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DYSMENORRHŒA.

BY

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Read before the British Gynæcological Society.

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DYSMENORRHŒA.

Few subjects have given rise to a greater amount of discussion, and have evolved a greater diversity of theories, than that of menstruation. When one attempts to wade through the plethora of literature which treats of its cause, its source, its effects on the individual, &c., he will speedily ascertain that he has set himself a task of no mean difficulty, and when his labour is completed he will find that he is probably more at sea than when he commenced his more extended investigations. I confess I hardly thought it possible such a variety of opinion could exist upon any subject, and I fear it is nothing short of presumption on my part to attempt to speak on a theme which has exercised the thought and ingenuity of so many able and eminent members of our profession, the result, however, to my mind being that as yet no definite and generally accepted theory has been established. My object, then, to-night, is to offer for your consideration, if not your acceptance, views which have for years been becoming more and more established in my mind, and which have for long dictated the line of treatment adopted by me. As we cannot disassociate the catamenia from dysmenorrhœa, it will be desirable, at all events, to devote a few minutes to the physiology as opposed to the pathology of menstruation. All seem

agreed that the discharge of blood which constitutes the menstrual flow proceeds from the lining membrane of the uterus. I have employed the term *lining* membrane in preference to that of the generally accepted *mucous* membrane, as I am very much inclined to think, with Emmet, that it is not strictly speaking a *mucous* membrane. It neither harmonizes in its structure nor its behaviour with any mucous membrane. It seems to me to partake more of the character of the granulating surface of a healing sore than any other structure, and, like healthy granulations, it partakes very much of the nature of a soil on which epithelium grows, instead of it becoming an essential part of the tissue, as is the case with the epithelium covering a mucous membrane. Mark me, I do not aver that it is composed of granulations, but I do hold that it resembles this growth in a great many important points. For example, the lining membrane of the uterus, like granulations, is very vascular and spongy, and therefore bleeds with little provocation. It is easily destroyed and readily renewed. Its vitality is less than that of a mucous membrane, and in this it again resembles granulations. In fact, there is hardly a particular in which the two growths do not agree. When maturation of the Graafian vesicle occurs, hyperæmia of the uterus takes place as a physiological sequence, being a result of the increased activity in the ovary. This hyperæmia stimulates the cell life of the lining membrane of the organ, and a great proliferation of its cells is the result. These naturally are renewed from within outwards, and in consequence the super-

ficial layer is shed during the prevalence of the hyperæmia. With this desquamation of the outer layer of the membrane the capillaries are ruptured, and hence the flow proceeds. The hæmorrhage in its turn acts as a depleting agent, thus relieving the congestion, which therefore gradually subsides, and the period passes away. When this epoch of congestion has terminated and the equilibrium of the normal blood supply is again established, we find the lining membrane quite renewed, and capable of becoming the nidus of an impregnated ovum. Now, as impregnation usually takes place shortly after the cessation of the menses, it is very appropriate that the ovum should be received into a newly matured and therefore more vigorous receptacle. We must not forget, however, to notice the important part the utricular glands play in this monthly *rôle*. When the superficial layer of membrane is exfoliated, naturally the epithelial covering goes with it, but this is speedily renewed, when the process is complete, by prolongation from the epithelial lining of these innumerable glandular apertures. An analogy to this can frequently be demonstrated when a superficial sore has been formed, and where the granulating surface heals over with amazing rapidity, each little cutaneous gland giving rise to an island of epithelium, which speedily coalesces with its neighbours, and completes the healing process. If we carry the analogy a little farther, and enter just within the regions of pathology, we will see in how many instances the two surfaces resemble one another. It is a curious fact that epithelium will not

extend its growth up an incline. It must have a level surface to develop itself upon. We observe this in sores where the granulations are too prolific. Who ever saw what is commonly known as proud flesh covered with epithelium? No, the redundant growth must either atrophy or be destroyed before we get this result. So with what we designate fungosities of the uterus, which are neither more nor less than an exuberant growth of the lining membrane, and which, moreover, are not covered with epithelium; hence a constant oozing of blood emanates from them. As soon as these are destroyed, the bleeding ceases, the lining membrane becomes covered with epithelium, and the bleeding orifices close. I hope, gentlemen, you will pardon me dwelling so long on one view of the natural history of this membrane, but as it appears to me to be nearly correct, my conclusions are naturally based upon it, therefore I have ventured to give you the result of my observations as briefly as possible. I have done so because I may frequently have to refer to this hypothesis in the course of my further remarks.

We must bear in mind that the uterine canal in health is not to any extent a secreting cavity; I refer, of course, to that portion above the internal os. As we all know, the cervical canal secretes a glairy mucus peculiar to itself. We then have the fundus and body in the healthy condition quite inactive (if unimpregnated), except at the monthly epochs. If, therefore, a discharge is observed oozing from the os which differs in character from the cervical secretion, it would suggest the presence of disease, but as any discharge

which is not really cervical is apt sooner or later to become purulent, there can then be no difficulty in indicating its source.

If normal menstruation is simply a disintegration of an old stratum of cells which have been thrown off because a more recent and therefore a stronger substratum has taken their place, the old layer degenerating in consequence of their cell-growth being over-stimulated by the monthly hyperæmia, we can understand how this monthly wave produces the menstrual flow in health. So far, however, I have not really touched upon the subject I came to discuss, and before I do so I must still further crave your indulgence, as, with your permission, I would like to say a few words about the discharge itself.

The menstrual fluid, I think, on all hands is acknowledged to consist of blood mixed with the *débris* of the exfoliated lining membrane. Now it is a well-established fact that blood in contact with a healthy membrane retains its ability to remain fluid, but if it comes in contact with a foreign body, this imparts to the blood a newly-acquired property, which enables it to separate into two distinct substances, each of which differs from blood itself, viz. clot or fibrin and liquor sanguinis or serum. This no doubt is the result of a vital action on the part of the blood, induced by contact with a foreign body. Were it not for this endowment, and the capability of the clot to become organized when it remains in contact with vascular lining tissue for a length of time, hæmorrhages would not spontaneously cease as they tend to do. Now

we know that a tissue, whose vitality is impaired by inflammation, acts on blood as an irritant and induces coagulation. Likewise, if the vitality of a tissue is reduced by an injury this catalytic effect is produced by the injured part. We can perceive, then, so long as the lining membrane of the uterus remains healthy, the blood will tend to remain fluid and thus no pain will result from the actual evacuation of the discharge, because it will ooze away without difficulty; that is to say if the os is patent. We cannot, of course, designate the periodic engorgement of the uterine tissue as disease, when it *completely* disappears with the cessation of the activity in the ovary, any more than we can call the condition of the stomachic mucous membrane, which induces the sensation of hunger, congestion. If, however, the loading of the blood-vessels goes beyond the physiological stage, and inflammation more or less active is the result, an irritant of greater or lesser intensity presents itself, which according to its intensity will act more or less energetically in inducing coagulation. The clots which result will therefore require an effort on the part of the uterus to expel them, and, moreover, the contraction of the walls of the organ will produce a degree of pain relative to the degree of hypersensitiveness which the amount of inflammation entails. The pain, then, is not due entirely to the circumstance that, instead of a fluid flowing freely away, a clot, or a series of clots, have to be expelled by a number of spasmodic efforts of the muscular walls, but also to the fact that these are in a condition of hyperæsthesia, their nerve filaments

being already in a highly sensitive condition consequent on their compression due to the hyperplasia. If, however, the clots were not present, there would be comparatively little aggravation of pain at the molimen. We have this demonstrated in cases where there is a copious catarrhal discharge during the interval of the menses, but where the catamenial flow is alone accompanied by pain.

We therefore are led to look upon a tissue weakened by inflammation as a foreign body would be viewed, in so far as it affects the blood. Yet this may gradate so gently and imperceptibly from the line which indicates health to that which points to disease in its most active and potent form, as to make it quite impossible to recognize a line of demarcation. It is, however, in the lesser degrees of inflammation that the discharge is most copious, and therefore the clots are both larger and more numerous than when the inflammation is more acute, but the intensity of the pain does not diminish in a like ratio. We may have a condition of the uterine walls which is certainly due to a form of congestion but which does not add *per se* to the hypersensitiveness of the organ. The result of this minor form of inflammation is hyperplasia accompanied by an œdematous condition of the tissues, arising from a partially obstructed venous return-flow. The result of this is naturally a feeling of weight and bearing down in the pelvis, and there is superadded an abundant catarrh, while the menstrual epoch is marked by a copious and clotted discharge often extending for days beyond the normal period. There is not always

pain, however, because the uterus is flabby and patulous, and therefore the discharge escapes rapidly. On the other hand, however, there is frequently very intense suffering, and this occurs notwithstanding the patulous condition of the canal and its normal direction. The question naturally arises, how are we to account for the different symptoms observed? It is obvious, I think, that in the one case we have a simple œdema consequent on an interference with the circulation in the veins, whereas in the other we have the hyperplasia due to an inflammatory condition of an active type, where the effused lymph is plastic and which therefore renders the tissues denser than when mere effusion occurs. Again, when inflammation exists the clots are more compact than when they are induced by contact with a merely weakened tissue. Moreover, the blood is more coagulable in the one case than in the other, and therefore a greater effort is required to expel these clots. Then we must not forget that the uterine walls are very much more sensitive in the latter case than in the former. It may be asked, how does this condition of things not tend to spontaneous cure, or why does it not proceed to a more intense degree of inflammation? for we frequently come across cases which have continued for years in very much the same condition locally, though the strength of the patient has gradually been waning. The explanation is that the monthly depletion acts as nature's antiphlogistic, and the excessive catarrh also tends to relieve the overloaded tissues. On the other hand, we should not forget that the anatomical position of the uterus tends to promote

any unhealthy condition of this nature, suspended as it is in the pelvis. If the tone of the muscular fibres is diminished, we can fully understand how the helplessness of the veins is increased, for while the arteries continue to pour blood into the organ, the veins which are destitute of valves become engorged and keep up the œdema in consequence of effusion of serum through their walls constantly going on. When this obtains it is of course impossible for the lining membrane of the canal to retain its health, as the cell-growth is overstimulated to such a degree that maturation of the cells never takes place, and they are cast off in large quantities, passing away along with a serous exudation, either as pus or a muco-purulent discharge, which is very copious and not unfrequently has imparted to it a fœtid odour. We thus have a degenerative lining membrane taking the place of the healthy, the consequence being that it readily breaks down at the monthly period and bleeds profusely, not only while the activity of the ovary continues (for the overloaded condition of the arteries persists beyond this), but for a time afterwards, varying from a few days to the whole period which ought to be that of cessation. When the discharge is so persistent as to give rise to menorrhagia or metrorrhagia, as a rule there is not much pain nor coagula either, for the reason that the bleeding relieves the congestion and the flow is so free that the blood does not remain long enough in the canal to permit of coagulation taking place there. If, however, the inflammation is more intense than this, the hæmorrhage is less, and clots within the uterus are more numerous.

This arises from the irritant being more decided, and therefore blood-clots are more readily formed at the orifice of the ruptured capillaries. Nay, more, clots may and actually do form within the vessels themselves and thus arrest the hæmorrhage, which otherwise would relieve the congested tissues, but this not taking place effusion of lymph goes on into the parenchyma, becomes organized, and produces rigidity and tension of the uterine walls.

Such a condition of the womb is associated with acute hyperæsthesia from the compression which the nerve filaments endure, and consequently every movement of the body, and to a much greater degree every contraction of the uterine fibres, are accompanied by intense suffering. This pain, however, is not confined to the actual seat of the irritation, but radiates throughout the whole area supplied by the trunks of the nerves, of which these are the peripheries. Moreover, this congestion reacts on the ovaries through excess of blood pressure being thrown upon them by the surcharged ovarian veins, and through these the whole pelvic vascular system is disorganized, either by direct venous continuity or through the vasomotor nervous apparatus. When this condition of the uterus exists we have the menstrual discharge diminished in quantity, and that being exposed to a more intense irritant coagulates more rapidly, and is retained, because of its scantiness, for a longer period. It therefore necessitates a greater effort for its expulsion, and, the neurasthenia of the organ being acute, the pain bears a close relation to the size of the os. This, however, cannot properly

be designated obstructive dysmenorrhœa, as many have named it, because, if the inflammation did not exist, in the majority of cases the os is quite patent enough to permit the discharge *of blood* to escape without pain. I will illustrate this by detailing a case which interested me very much. Mrs. K., *æt.* 28, consulted me in May, 1883. She was married at 24; menstruated for the first time at 16, and continued to do so regularly and without pain till she was 20, after which she began to suffer at each catamenial period. This gradually increased in severity, and as it did so the discharge became more and more clotted, till eventually a complete cast of the uterine cavity was thrown off at each period. This was always preceded by severe suffering, accompanied by a red, watery discharge, which continued for two days, when the pain would come to a climax and the cast be expelled. With this the pain ceased, though a few smaller clots would come away with some fluid discharge for another day, but with very little inconvenience. There was dyspareunia, also a copious muco-purulent discharge. Vaginal examination disclosed hyperplasia. The uterus was acutely sensitive to touch. When the sound was passed, the internal os was found to be spasmodically stenotic, and the canal beyond very painful when the sound was in contact with the lining membrane. The general health was very indifferent, and there was great nervous prostration with despondency. The bowels were constipated, and there was polyuria.

General treatment consisted in the administration of an enema every second day, and a pill containing

2½ grs. valerianate of zinc and 2 grs. extract of conium was ordered to be taken forenoon and afternoon. Local treatment: I dilated the internal os twice a week, and applied to the whole area of the canal a saturated solution of iodine with carbolic acid, and a tampon soaked in glycerine of alum and boracic acid was introduced into the vagina and placed in contact with the uterus. This treatment was continued for four months, at the end of which time the hyperplasia was reduced and the metritis removed, when the menses were discharged free from clots and without pain. She shortly afterwards became pregnant, and was delivered at the full time of a well-nourished child. She called on me only a few days ago to report herself as being in perfect health.

This is, perhaps, the most perfect example of dysmenorrhœa accompanied by clotted discharge and due to endometritis that I have seen, though I hold it is only typical of the effect of an inflamed surface upon blood which is in contact with it. On looking at the specimens it will be observed that their short retention in the uterine cavity has resulted in their taking on a kind of fibrous formation, due to their having become to a certain extent partially organized. I have no doubt that such cases are frequently confounded with membranous dysmenorrhœa, whereas the casts, or rather masks, which are pathognomonic of this disorder, are the results of a more intense form of dysmenorrhœa than that which produces these coagula. If the inflammation is still more intense, plastic lymph is effused into the superficial layer of the membrane, whose vitality has been destroyed by the same agency, and it

is shed *en masse*, and expelled as a complete mask of the cavity, the exfoliated cellular structure having been rendered coherent by its being impregnated with the effused lymph, just as we have a slough thrown off when inflammation attacks the granulations of a healing sore. Again in croupous dysmenorrhœa we have this plastic lymph partly organized, and retaining the contour of the cavity when it has been deposited on the surface of the lining membrane.

It is worthy of observation that the more severe the inflammatory symptoms are, and the more intense the dysmenorrhœa is, the discharge as a rule is in a like proportion scantier, and in the ratio of the pain losing in severity the discharge becomes more copious. This is only to be expected when we take into account that the more acute the inflammation, the farther removed it becomes from healthy structure, and consequently exerts its catalytic power more powerfully in producing coagulation, and therefore lessens the tendency of the ruptured vessels to bleed. Take a case of slight congestion of the integument as an example illustrating what I mean to demonstrate; the congestion produced by cupping, say; when an incision is made into it what is the result? a copious flow of blood of course. How different is the effect when an intensely inflamed part of the integument is incised, a carbuncle for example. Here effusion has taken place into the cellular tissue consequent upon the occlusion of the capillaries by clots having formed within them, this being due to the blood having been acted upon by the irritant destroying either partially or completely the vitality of their walls. We

will have in this instance comparatively little hæmorrhage, and, moreover, we will be able to note how much more rapidly the effused blood coagulates than when it flows from a wound when the congestion is less intense, thus accounting for the more rapid cessation of the bleeding in the one case than in the other. It is not unnatural to conclude then that the same argument holds good in uterine hæmorrhages. But it may be asked, how does it happen that the cast in fibrous dysmenorrhœa is firm and coherent, while a slough is soft and flabby? Well, in the one example, that of the cast, it has not been exposed to the influences of decomposition, the uterine canal being aseptic, and in consequence it is not at once deprived of all its vitality, and therefore becomes slightly organized by remaining in contact with vital tissue; whereas in the slough, before it is actually dead, it becomes a prey to the germs of putrefaction which surround it, and thus speedily breaks down by decomposition. We perceive then that, when the inflammation is less intense, we have less tendency to occlusion of the uterine capillaries than when it is severe, and consequently there is a freer discharge with which there is comparatively little pain, but as a result of the continued hyperæmia a hyperplasic and flabby condition of the uterus takes place, and this want of tonicity of the walls favours the liability to the occurrence of flexions. When these become established, however, although they may not actually act mechanically in obstructing the flow, a much more painful array of symptoms is to be dreaded, for although the calibre of the canal at the seat of

flexion may not be interfered with sufficiently to obstruct the exit of the discharge, yet, in consequence of the bend interfering with the venous return flow, the body and fundus take on a more decided inflammatory condition and so become much more sensitive than before, this being due to the walls becoming more rigid and the nerve filaments being more compressed. When the menstrual period comes round, therefore, this condition is still further aggravated by any contractile effort, being accompanied by increased pressure for the time being, and consequently by more intense suffering. The transition from flaccidity to rigidity of the uterine walls in flexions is of course gradual, and it is not necessary that rigidity should result at all, for if the flexion is recognized at an early stage a vaginal pessary will prove of immense service and a speedy cure will result if the metritis is treated *pari passu*. If, however, the latter condition has been established, it would be highly dangerous to force the uterus into its normal position, and retain it there, without first taking measures to reduce the inflammatory condition of the walls by tampons and intra-uterine medication. Let me give details of a case in point, which I copy from my notes. Miss L. consulted me on April 28th last. She was suffering from acute retroflexion of the uterus and was wearing a Hodge's pessary, which she said had never given her any relief, but on the contrary aggravated her symptoms. A year and a half ago she had sustained a severe fall while playing lawn tennis, and from that date her sufferings began. She had been under treatment for some time by the family attendant

without deriving any benefit. She was then taken by him to a specialist, who took her in charge and treated her in the orthodox way by means of pessaries and rest. This was continued for five months without the slightest benefit resulting, when he said he could do no more for her, and advised her to go to Edinburgh and consult an eminent gynæcologist there. The patient naturally shrank from the idea of leaving her home, and I was asked to see her. As has been stated, there was retroflexion, but arising from this there was regularly recurring most severe dysmenorrhœa and menorrhagia, which so prostrated the poor girl that the intervals between the menses were simply occupied in preparing for the next epoch, when the strength was again completely exhausted. This unsatisfactory state of matters had gone on for months, and the patient and her friends began to lose all hope of her ever being well again. She was unable to stand or walk without severe pain, and in every sense of the word she was an invalid.

On making a vaginal examination, I found the os quite patent, and exuding from it an acrid purulent discharge which had given rise to vaginitis, so much so that the mere passing of the finger along the vagina caused intense pain. The uterus, but especially the fundus, was likewise acutely sensitive to the touch. Metritis, therefore, accompanied the malposition, and this was evidently the cause of the dysmenorrhœa and menorrhagia. The metritis, on the other hand, was due to the disturbance of the circulation consequent upon the flexion. I did not attempt to pass the sound,

but contented myself with gently supporting the fundus by means of a medicated tampon, which I renewed twice a week, the object being to deplete the congested tissues and constringe the uterine walls and those of the vagina. Gradually but surely relief ensued, so much so that at the end of fourteen days I was able to pass the applicator charged with carbolized iodine into the uterine canal, and thus attack the endometritis directly, which aided very much the treatment directed to the inflammation of the parenchyma. The result was that the discharge lost its purulent character in a few weeks, and the vaginitis rapidly disappeared. A great point of advantage was thus gained, as the displaced and otherwise diseased organ could now be treated without entailing much suffering, whereas, until the excessive sensitiveness of the vagina had been removed, the local applications produced great pain. The tampon was applied bi-weekly for two months, and the endometrium was swabbed every eight or ten days with carbolized iodine. At the end of this time the dysmenorrhœa and menorrhagia were very much abated; the backache, which had been such a marked feature in the disease, was almost gone; and the patient, instead of being low-spirited and depressed, became bright and cheerful. For two months more I saw her once a week, by the end of which time all symptoms of disease had disappeared, and the position of the womb was normal. I may add that up till quite recently the patient has been under my observation once a month, but she is now so well that she is able to resume her wonted duties.

It will be seen, then, that I do not hold in much favour the views so much advocated by some, that flexions act mechanically in producing dysmenorrhœa; however, I would not like to deny that there may be *some* truth in such a theory, but it is so far removed from the main cause that prominence ought not to be given to it, for if this is done our treatment is sure to be productive of but little relief at the best. I have frequently seen cases of severe dysmenorrhœa when the direction of the canal was normal and quite patulous, so much so that the sound passed with the greatest ease. In these cases, however, metritis was present, and when this was removed the menstruation became painless, and the general health rapidly improved. Again, I have not unfrequently seen cases of traumatic retroflexion from a fall result in severe metrorrhagia without the slightest pain accompanying it, and certainly where no evidence of obstruction was present. Here you have an unloading of the overcharged veins, the hæmorrhage keeping down inflammatory action by not permitting congestion to supervene. In these cases, as soon as the malposition was rectified, and the uterus retained in its normal position, the hæmorrhage ceased, proving, I think conclusively, that it was the interference with the venous return flow that caused the overstrain upon these vessels. But, on the other hand, when the flexion takes place gradually in a uterus which was not healthy to begin with, the congestion becomes gradually intensified, and the blood coagulates as it is effused, so that which otherwise might be a copious flow to a great extent is checked, though really its duration is

not unfrequently prolonged as a consequence of the displacement.

The more I see of dysmenorrhœa, the more convinced do I become that the mechanical obstruction theory is weak. I cannot view stenosis, even when it exists, as a cause *per se* of this disorder. If the os is so far patent as to permit the passage of a very fine uterine probe, and the endometrium is healthy, it is quite possible for the discharge to escape without pain. Yet I can understand, if the discharge is profuse, it may flow from the lining membrane in a greater amount than it can be evacuated, and so give rise to painful uterine spasm; but if this contraction were the sole cause of the pain, we would have the pain continuing throughout the whole period of the flow, instead of which it generally subsides after the first or second day, or, in other words, when the depletion has relieved the congestion of the organ. A stenotic os, then, may, and does frequently, become a factor in producing an irritable and sensitive condition of the parts, and will eventually lead to inflammation. If this narrowing of the outlet were really the sole cause of the pain in these cases, we would be certain of a cure were it dilated or incised, but how frequently do these operations fail to give relief if measures are not likewise taken to improve the health of the organ itself. It very frequently transpires that a woman with stenosis suffers more at one time than another, and this, although the calibre of the canal remains *in statu quo*, showing, I hold, that the narrow aperture does not explain the *casus morbi*. Constipation is a notable accessory to the production

of the pain, however, and this because a loaded colon and rectum interfere not only with the pelvic circulation, but also with the tone of the system at large, inducing an overcharged state of the veins, and therefore a more copious discharge, also reducing the general tone, which develops a neuralgic condition. If this sluggish condition of the bowels is removed, in many instances the dysmenorrhœa will cease simply because the quantity of the discharge is reduced, and the general tone improved. But it is not always an easy matter to get hold of such cases in their initial stages, on account of the delicacy that naturally exists in young girls to submit to any kind of treatment for dysmenorrhœa. So much, indeed, is this the case that it has come to be looked upon as a natural consequence of menstruation, and so it is allowed to develop till it is beyond the reach of simple remedies. When, therefore, this state of matters is allowed to continue for a lengthened period, the organ, in consequence of repeated spasmodic efforts and continuous loss of tone, becomes the seat of disease, so that we have not only a hypersensitive, but an inflamed uterus to deal with. We then have a clotted discharge, and such a prolongation of the period that it merits the title of menorrhagia. It is because of this free discharge, however, that the pain ceases when the flow begins to be copious, from the fact which I have so often pointed out, that the overloaded vessels relieve themselves for the time being, and the congestion is in abeyance, nature acting as her own physician.

It will thus be perceived that I look upon a hypersensitive condition of the uterine walls, due to an in-

flammatory condition, as the essential cause of the majority of cases of dysmenorrhœa, and this for the following reasons:—1. A congested uterus being already hyperæmic has this condition aggravated at each molimen. 2. Because every contraction of its walls is accompanied by intense pain in consequence of their congested condition. 3. Because the inflamed tissue exerts the influence of an irritant upon the constituents of the blood, causing it to separate into clot and liquor sanguinis, the clot necessitating uterine contractions for its expulsion.

We, however, meet with many other conditions besides those mentioned which tend to set up inflammatory action in the womb, and consequent dysmenorrhœa, and perhaps one of the most common is an elongated cervix. Here we may have no narrowing of the canal, and yet it is a frequent source of mischief, and this is proved by the fact that, although the dysmenorrhœa is removed by judicious treatment, it will tend to return again, and this almost invariably, if we do not take the precaution to remove the redundant tissue. So much am I convinced of this that I have for some time absolutely refused to have anything at all to do with cases of dysmenorrhœa where this deformity exists, without I first get permission to remove what is abnormally developed, and not infrequently this has been all the treatment required.

Much, however, as I adhere to the belief that an inflamed condition of the uterine tissue is the chief factor in producing this painful affection, I do not for a moment wish it to be thought that I believe this

accounts for every case; but as during the past five years close upon 1000 cases of this disorder have been treated by me, and as I have found that most of these have been the subjects of endometritis, and it was only when this was removed that the painful symptoms disappeared, it cannot be a matter of surprise that my convictions are what I have expressed.

The cause of metritis may, and most assuredly does, proceed from a variety of sources. It may, and frequently does, arise from an atonic state of the system at large, inducing flaccidity of the uterine walls, and, moreover, interfering with the circulation in the uterus by diminishing the heart's power. If atony of the heart exists, then the uterus, from its anatomical relation to the neighbouring parts, is the first or one of the first organs to suffer, so that in the treatment of all uterine disorders it becomes of necessity a part of our plan of action to take means to restore the general health simultaneously with our efforts to relieve the local symptoms. A first duty, then, is to take particular note of the condition of the bowels, for if these be inactive we have present a common source of anæmia and bad health, in *young* women especially. Not only does a sluggish action of the colon act mechanically by interfering with the circulation in the pelvis, but in not a few instances by actually displacing the uterus. It also permits of a constant absorption of fœtid matter into the blood which destroys the health and even the vitality of the red corpuscles, thus reducing their number and quality. By this not only is the blood deteriorated, but through it the nervous system is injured,

and the whole of the functions of the organism thrown out of gear. I do not think I need remind you, gentlemen, how almost invariably this condition of things is prevalent among delicate young ladies, and how dire are the consequences; but I must confess I am surprised to observe how uniformly this important point is overlooked by medical men, and to what a small degree it is recognized as a *casus morbi.* It would seem as if they were satisfied if they order a dose of laxative medicine occasionally. But, gentlemen, this will not suffice. The bowel must be emptied thoroughly every day, or second day at least, and this can only be accomplished by a systematic and prolonged use of the enema, together with the administration of a tonic which will tend to restore the functions of the atonic muscular fibres of the intestine. It were time to a great extent wasted in the treatment of uterine disease if we neglect this important feature of the patient's condition. But to return to the subject more immediately under discussion, other ascribed causes of dysmenorrhœa must be referred to.

1. It may arise in conjunction with stenosis, but, as I have before observed, the stenosis cannot be the sole cause, or why does the pain cease when the flow has become thoroughly established? Or why do some women suffer while others do not, the outlet being of equal calibre in the various cases? Or, again, why do some suffer at one time and not at another?

2. Dysmenorrhœa may accompany a neuralgic condition of the uterine walls, and frequently does so, for well we know that a neuralgic woman always suffers

most at the catamenia, not only in the pelvic organs, but elsewhere, and it is quite natural that the activity of the uterus at that time will render it doubly liable to neuralgia then.

3. Dysmenorrhœa has been said to be due to spasm of the uterus, and has been compared to the spasm which produces asthma; and by way of argument, Dr. Matthews Duncan says: "Asthma is cured by a copious secretion of the mucous membrane, just as dysmenorrhœa is generally relieved when the menses flow freely." Now, I hold that the very reverse is the case, for it is only when the spasm in asthma ceases somewhat that the mucous membrane is *able* to secrete mucus to any extent. When the spasm is severe, the nerve centres which control the mucous secretion by reflex action are paralysed temporarily, and it is only when the irritating effect of the spasm subsides that they are able to act, when the modified irritation which remains stimulates them to free action, and a copious flow of mucus results, just as when a severe inflammation of the Schneiderian membrane occurs no mucus is secreted, but when this subsides the more intense irritant ceases to act so powerfully on the ganglionic centres, and in consequence their activity is restored and afterwards stimulated by the moderate degree of irritation which the less congested condition of the mucous membrane conveys through their afferent filaments. So that the relief in asthma when mucus is secreted freely is *not* "post hoc propter hoc," whereas the relief obtained in dysmenorrhœa when the flow is established *is* "post hoc propter hoc."

4. The obstruction theory has had many advocates, amongst whom is numbered Dr. Barnes and the lamented Dr. Marion Sims, but, unwilling as I am to differ from these veterans in the science of gynæcology, I must confess that I fail to see how fluid blood should be less able to escape without pain than the catarrhal discharge which is so copiously excreted in the intercatamenial period, nor can I understand why the pain ceases after the menstrual flow has been thoroughly established.

But, gentlemen, I came here to give my views on this important subject, and not to criticise those of others, and as I have expounded these as explicitly as the time at my disposal permits, I with deference leave them for your consideration.

OPENING ADDRESS.