ON THE ETIOLOGY AND PATHOLOGY OF AREOLAR HYPERPLASIA OF THE UTERUS.

By A. F. A. KING, M.D.,

Prof. of Obstetrics and the Diseases of Women in the Medical Department of the "Columbian University," Washington, D. C., and in the University of Vermont; one of the Attending Physicians to Providence Hospital, Washington, D. C., etc.

In accordance with the "*New Basis for Uterine Pathology*," which formed the subject of my paper in the last number (August, 1875) of this Journal, I propose here to attempt an explanation of the pathological phenomena of "areolar hyperplasia," as we now, since the publications of Dr. Thomas, understand that term.

It is almost needless to say that the principles of biology upon which my "new basis" rests are those given to the world twenty years ago by Herbert Spencer (in his "Principles of Biology," 1866, and "Principles of Psychology," 1855)—principles which have been so generally admitted and so frequently elaborated by other writers, that they have now become common property in the treasury of scientific knowledge.[1]

The main principles or facts upon which the theory of the present paper rests may be thus briefly stated:—

1. Normal functional activity secures perfect structural integrity (other things being equal); but total absence of function *necessitates* organic atrophy.

2. In the structural integrity of the active organ, as also in the atrophy of the idle one, we recognize *a conservative adaptation of structure to function.*

3. Such conservative modifications of structure naturally follow a typical course, and are then usually latent and extremely slow in their development.

4. The *primary* causes of *atrophy* are such modifications of environment as fail to bring the function of the affected organ into play.

[1] In my first publication on this subject (see this Journal for November, 1872, p. 438) I could not forbear referring to Spencer by name, but deemed it unnecessary to do so in my last production.

5. Conservative modifications of structure of the kind specified can only reach their naturally designed typical termination, when the *new environment remains constant for a long time.*

6. Sudden vacillations of environment, especially if they are very decided, and often repeated, lead to inflammation of the modifying organ, particularly if the organism is at the same time chilled by exposure to cold; and in the absence of inflammation, they interfere with the typical source of the structural modification, causing it to assume a half-and-half, mongrel construction, in part adapted to a plurality of environments, but wholly and perfectly to no single one.

I must next call attention to the more common and decided anatomical phenomena of organic atrophy. They are these:

I. In *mucous membranes*, loss of epithelium (by desquamation and fatty degeneration); reduced vascularity from obliteration of blood-vessels and their conversion into fibrous tissue (hyperplased connective tissue).

II. In *muscular structures*, gradual disappearance of muscular tissue and its supplantation by connective tissue; later transformation of the new connective tissue into tissues resembling successively fibro-cartilage, cartilage, and bone. Coincident induration of organ and obliteration of many of its blood-vessels. Impaired contractile power.

III. In *serous membranes*, loss of epithelium on surface; agglutination of opposing surfaces together, from adhesions formed of connective tissue; impaired mobility of affected organ and obliteration of its vessels.

IV. Organs designed for active function have full supply of blood; when they do not perform their function blood accumulates in them, leading to congestion; prolonged congestion leads *invariably* to hyperplasia of connective tissue, and this last in time supplants the normal tissue elements of the congested organ.

In accordance with these principles I now propose to venture the somewhat startling announcement that the reproductive apparatus (I shall confine myself hereafter to the *uterus* alone) of the celibate female begins, after puberty, to *undergo a process of more or less latent, very gradual, organic atrophy*, which is naturally designed, when it shall have reached its

completion, to adapt the organs concerned to the total absence of function entailed on them by the abnormal environment of protracted celibacy.

I have said this atrophy is *more or less* latent:—not entirely so, however, be the case ever so typical a one. And why? Because the change of environment has necessitated so *very wide* a functional departure from the normal state. With the reproductive function there are no intermediate grades of functional exercise, such as belong to digestion, respiration, etc. Naturally each reproductive act is complete in itself. A woman is either pregnant or not pregnant: we cannot say she is more or less so. She is either a mother or she is barren. Environing changes necessitating so *extreme* a deviation from normal functional action can never be completely latent.

It is from this latent and partial, but progressive atrophy, from which arises the more marked evolution of connective tissue, to which Dr. Thomas has given the name of "areolar hyperplasia" (as will be presently explained), and from it also, as a beginning, the great majority of uterine diseases and displacements may be said to have their origin.

At present, unfortunately, direct anatomical evidence of this view is wanting. Histologists have not yet shown, nor indeed, that I know, have they yet sought to find the difference in elementary structure between a uterus that has reached mature development, and is prepared for procreation, but which has not yet been the seat of menstrual congestion, and one that for a series of years *has* been the seat of the menstrual process. I make no doubt, however, that evidence showing that in the latter case there exists a partial substitution of the normal tissues by connective tissue is forthcoming.

In old cases of "areolar hyperplasia," which are nothing more than cases of interrupted or disturbed instances of what was, before pregnancy, latent atrophy, we find the anatomical conditions very striking. Klob thus describes the second stage of the disease:[2] "The parenchyma on section appears white, or of a whitish-red color, deficient in blood-vessels, from compres-

[1] Abortions are *ab*normal; and while a female may bear one child or many, and have single or multiple births, these facts do not impair the meaning of the text.

[2] Quotation by Thomas, Dis. of Wom., 4th ed., p. 288.

sion of the tissues by the contraction of the newly formed connective tissue, or from partial destruction or obliteration of vessels during the growth of tissue; the firmness of the uterine substance is also increased, simulating the hardness of cartilage, and creaking under the knife."

Under the old term, "chronic metritis," Schroeder[1] thus describes the late anatomical changes of areolar hyperplasia: "When the proliferating process has ceased, the newly formed connective tissue undergoes cicatricial retraction, the vessels become imperforate, and the young mucoid connective tissue becomes firm and fibrillated. The uterus then again diminishes in size, and on section exhibits an exceedingly firm, almost cartilaginous tissue, which creaks under the knife, and has a white, anæmic, cicatricial appearance."

He further states (Ibid., p. 110) that in certain cases "the affection proceeds to induration (described by Scanzoni as the second stage of chronic metritis). The newly formed connective tissue undergoes cicatricial retraction, the uterus becomes harder and smaller, and premature amenorrhœa occasionally supervenes. The worst symptoms, the acute exacerbations, however, cease, and this process must therefore be considered at all events as a relative cure."

Simpson mentions the transformation of the uterus, in long-standing cases of "chronic inflammation," into a substance resembling bone.

That these are the usual final terminations of the disease, and that they usually entail sterility, or a tendency to frequent abortions, is well known.

To show that such cases begin from having been preceded by the proliferation of connective tissue incident to the gradual atrophy that occurs from prolonged celibacy, we remark:

First. That they only or chiefly occur *after the female has borne a child.* Now if the uterus were perfectly normal before pregnancy why should the occurrence of gestation and delivery —the natural functions of the uterus—generate disease? Is it common for organs to become diseased by the simple performance of their natural office? Certainly not. And we can only account for the occurrence of such postpartal disorders by admitting the existence of previous structural change—the change

[1] Ziemssen, vol. v., p. 106.

of partial latent atrophy, which had in part adapted the organ to total idleness, and in so far rendered it unadapted to the reproductive function.

Second. The disease ("areolar hyperplasia") is less frequent in females who become pregnant early in life than in those who only do so after having passed many years of celibacy, or who, after having borne one or more children early, become widows, and remain sterile for a series of years before again bearing children. The woman who, very often correctly, dates all her uterine ills from labor, when she was comparatively well before marriage, is one in whom the atrophy of celibacy was gradually progressing, when the sudden change of environment (marriage) entirely changed the order of things, and necessitated a reconstruction of the uterus in order to fit it for reproduction. Such sudden vacillations of environment are always more or less disastrous.

Third. Partial atrophy, prior to pregnancy, *leads to subinvolution after delivery.* Under the stimulus of gestation the new growth of proliferating connective tissue (which, before impregnation, had already supplanted some of the muscles and vessels, and welded together the fibres of the former so as to impair their function and development) itself undergoes a much more rapid growth, just as do the tissues composing fibroid tumors under the same condition; so that while the remaining normal tissues of the womb are undergoing their exalted evolution, the proliferating connective tissue does the same. Thus the uterus loses some of its elasticity, and its muscular walls, stiffened by the parenchymatous layers of connective tissue, cannot perform their function thoroughly well, hence follows, after delivery, *imperfect contraction of the uterus,* with consequent hyperæmia, subinvolution, and still additional productions of connective tissue. Such a case is then recognized as "induration," "chronic metritis," or "areolar hyperplasia," due to pregnancy, when in reality it is but an exaggerated phase of a previously existing structural change.

Should pregnancy again occur, such a uterus would be less than ever fitted to perform the reproductive office, hence there would arise the greater liability to abortions; or, if the gestation went to term, the imperfect contraction of the uterus after labor would be further manifested by the occurrence of "*after-*

pains," and to which, for the reasons stated, *primipara are less subject* than multipara. In other words, the "areolar hyperplasia" of Dr. Thomas arises as follows: During prolonged celibacy, and consequent prolonged menstrual congestion, a moderate amount of connective tissue begins in the uterus as a formative tissue—a new growth; during a succeeding pregnancy this new growth proceeds rapidly *pari passu* with uterine development; after delivery (owing to imperfect uterine contraction, subinvolution, and increased congestion) the new growth of connective tissue proceeds even more rapidly than ever, and the womb is now recognized as an enlarged, indurated organ, while the usual symptoms of uterine disease become well marked.

In the case of a virgin uterus, the environment of celibacy remains constant, the organ is slowly, and without much inconvenience to the female, wending the even tenor of its way towards a structural atrophy that is naturally designed, in the end, to adjust the organ to its total want of function; but when this has been progressing for years, the sudden change of environment that entails reproduction is most unfortunate as regards the structure of the uterus; under such circumstances the female not unfrequently becomes a decided invalid, and since she was comparatively well while a virgin, now attributes all of her troubles to marriage, and in this *she is right*.

It may be asked if every woman who lives single for a considerable time becomes so unadapted to procreation as that the performance of that function induces disease, why is it that the occurrence of areolar hyperplasia and its attendant ills is not more frequent? I answer that many circumstances (besides time) modify the *extent* to which the latent atrophy of celibacy has progressed, and so great are the recuperative powers of Nature, that, amid all the confusion of organic changes that take place, the organ can still often so far readapt itself to its changes of environment as that the female gets along well enough to escape the clutches of the gynecologist. If, however, I am correct in my explanation of subinvolution and "hyperplasia" after delivery, its lack of frequency cannot be alleged against me. "Arrest of involution," says Dr. Thomas,[1] "of the puerperal uterus is an occurrence of *very great* frequency. It constitutes the *chief cause* of *all* chronic uterine disorders, and

[1] Dis. of Women, 4th ed., p. 264. The italics are mine.

for this reason its importance cannot be over-estimated. Until this subject receives the attention which it deserves, the present confusion as to the causes, pathology, and general features of chronic metritis, which helps to weaken uterine pathology, must continue."

Fourth. During the prolonged functional inactivity that attends protracted celibacy, it can hardly be otherwise than that the folds of serous membrane (naturally designed to allow a free mobility of the uterus during its enlargement while pregnant, and its perfect contraction after delivery) lose something of their normal structure, becoming to a certain extent fibrous and adherent to each other, just as do the pleural surfaces of a sedentary consumptive who does not fully expand his lung, and the parts about an articulation (destined to secure its free mobility) when the joint is kept for a long time at rest. So-called "false adhesions" from development of fibro-connective tissue is one of the common phenomena in serous membranes belonging to organs undergoing atrophy from want of function. That such perimetric adhesions (in the case of the uterus) occur in celibate females as a latent adaptive growth, is perhaps not yet proven by anatomical evidence; but that such adhesions are frequently found after death, when they were least suspected to exist during life, is unquestionable. It seems highly improbable that serous surfaces naturally designed for extensive mobility, when left in contact and at rest for a period of ten years (from fourteen (puberty) to twenty-four for instance), should *not* become more or less adherent to each other. It is not impossible that the abdominal pains experienced during the later months of pregnancy by (especially) primiparous women, and which have hitherto been attributed exclusively to the resistance which the abdominal walls offer to distention, may be found hereafter to be due, in part at least, to the stretching or separation of peritoneal adhesions that have occurred latently as the result of prolonged functional inactivity. Should such adhesions be found hereafter to exist, it would sustain the views I have presented as to the origin of uterine hyperplasia, for, as Dr. Barnes tells us, "it may be laid down as an aphorism that whenever the mobility of the uterus is arrested, whether the cause be external or internal, a degree of hyperplasia is the

result. Thus, as in the case just mentioned [1] of perimetric adhesions, *imperfect involution* and a process of slow infarction follows." (Barnes on Women, p. 408.)

Fifth. The theory I have proposed implies that the reproductive organs of the celibate female have in some degree departed from the typical line of health, and that in truth every such celibate woman is consequently more or less a "patient."

However distasteful this admission may be, the question, "Is it a fact?" must be considered. Now, laying aside all preconceived prejudices, let us ask: Is such a woman perfectly well, or does she have *symptoms?* We answer: She is the victim of *uterine hemorrhage* once a month, and to show that this bleeding is accompanied by unpleasant symptoms (all of us know it, but I quote from the latest authority (Schroeder in Ziemssen's Cyclop., v. x., p. 326) as follows) the *italics* being mine:

"Menstruation, *even when running a perfectly normal course*, has a disturbing influence upon the general condition. The women are slightly irritable. Disturbances of the circulatory apparatus and of the digestive organs, and particularly of the nervous system, are quite commonly developed; also slight dragging pains in the back and loins may be present without pathological changes in the uterus.[2] It may be regarded as highly characteristic that the women themselves say 'they are *unwell*.'"

Dr. Schroeder follows this paragraph with the usual orthodox but inconsistent statement that "all such symptoms *can* (only) *be considered as physiological* as long as they are present to only a slight degree and do not lead to more serious disturbances of the system at large. In *many* persons, however, they lead to the most serious disturbances in bodily and mental condition," etc. So then: to be ill *a little*, is to be well; but to be a *little more so*, is disease. This is indeed strange logic. Who will draw the line which determines whether the patient is "well" or "unwell"?

In presenting in his work a chapter on "*Normal Menstrua-*

[1] He is here referring to perimetric inflammation during childbed, still adhering, probably, to the idea that such adhesions are *always* of inflammatory origin, when in truth they are growths of an abnormal kind that become themselves the seat of inflammation.

[2] I do not, but should like to know, what are Prof. Schroeder's grounds for this statement.

tion," preliminary to treating of menstrual disorders, Prof. Schroeder acknowledges that he is abandoning the plan "of considering the diseases of the separate organs *on a basis of pathological anatomy*," which he had till then adhered to, and goes on to state that "we must not fail to recognize the fact, that in the gynecological manual of the future, menstruation and its derangements may no longer find a place, for menstruation itself is to be treated of in lectures on physiology." I, however, affirm exactly the opposite, namely, that in the physiological manual of the future menstruation and its derangements will not find a place, but will be treated of in works on gynecology, when the missing link of "*pathological anatomy*" in the menstruating uterus will have been made out, and when, consequently, the apology of Prof. Schroeder will be seen to have been supererogatory. It is difficult to see how any process whose description requires the enumeration of "*symptoms*," can be consistently embraced in works on physiology; and equally so to understand why, if menstruation is a perfectly normal phenomenon, it should be so elaborately considered in almost every work on uterine pathology.

Sixth. The mucous membrane of the uterus during the menstrual process *undergoes fatty degeneration.* In corroboration of this fact, I need only refer to the elaborate paper of Dr. Engelmann (see this journal for May, 1875, p. 40), in which he states that the fatty degeneration "involves not only the cells of the interglandular tissue, but also the blood-vessels and the glandular and surface epithelia. That these structural changes take place in the tissue coincident with the menstrual discharge, is unquestionably proved." Already, therefore, the light of a better understanding of the *pathology* of menstruation begins to dawn upon us—an understanding, too, be it noted, that *is* based upon "*structural changes.*" But it remains to be decided whether these structural changes are purely physiological or whether they are pathological, whether natural or *un*natural.

In his chapter on the "*Conditions of Involution*" (foremost among which is mentioned fatty degeneration) Rindfleisch [1] determines for them the general character, "that a gradual transformation and final destruction of the normal form goes hand

[1] Text-book of Path. Histology, Kloman's Trans., 1872, pp. 32 and 33.

in hand in them with a parallel *decrease and final extinction of the normal function of the parts.*" This, of course, is generally admitted. We witness it very constantly in the decline of function and nutrition attending senescence—in the atrophy of old age. The same "decrease" and "final extinction" of function occurs at each menstrual period in the uterine mucous membrane. But can it be that this is the designed typical career—the natural functional and structural destiny of a membrane so highly elaborated in construction and so amply supplied with blood as is that of the uterus? The atrophy of senescence occurs from *diminution of blood;* hence, it is seen first and most prominently in the cornea and cartilaginous organs; in the uterine mucous membrane degeneration occurs at the very time when the *supply of blood to the part is most plentiful.* Again, if successive monthly renewals and degenerations of the uterine mucous membrane are in conformity with the *natural* design of its formation, let us ask, what good purpose in the organism does it accomplish? Of what *use* is this process? And if it is perfectly normal for this formation and involution of mucous tissue to go on for five or ten years after puberty, why not also for twenty or thirty years, or even to the menopause? Under such circumstances will it be said that the organ has performed the function for which it was naturally designed? Evidently not. The normal office of the mucous lining of the womb is its well-known participation (after impregnation) in the process of procreation. This is the only function for which it was naturally designed, and in the absence of which there is absolutely no other normal functional exercise for it to perform. Between the ovulatory periods the mucous membrane undergoes a normal hypertrophy preparatory to receiving an impregnated germ, and we know well enough that under the physiological stimulus of impregnation it is destined to undergo a still higher development into decidua vera, reflexa, etc., in accord with the designs of nature; but, impregnation not taking place, involution occurs in the membrane by fatty degeneration, just as it does in the muscular walls of the womb after delivery; with, however, the very manifest difference, that in the latter case it begins at the *end* of a natural process, the degenerating muscles having *finished the reproductive function for which they had been so highly developed;* while in the former

the fatty degeneration takes place nearer the *beginning* of the normal reproductive function and *before the mucous membrane has at all accomplished the office for which it had been so highly developed.* Nature will not support the nutrition of a useless organ; in the physiological offices of the system she will allow no sinecures; hence we always find that organs performing no useful purpose become prone to disease, degeneration, and decay. It was in obedience to this principle that the learned president of the Clinical Society of London (Sir William W. Gull) remarked in one of his addresses before that body: "*Were I to make a man, I do not think I would put tonsils in him;*" and hinted that the extirpation of superfluous organs was "a grand prospect for the surgeons of the future."[1] Exactly on the same principle might I venture a somewhat analogous statement, to wit: *Were I to make an "old maid," I do not think I would put a uterus and ovaries in her.*

Under this head I may add, that the natural separation of the mucous membrane of the uterus that takes place at the end of pregnancy differs in a very important particular from that which occurs at the menstrual period, namely, in the circumstance that at "term" the mucous membrane has so slight or insecure a connection with the uterine walls that it can be separated without such violence as to cause hemorrhage; while at the menstrual crisis its adhesion is so exceedingly intimate and with-al vascular, that it cannot be separated without the opening of blood-vessels by fatty degeneration and consequent bleeding. The provision therefore which exists for the natural separation at term does not attend the *un*-natural separation that occurs at the catamenial periods.

Seventh. Among the recognized exciting causes of "areolar hyperplasia" must be mentioned prolapsus and other displacements of the uterus. And we note here that such displacements, as a rule, are less frequent in virgins, more so in mothers. To say that they occur in the latter from the reproductive system of the female having become, by protracted celibacy, unadapted to childbearing, would be only repeating what has already been said. To explain further, therefore, we must remember that the vagina undergoes the same phases of increased

[1] "*Popular Science Monthly*," vol. i., No. 1, p. 122, quoting from "*Brit. Med. Jour.*"

development and involution as does the uterus. During each catamenial period it is congested; then occurs *some* hyperplasia of connective tissue, supplanting the normal muscular structure; pregnancy occurring, the muscles together with the new growth of connective tissue both undergo increased development. After delivery there occur imperfect contraction, subinvolution, and further areolar hyperplasia of the vaginal walls exactly as before explained in regard to the womb. In this way the physical tone and muscular contractility of the vagina is permanently impaired, so that it cannot contribute its usual support to the womb sufficiently to keep it in place, even if it (the uterus) were of normal size and weight, much less after it has increased in both particulars from coincident uterine hyperplasia.

Finally, it is not improbable that the other supports of the womb,—the ligaments, perineum, and muscles composing the pelvic floor, which are naturally adapted to undergo certain structural modifications coincident with pregnancy when the latter takes place at the proper age, may be less disposed to go through those cyclical oscillations of development and involution, when they have become habituated to structural stability from long years of barrenness: for them, then, "the die is cast," and the supple age of easy modificability is forever gone.

In conclusion, it may be added that the orthodox treatment of areolar hyperplasia by the highest authorities is exactly that which the views I have proposed as regards the pathology of the disease would have suggested. Many of our best remedies are but imitations of Nature, and the best philosophy of the physician is to find out what Nature is attempting to accomplish and assist her.

Areolar hyperplasia begins, as I have said, by the uterus attempting to modify its structure, so that it may be adapted to sterility and celibacy. To secure the completion of the process it is necessary for the *environment of celibacy to remain constant*, for the sudden call for function upon an organ partially structurally adapted to total idleness, leads to congestion and unnaturally rapid connective-tissue hyperplasia.

This, the physician imitates in his treatment by recommending abstinence from *coitu* and the avoidance of pregnancy. Nature allows the disintegration of the useless mucous mem-

brane by fatty degeneration, leaving a film of cicatricial fibro-plastic tissue in its place: the physician burns out the mucous membrane by nitric acid, and attempts to do it so deeply as to leave a thicker layer of fibro-plastic tissues, so as to prevent the re-formation of any new mucous membrane, and thus reduce the organ to such a state that absolute sterility is abruptly as certain as Nature in the course of years would also have made it.

Nature relieves the periodical congestions that occur during the excitement of ovulation, by the salutary bleeding of the catamenia; the physician relieves any discoverable congestions that may have arisen from the excitement of *coitu* or other forbidden imprudences, by the salutary bleedings that follow scarification, leech-bites, etc.

The coincident discharges of vaginal and uterine mucus which also lessen congestion and keep the development of areolar hyperplasia within the bounds of typical rapidity, are again imitated by the discharges produced by the glycerine plug, and cantharidal collodion applied to the cervix by the gynecologist. The same sort of drain from the uterus also is promoted, and the excessively rapid growth of connective tissue restrained, by clipping off a piece of the cervix with the scissors or galvano-cautery. In this way the remainder of the organ will undergo a diminution in size, much in the same manner (says Dr. Thomas)[1] as enlarged tonsils will do when their faces have been shaved off. Thus the useless tonsil referred to by Dr. Gull, and the useless uterus of the celibate female, again meet, and rather unexpectedly, as regards their surgical treatment.

But whatever the treatment of areolar hyperplasia, it is well known to be very difficult of cure, and the best termination that can happen in cases to any great degree advanced is premature atrophy of the uterus and consequent absolute future sterility—a termination that is often sought to be attained by the gynecologist in vain, until Nature achieves it at last, by the redoubled tendency to atrophy that occurs after the menopause.

Perhaps the time may come in the exaggerated civilization of the future when females, to escape the ills of maternity and the menstrual aches of celibacy will submit to "normal ovariotomy" (which indeed has already been done), or to ligation of the uterine and ovarian arteries, but the aspirations of the

[1] Diseases of Women, 307–8, 4th edit.

gynecologist do not properly lie in this direction. His mission is to study the *natural functional career* of the reproductive organs, and recommend such a course of life to the female as will maintain them in the exercise of their natural functions only. At present both physiologists and gynecologists are profoundly ignorant as regards many important questions bearing upon this subject. None perhaps has been more carelessly treated than that which refers to the marriageable age of the young female and the frequency with which the act of procreation (and indeed of *coitu*) should be repeated. At first sight the theory I have suggested would seem to indicate that procreation beginning with puberty ought to be repeated every year or two until the menopause, so as to forestall any intermediate recurrence of the abnormity menstruation, which would necessitate, *apparently*, the bearing of twenty or thirty children. But this conclusion is too hastily drawn. It is questionable whether the so-called menopause would not occur much earlier than it does, if the reproductive powers of the female were exercised to their full, but still normal extent. Thus the atrophy of the uterus which usually occurs after the climacteric, might under those circumstances occur earlier, and coincidently the processes of menstruation and ovulation might cease. In fact Dr. Barnes tells us that such cases have already been observed in "younger persons, in consequence of the rapid succession of labors."[1] But the whole subject needs a thorough reconsideration, of which it is hoped the papers on my "New Basis" may perhaps be a salutary beginning.

[1] Diseases of Women, p. 402.