection of both glandular and interstitial tissues. It differs from the atrophic (interstitial) type, in that no disappearance of tissue occurs as a terminal event. The glands increase in number and size, and in some places the lining epithelium may become proliferated and exhibit several layers, but never any lawlessness of growth as in malignancy. Through growth and infiltration of a vascular stroma, the glands may be thrown into folds, or by pressure, dilated with cyst formation. The changes in the stroma correspond closely to those already described.

Non-inflammatory conditions present in the mucosa result in what Abel styles hyperplasia, *viz.*, overgrowth of the mucous membrane without microscopical evidence of inflammation. The mucous membrane may be thickened throughout or only in part, giving us (1) hyperplasia of the endometrium, which may assume a diffuse or circumscribed (polypoid) form, (2) hyperplasia of the glands, which we usually designate "chronic hyperplastic en-

dometritis." In this latter form, the interstitial tissue is relatively much reduced, the lining epithelium of the glands may be thickened and in several layers. From the fact that, while such a type, resembling, as it does, malignant adenomas, yet retains its glandular formation, and its epithelial cells always respect the boundary of growth formed by the membrana propria we may style it 'Benign Adenomatous Disease' of the endometrium (Roberts). Such are the cases which require careful microscopic differentiation from neoplasms.

It remains to speak of a type of true inflammatory endometritis usually occurring after an abortion, *viz.*, deciduomal endometritis. While this retention of placental tissue may be the primary cause of the existing inflammation, the latter is, as a rule, a continuance of or a flaring up, of an old septic or gonorrheal infection. The changes are confined principally to the interstitial tissue which shows the decidual cells being separated by numerous round cells with the interpolation of mononuclear leucocytes.

Pathology has been observed to recognize non-inflammatory affections of the endometrium resembling ordinary forms of endometritis in hypertrophy of both glandular and interstitial tissues. Such recognition has not yet attained a firm basis in clinical routine. We still usually label a case with persistent menorrhagia, metrorrhagia, and leucorrhea as a chronic endometritis. Probably two reasons will account for this general tendency on the part of practitioner and specialist alike: (1) a trend in gynecology to avoid minor lesions, (2) the revelations of bacteriology as to the bacterial origin of endometritis, causes the assumption of an infectious nature for any similar symptoms complex. The following case illustrates my contention:

Mrs. S., 23, newly married. Her menstrual periods had always been regular, lasting only five days. She became pregnant and passed through a protracted confinement with a puerperium which, though free from fever, was not ideal. Subsequently, her periods lasted eight to nine days and were accompanied by a profuse leucorrhea. Endometritis was diagnosed and patient continued so for four years, when she again became pregnant. Her confinement was easy and natural, her puerperium satisfactory, except for a continued colored discharge lasting for eleven weeks. Ten days after its

cessation, it returned, lasting two weeks. Her periods now continued for ten days, yet she became a third time pregnant. Thus her condition was attributed to chronic endometritis, in spite of the fact that she never had any inflammatory disturbance, nor any sign of infection, and no possible irritation by local medication.

Fortunately it is more and more now recognized that many conditions still termed endometritis are non-infectious. Such conditions we meet with as the French "*Mérite des vierges*" or in German authorities as "*Essentielle Blutungen*." While imperfectly understood, these conditions are excellently reviewed by Fothergill. The basis for his conclusions rest to a large extent upon an early article by Dr. Donald, "*Chronic Endometritis and Metritis in Virgins*," which I must briefly outline. Dr. Donald reports some forty cases of such conditions in women where all possibility of infection or neoplasm was excluded by selection, *viz.*, all were virgins with history of no operative interference, etc. His study, resulting in the following three groups of cases, prove that the conditions were non-inflammatory: (1) With small cervix,—acute ante flexion or retroflexion. Small corpus, which, under ether, is found to be slightly enlarged and elongated. There was often stenosis; (2) with ordinary cervix, a large and heavy corpus, either anteverted to excess or retroverted; (3) hypertrophied cervix with cervical catarrh. He found pain in lower abdomen in 36 cases; dysmenorrhea in 33; leucorrhea in 31; and menorrhagia in 16. Microscopical examination of curettings obtained generally revealed the utricular glands of normal structure, but increased in number; the interglandular tissue swollen and oedematous, but poor in cellular elements, or else normal; occasionally the interstitial tissue showed more fibrous tissue than normal. The vascular structures usually presented no marked changes. Hence his conclusion was a general hypertrophy of the uterine mucosa.

This form Donald has styled idiopathic endometritis or benign adenoma of the endometrium. Such cases have been reported by Donald, Walls, and Ostrom. It was first suggested by Sinclair that these hypertrophic conditions of the endometrium resulted from defective development of the uterine muscle.

Theilhaber, who stands prééminent amongst German research workers in this line, points

also to the common diagnosis of chronic endometritis in cases of excessive bleeding and leucorrhea without obvious cause. This diagnosis of primary endometritis, he believes to be erroneous, its real nature being hypertrophy secondary to a primary lesion of the mesometrium. Similarly, chronic metritis is generally believed to be an inflammation of mesometrium secondary to infective inflammation of the endometrium. He holds that in the majority of cases of metritis there is no primary inflammation of the endometrium but rather a primary defect of the uterine muscle with secondary hypertrophy of the endometrium.

Basis for this belief is worked out on the fact that uterine contractions play a great part in regulation of the pelvic circulation. As inefficient and infrequent muscular contraction may give rise in people who stand a great deal, to varicose ulcer, so with insufficient uterine contractions we may have a resultant venous stasis and consequent overnutrition, and hypertrophy of the endometrium. This muscular inactivity may be especially marked during menstruation, when the pelvis is already congested. Thus we may have the periods prolonged, and an intermenstrual leucorrhea.

Normally, the uterine musculature develops in physiological relation to the vascular element. In the child's uterus only one-third the tissue is muscle, and the blood vessels are narrow and thin walled; the remainder is connective tissue. Towards puberty, the muscle and vessels begin to increase; the connective tissue becomes less and less in amount, until at the age of 10-20, when menstruation is usually established, the uterus contains twice as much muscular as connective tissue. In the healthy woman, who remains unimpregnated, these proportions remain until about 40 years of age. When a pregnancy intervenes, the muscular and connective tissue both increase rapidly to the time of parturition, then quickly fall back. The uterus remains larger after each successive labor, but this is due to a relative increase in connective and not in muscular tissue, since the involution of connective tissue does not proceed to the same degree as that of muscular tissue. The weight of an average multiparous uterus is 35-40 gms., while that of the average parous organ is 60-70 gms. Another change begins some years before the last menstruation; the uterine muscle fibres

atrophy, vessel walls thicken, the lumina being lessened by a sort of physiological arteritis obliterans, the amount of connective tissue increases. When menstruation ceases, the process is not complete, but continues until at 60 years of age, the muscle, as at childhood, forms one-third of uterine tissue. Theilhaber has a curve to illustrate this process of evolution.

We are now in a position to appreciate the clinical conditions which may arise from the muscular inefficiency above spoken of. After puberty menstruation may be excessive and metrorrhagia may persist, due to the fact that muscular development has not kept pace with vascular development. Curettings at first show a normal, but finally an hypertrophied mucosa. Again at the climacterium we get a good deal of muscular atrophy, which is accompanied normally by stenosis of vessels and lessening of blood stream. If the vascular change be delayed, we get venous stasis and engorgement, and hence hypertrophy of connective tissue, giving a chronic metritis and endometritis, characterized clinically by metrorrhagia and leucorrhea. Frequent parturitions may eventually produce a similar condition, resulting in subinvolution which, according to Theilhaber, is an exaggerated muscular atrophy during the puerperium, followed by hypertrophy of connective tissue, and not, as generally taught, an incomplete physiological postpartum atrophy of uterine muscle. In fact, it is superinvolution plus hypertrophy of connective tissue. A similar muscular inefficiency, due to degenerations, may account for the metrorrhagias so common in chlorosis, phthisis, and the acute infections.

The later microscopic researches of Dr. Anton Meier, in some 61 uteri, determine the relative percentages of muscular and connective tissue at varying ages (5 months to 75 years). His results corroborate Theilhaber's clinical curve, establishing beyond question the influence of parturition, age, and the menopause. Lorentz investigated nine uteri removed on account of intractable hemorrhage and found thickening of the wall, numerous large vessels, with microscopic increase of connective and decrease in muscular tissue.

Theilhaber, in summing up his own work and that of Meier, proposes two forms of endometritis, (a) primary endometritis with or without secondary mesometritis; (b) primary disease of mesometrium with or without sec-

ondary hypertrophy of the endometrium. Under (a) come infective conditions of the endometrium, as gonorrhea, puerperal fever, tubercle bacillus, etc. Under (b) come hypertrophy of the virgin uterus, muscular degenerations from general diseases, uterine fibrosis following diseased appendages, subinvolution, change in fibroids, and senile uterine changes. Of course, you may have inflammatory changes in any uterus, along with the results of muscular inefficiency. Donald's cases prove beyond question the existence of these non-inflammatory conditions. In the light of Theilhaber's work, the term endometritis should be reserved for infectious conditions and not applied to every case presenting leucorrhea and menorrhagia.

Differential diagnosis of endometritis is of interest and importance chiefly as to the question of early malignancy. With carcinoma we have hemorrhage, discharge and often pruritus vulvae. The character of the hemorrhage differs greatly from that of endometritis; it begins with a greater or less leakage, usually first noticed after sexual intercourse and defecation. There may be only a slight staining of a continuous leucorrhea; there may be only prolonged menstruation or continued flowing after the climacterium. Rarely is hemorrhage profuse, and no relation exists between the quantity of blood lost and between the loss of strength. The discharge is watery in character at first and of offensive odor, and as ulceration advances, we get mixture with blood, pus, broken down tissue, feces, etc. Pruritus vulvae often results from the irritating discharges. In endometritis, the hemorrhage is usually intermittent and profuse in character, not preceded by leakage. It usually bears more or less relation to the menstrual periods. Discharge is usually of mucoid or purulent character with less disagreeable odor. Vaginal examination at once reveals the hard, nodular infiltrations and the "crater-like" vagina, etc., of carcinoma. The importance of an early diagnosis of malignancy cannot be too strongly insisted upon, as the hope of cure by radical operation depends wholly upon it. Hence, we must, in every case of menstrual excesses, or of irregular uterine hemorrhage, take the precaution of an early microscopic examination of curettings, or of excised portions of the cervix in trachelorrhaphies; for only upon such a measure can a positive diagnosis of malignancy rest.

In this connection, the work of Cullen is especially valuable. He insists that, in examination of such tissues, we note whether there is any dipping down of the squamous epithelium on the vaginal portion of the cervix; whether finger-like masses protrude; is there loss of substance on the cervical surface, or any rounded infiltration of stroma; are the glands normal in number, and are they lined by one layer of epithelium; do they invade the deeper cervical tissues. In his work, Cullen points out cases of endometritis which present suspicious signs of early malignancy. Thus he instances swelling of the surface epithelium with proliferation to five or six layers, the individual cells being swollen and containing pale staining nuclei. He cites six or seven such cases with no evidence of nuclear division nor any tendency of epithelium to invasion of the underlying tissues. In one case there was proliferation even to five or six layers of lining epithelium accompanied by the formation of several new gland structures lined by a low cuboidal epithelium with enlarged lumina. In true carcinoma the glands are usually smaller, the epithelial cells cylindrical, closely packed together and stained deeply, also invading the underlying tissue.

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APPOINTMENT OF DR. RICHARD A. BERRY.—Announcement has been made that Dr. Richard A. Berry, professor of anatomy in the University of Melbourne, has been appointed Stewart lecturer in that university for the year 1921. In conjunction with Mr. S. D. Porteus, director of the research laboratory of the training school at Vineland, New Jersey, Professor Berry recently issued a report describing a practical method for the diagnosis of mental deficiency and other forms of social inefficiency. He will consider this subject in his Stewart course.