Thesis for M.D.

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DYSMENORRHOEA

Menstruation, scanty or profuse, regular or irregular, if attended with so much pain as to interfere with a woman's daily work is called dysmenorrhoea.

It is of frequent occurrence, about 110 out of 1000 cases met with at a gynaecological clinique (More Madden) coming under this class. But the relative frequency will vary greatly with individual gynaecologists according as they are guided in nomenclature by symptoms or by physical signs. Far more than one in ten of the women coming to their cliniques will complain of dysmenorrhoea, but where there are gross lesions the lesion will generally classify the disease.

It is clear that dysmenorrhoea is a symptom, not a disease. Any useful subdivision of dysmenorrhoea must, therefore, be based on our knowledge of the pathological conditions with which it is associated. No doubt this is generally admitted. But if this was sufficiently kept in view there would be less inclination in some London Schools to define it as a neurosis. Our knowledge of the subject is more definite than some authors would have us believe, and warrants a much broader treatment of dysmenorrhoea than it usually receives. It ought to be treated in the same way as such a symptom as jaundice.

At the outset it may be well to state the facts known about menstruation that have a bearing on dysmenorrhoea.

The menstrual period, in a healthy woman, lasts a few days; is accompanied with little pain; and recurs about once a month. There is increased general vascular tension for some days before the periods (varicose veins become more prominent and goitres swell), and the breasts may be swollen and tender (vicarious menstruation). Locally, there is hyperaemia of the uterus and its appendages (a hernia of the ovary becomes swollen and tender); and this is followed by sanguineous discharge from the body of the uterus (removal of both tubes and both ovaries has been often known to be followed by regular menstruation).

Normally there are no clots passed of a size to attract attention.

Ovulation as a rule accompanies menstruation, but may occur at other times (examination of ovaries removed by operation showing Graafian follicles ready to burst at a considerable time before the period: pregnancy has occurred before menstruation).

It is not definitely known whether the discharge is expelled by uterine contractions or drops away in normal menstruation; but it is a matter of observation that it is expelled in gushes in many cases of painful menstruation.

The object of this paper is to enquire into the origin of the dysmenorrhoeal pain, and to suggest a classification of dysmenorrhoea which, in my limited experience has seemed clinically useful.

Normal menstruation is practically free from pain. How are we to explain the origin of the pain in dysmenorrhoea? In the first place, then, menstruation is essentially a recurrent hyperaemia of the sexual organs with haemorrhage from the uterus. And it is pertinent to enquire whether the hyperaemia and the haemorrhage have anything to do with the production of the pain in dysmenorrhoea. Any pathological conditions present are no doubt existent during the intermenstrual periods, but in the great majority of cases the pain is at the periods only (if we include in 'period' the day or two days preceding the flow). Speaking generally, there are two varieties of dysmenorrhoeal pain which have been recognised in practice for many years, and my limited experience entirely supports the contention of Dr Champneys that we can only distinguish two kinds. One occurs during the day, or two days, or even more, that precede the flow: the other comes on with or during the flow and may last to the end of the period.

Obviously pain preceding the flow by two days cannot be due to the haemorrhage, when there is no blood as yet in the cavity of the uterus. "Vedeler passed a sound during the pain in patients suffering from severe dysmenorrhoeal pain before the flow, and not a drop of blood followed, though the pains were "obstructive" in character." (Champneys' Painful Menstruation, p. 66)

Vedeler's observations quite conclusively proves, no doubt, that the mechanical or obstructive theory is not all-

sufficient. But I refer to the cases, common cases in general practice, in which young girls suffer from severe pain of a constant character, sometimes very acute, sometimes dull aching pain, coming on two days before the period and persisting right up to the period and vanishing as the flow appears. Is this pain due to the hyperaemia?

Pain due to hyperaemia ought to precede the flow, because the hyperaemia does. And pain due to local hyperaemia ought to be relieved by local bleeding, and, therefore, by the menstrual flow in this case, especially if the flow is profuse. Now it is a fact that in the dysmenorrhoea of unmarried girls the pain is usually during the day or two days preceding the flow; and that, in most of these cases, the flow brings complete relief. Further, this pain is of a dull aching character, persistent but not paroxysmal - just as we would expect in the case of pain due to hyperaemia. It seems difficult to deny that the hyperaemia is the exciting cause of this pain. How the hyperaemia causes the pain must be inquired into later on.

But supposing that the hyperaemia does bring on the pain, can a pain that distinctly precedes the menstrual flow be said to be menstrual pain? I think so, because hyperaemia is as much a part of menstruation as haemorrhage.

It is to be understood that, though generally relieved, this kind of pain does not necessarily cease when the flow begins.

The second variety of pain comes on with or during the flow. It is not likely to be due to hyperaemia, as this has existed for a few days before the flow. Can it be due to interference with the free passage of the haemorrhage from the uterus? The fact that the pain is typically a paroxysmal, spasmodic or colicy pain, seems to indicate that the uterus is labouring to expel the discharge. The proof that it is in some cases, is the observation that with the pain there is a little gush of blood often accompanied with a clot. Women with spasmodic dysmenorrhoeal pain often are clear about this when questioned, and this is often more marked on getting up after lying down for a time.

The theory that this kind of menstrual pain, known as spasmodic dysmenorrhoea, is a neurosis has been supported by Dr Matthews Duncan and Dr Champneys. But this seems to make a disease of a symptom. Further, the uterus, so far as one can judge from the action of drugs, is as stable in its nervous organisation as any other organ. Oxytocics are neither numerous nor reliable. In medicinal doses ergot rarely causes severe uterine colic, though I believe its habitual use after labour may be condemned on the ground that it will often cause severe after-pains. Picrotoxine and muscarine act through the nerves on the uterus causing contractions, and pilocarpine has even been used as an oxytocic (L. Brunton) but this action is not a prominent feature in any of them. Physostigmine, too, acts on the uterine muscles but not till all the other

effects of the drug are in full play.

Besides the neurotic theory does not help us very far. For in the case of other neuroses, e.g. hysteria, we still need an exciting cause, and the exciting cause is what is practically important. A neurosis is latent till the exciting cause comes into operation. Cramp of the stomach is no neurosis, neither is colic. Hepatic colic and Renal colic are not neuroses. As we get to know asthma better reflex exciting causes take a more prominent place, and fewer cases can be looked on as pure neuroses. And because there are some cases we can only explain as neuroses, must we keep those we understand better in the same class as those we do not understand at all?

If, then, we can in any other way account for spasmodic dysmenorrhoea, we may be able to drag a few more cases into the region of practical medicine. And in the course of the next few pages an attempt will be made to show that spasm is sometimes the expression of obstruction, in the uterus as in other organs as for instance the bladder.

For these reasons I think I am warranted in trying to distinguish between two varieties of dysmenorrhoea: one due to engorgement of the sexual organs with blood, congestive; the other due to interference with the egress of the flow, obstructive.

DYSMENORRHOEA FROM OBSTRUCTION

As the existence of dysmenorrhoea from obstruction under any condition (except complete occlusion - which cannot be denied) has been called in question by some, it will be necessary to consider one by one the pathological conditions which have been alleged to give rise to obstruction. The obstruction, of course, must be situated in the uterus or vagina; or must depend on the character of the discharge being such as to pass with difficulty along the canal of the cervix.

I. Vagina:

Obstruction here is rare and is caused by imperforate hymen which is congenital, or ~~atresia of the~~ occluded vagina which may be congenital or acquired as the result of infantile vaginitis, cicatrisation of ulcers or tears occurring during labour. To show how complete the obstruction must be I may mention a case I have met. It was found impossible to introduce the finger into the vagina of a woman in labour. A thorough examination showed the existence of a small hole through which nothing larger than a surgical probe could be passed. The urethra easily admitted the little finger. Yet the woman had had no symptoms to complain of, and impregnation had taken place. Intercourse must have been imperfect or effected into the bladder.

These conditions give rise to occult menstruation often with intense pain at the periods, and dilatation of the uterus

from retention of menses. Haemato-Salpinx and pelvic haematocele may ensue.

II. Cervix uteri:

A. Imperforate os and imperforate internal os are rare. Pozzi gives references to P. Müller, G. Lowe (Vol.II, p.250 Syd. Soc. Translat. note) who have reported cases recently.

An imperforate condition of the whole canal is doubtful.

Atresia of cervix may follow abuse of stronger caustics, and I have known a case due to amputation of cervix. It sometimes follows laceration in labour.

The symptoms of atresia of the cervix are the same as in occlusion of the vagina.

B. Stenosis of the Cervix.

This is of much greater importance because it is so common. Simpson and Sims gave great prominence to this as a cause of dysmenorrhoea and sterility. Sims' description of the pin-hole os; of the long, narrow, gristly cervix; and of the conical cervix &c are well-known. On these he based his mechanical theory of dysmenorrhoea and his treatment of it.

"Thomas, Barnes, Schröder and De Sinety all accept this theory, more or less, in their textbooks" we learn from Hart and Barbour.

Pozzi admits (Vol.II, p.253 Syd. Soc. trans) that dysmenorrhoea is sometimes absent in stenosis, but we must conclude that he is a supporter of the mechanical theory. At the same time he draws attention to the great frequency of

Metritis as a consequence of the difficulty of evacuating the mucus and blood. Sterility he thinks is due not to the stenosis, but to the plug of mucus that blocks the canal.

Of 252 women who menstruated painlessly Vedeler found that Stenosis was present in 6 per cent (Champneys' Painful Menst. p.65).

Champneys gives his own experience (ib. p.66) of 16 cases of Stenosis, some of them markedly stenosed. Half of them menstruated painlessly, while some of the others presented other possible causes of dysmenorrhoea (e.g. retroflexion with prolapse of ovaries).

It is obvious that there can be no question of Stenosis as a cause of dysmenorrhoea:-

1. Where menstruation has been painless during the first years of menstrual life.

2. Where menstruation is painless for a period or two from time to time.

3. Where a moderately large sound can be easily passed (and all writers insist on the greatest care being taken to guard against mistake here, as the sound is often passed with difficulty owing to folds &c of the canal and not stenosis), or withdrawn easily (Champneys).

4. Where there has been pregnancy (excluding possible atresia).

On the other hand if the converse of these is present, and we find the cervix corresponds to Sims' description and

hardly admits a probe, after excluding other causes, we are justified in looking on it as a case of so-called stenosis dysmenorrhoea.

It is certain, however, that Stenosis is not nearly so common as Sims believed. Indeed we must admit that Stenosis alone rarely causes obstruction, because

1. Vedeler, Champneys and other eminent observers have met many cases of marked Stenosis without dysmenorrhoea; and ~~because~~

2. In cases of Stenosis with dysmenorrhoea these observers often find other possible explanations of the pain.

After noticing Flexions I shall attempt to show that the Metritis which Pozzi so commonly found in cases of Stenosis is more likely to be the cause of the dysmenorrhoea in these cases.

C. Flexions.

Flexions are very common indeed. Physiologically, of course, there is a moderate degree of Anteflexion, and a great proportion of all anteflexions are congenital. Retroflexion, on the contrary, is almost always secondary to a confinement. And pregnancy seems to correct anteflexions as these are much more common in Nulliparae. Simpson and Sims regarded ~~those~~ flexion as a very important cause of dysmenorrhoea. But Matthews Duncan has done much to show the insignificance of the commoner degrees of Flexion as causes of dysmenorrhoea.

It is quite certain that in many cases of marked ante-

flexion there is no discomfort of any kind.

Herman and Vedeler have enquired into this point (quotations and references to both given by Champneys' 'Painful Menstruation' p.69 et seq.). Vedeler found in 3012 women that 54 per cent had anteflexions, 8 per cent retroflexions, and that 15 per cent were normal. In women who had children 38 per cent of those with uterine symptoms had anteflexion, and 37 per cent of those without uterine symptoms had also anteflexions. Hart and Barbour (Manual p.352) on analysing Vedeler's paper find that, taking Nulliparae only, 37·3 per cent with dysmenorrhoea had anteflexion, and 33·3 per cent without dysmenorrhoea had also anteflexion.

Herman's paper shows similar results.

Hart and Barbour complain that Vedeler does not keep 'pathological anteflexion' in a class by itself. As Champneys justly observes (ibid p.74) the pelvic inflammation which causes thickening of the utero-sacral ligaments and the anteflexion is quite sufficient in itself to explain the origin of a dysmenorrhoea. No doubt this class would show a larger per centage of dysmenorrhoeas, but no one could assert it was due to the Anteflexion.

Retroflexion is much less common than Anteflexion. Dr Herman found that in 85 cases of Retroflexion menstruation was painful in 64 (75 per cent) (reference and quotation Champneys' ibid p.72). Vedeler found that of 313 cases of Retroflexion 40 per cent presented no morbid symptoms.

(Hart and Barbour p.366 reference). Roughly speaking, then, Herman and Vedeler's statistics prove that 60 per cent of Retroflexions have dysmenorrhoea. Hart and Barbour (Manual p. 366)say dysmenorrhoea is not so frequent here as in Anteflexion, because as retroflexion occurs in Multiparae the canal is more patulous. This statement seems contrary to their own(quoted) statistics. Nevertheless, retroflexion is very much more common in multiparae.

Now in spite of all statistics it is certain that a curved canal cannot be so free as a straight one of the same calibre. As Barnes points out the bend must cause a break in the stream. And Dr G. Hewitt has described and figured (B.M.J. March 3rd 1888) a specimen in University College Museum in which the anterior and posterior walls are actually in contact. It must, therefore, be admitted that Flexion may cause obstruction.

At the same time statistics make it clear that Flexions may exist without causing the least trouble. Indeed a flexion can rarely be of much importance in the case of a roomy canal. In the case of stenosis, or of a canal partly filled up by swollen mucous membrane the result of endocervicitis, it will be quite another matter. So that if we admit that Flexion is rarely in itself the cause of undue obstruction, we can still point to statistics which conclusively prove it is often associated with dysmenorrhoea.

How does the dysmenorrhoea arise?

1. Simpson and Sims said it was due to obstruction, and it is certain that they cured many cases of dysmenorrhoea by treatment based on this view.

Pozzi (Vol.II p. 120 Syden. Soc. T.) says "it is difficult "to help attaching very great importance to the obstruction, "owing to the paroxysmal character of the crises and the flow "of blood."

Hart and Barbour (p. 351) object that "the discharge is "not always clotted, that in some cases it is small in quantity, "that it is doubtful whether the blood coagulates in the uterus, "and that in many cases the pains complained of have not the "distinctive character of labour pains." It may be urged that the discharge need not clot at all, and that in no case need it be large in quantity, because apart from ~~stenosis~~ narrowing the tough mucus due to the catarrh that so frequently accompanies flexions is sufficient, as will be shown just now, to obstruct the flow. In this case, too, a small quantity of discharge may cause pain. Then, in many cases, the character of the pain is quite distinctive as Pozzi points out. Nevertheless one may admit that pathological anteflexion may have a pain of a different character.

2. Fritsch (Hart and Barbour p. 351) thinks the dysmenorrhoea is not due directly to the bend on the canal. He refers it to irritation of the nerves due to congestion and to the abnormal tension of the bent vessels on the level of the flexion.

It may be said that Williams' experiments in injecting bent uteri, and the anatomical conditions present in the uterine circulation are against this theory.

3. Hart and Barbour suggest as an explanation (p. 352) pain due to the flushing of a chronically inflamed organ with blood. This no doubt applies to some cases of Subinvolution and to most cases of 'pathological' anteflexion. In the one case the organ is large and sensitive, in the other the pelvic inflammation has to be taken into account.

Champneys sees his way to deny that anteflexion is ever a cause of dysmenorrhoea, even after citing Graily Hewitt's case where the walls of the canal are in contact. The only possible explanation of dysmenorrhoea in connection with retroflexion Champneys thinks is

4. ~~Champneys thinks, is~~ "the compression of the vessels in "the broad ligaments when the uterus is acutely flexed into "Douglas' pouch and strangulated. This will lead to conges- "tion of the uterus, and possibly to dysmenorrhoea". And for this explanation he is indebted to Dr Williams, he admits.

In the majority of cases the simplest and oldest explanation is quite sufficient. And where this is the sole explanation the pain will not come till the flow begins, or will just precede the flow by an hour or two. And it will be paroxysmal. And in a typical case, such as is often met with in married women, it lasts right up to the end of the flow.

When the pain is due to the congestion the pain ought to

precede the flow by a day or two. The flow ought to relieve this pain, but as a matter of fact flexions complicated by pelvic inflammations, e.g. pathological anteflexions are always, as we shall see in tubo-ovarian cases, so complicated with metritis that the flow is apt to make matters worse. That is to say these cases are cases of spasmodic or obstructive dysmenorrhoea plus something more.

D. Inflammatory Obstruction at the Cervix:

After labour a febrile condition with pelvic pain may induce us to wash out the uterus. Sometimes it will be found difficult to insert the intra-uterine douche, and when we force it through the os a sudden gush of fluid shows that it has been confined under pressure. Oedema of the cervix has closed the os sufficiently to prevent the escape of the lochia. I have found this ten days after labour.

In his textbook (1891 p. 160) Lewers points out that in the corporeal metritis of old women there is sometimes, without occlusion of the os, accumulation of purulent secretion in the cavity of the uterus - pyometra. The explanation seems to be either that the discharge is too viscid to escape, or that the swelling consequent on the metritis is sufficient to interfere with the patency of the canal.

Pozzi (Vol.II p. 253) figures dilatation of the cavity of the cervix "owing to retention of mucus in a case of cervical metritis with narrowing of the external orifice." In the text he says that acquired stenosis is soon complicated

with inflammation of the mucous membrane, and that "the secondary lesion before long plays the principal part, while increasing in its turn the abundance and viscosity of the mucus."

We are familiar with very complete nasal obstruction following a common cold; catarrhal jaundice is of quite common occurrence; and at the beginning of a bronchitis the tough mucus requires much coughing for its expulsion. Surely, then, there can be no difficulty in understanding that the canal of the cervix, though normal in calibre (and much more so when there is stenosis or flexion), may be obstructed by inflammatory swelling of its mucous membrane. And we know that it is no easy matter to remove the stringy mucus of endocervicitis with the probe. In their textbooks, Sims, Jones, and Lewers give special directions as to how this may be done most thoroughly. This plug of mucus is likely to increase the obstruction due to the swelling of the mucous membrane. Some authors, e.g. Jones (textbook 1891, p. 295) recognise dysmenorrhoea as a symptom of the endocervicitis requiring treatment. Now endocervicitis is very common in pluriparae, and it may be objected that few women who have had children would be without dysmenorrhoea were this theory of its causation correct. Probably it will only be where the endocervicitis is of long standing and the mucus more stringy that the obstruction will be of importance. It is fortunate that most cases of endocervicitis are no more than slight chronic catarrhs, attended with no other symptom than less or more leucorrhea -

especially in multiparae where the os is patulous.

Yet it is certain that many dysmenorrhoeas show ~~us~~ no more abnormality than slight anteflexion and a congested cervix, with tough secretion hanging out of a somewhat abraded os. The sound passes easily. If the secretion is wiped off, and carbolic acid applied a few times to the canal and fundus, the woman is often completely and permanently relieved. Cases such as these are common in ordinary practice. The patients are generally married women who have been sterile for some years, and soon after treatment, they, as a rule, become pregnant. The benefit following such treatment tells in favour of the origin assigned to the dysmenorrhoea.

The fact that the sound passes readily proves nothing. A No 10 catheter may often be passed in the male when there is retention of urine.

Under stenosis attention was directed to the observation of Pozzi that metritis, as a rule, goes along with stenosis.

Almost all authors in considering the treatment of flexions agree that the treatment of the metritis is of the first importance. The use of pessaries is not nearly so important in most cases, and in many a pessary is not required at all. In many cases of stenosis and of flexion there is dysmenorrhoea as all statistics show . And we have seen that marked flexions and stenoses occur without it. Is it not probable that cervical catarrh is the key to the puzzle?

At the same time I think it has been proved that extreme

degrees of flexion and of stenosis are sufficient causes. Edis argues that the canal is at least 3/16th inch in diameter; and that the average flow is not more than 2 oz in 24 hours, giving 40 m. per hour, or 2/3rds of a drop every minute: and finds it difficult to imagine how there could be any impediment to the passage of so small a quantity. (Textbook 1882, p. 454).

Champneys argues in the same way.

Now let us suppose the flow is a mere dribbling of blood from the uterus. It is clear that this blood must be displaced from the uterus either by its own pressure, or, as happens in the emptying of a bottle, by air. As the vaginal walls lie in contact and the cervix is narrow, the entrance of air is excluded, so that the blood that comes from the uterus is displaced by fresh blood oozing from the endometrium. If the cervix is patent all will work smoothly. If there is any considerable narrowing, there will soon be an unusual degree of pressure in the uterus. This will stimulate the uterus to contract, and if the obstruction is serious uterine colic is the result. But it seems probable that contractions take place during normal menstruation. We have the contractions of the gravid uterus; labour pains; after-pains; occasional regurgitation of menstrual blood through the tubes; expulsion of membrane in membranous dysmenorrhoea; and spasmodic dysmenorrhoea. And without contractions a certain quantity of blood must remain in the uterus after menstruation

till it is displaced by mucus, and the uterus be always filled. Champneys (p. 12) thinks contractions take place during menstruation. Hart and Barbour (p. 586) have felt indications of it. In Metritis, occasionally, I think I can refer intermenstrual pains to spasmodic expulsion of whites, as women frequently tell one that they have gushes of whites with the pain. And can the uterus empty itself as quickly through a narrow cervix as through a roomy one? Is not a narrow cervix a sufficient cause for uterine colic? I don't think it requires a very lively imagination to see that such a thing is probable. In this matter, it seems to me, agnosticism has been pushed too far.

E. Obstruction due to Polypi and Fibroids:

Champneys appears to deny that these cause dysmenorrhoea in any circumstances. Pozzi says (Vol.II, p. 251) Atresia "may be due to presence of tumours in the cavity of the cervix" And he quotes a case reported by Meredith of "haematometra associated with a degenerating fibro-myoma." Rhinologists know that polypi cause nasal obstruction. There is no reason why one should not act in the same way at the cervix, even admitting that dilatation of the cervix occurs.

In the case of both fibroids and polypi, in any situation, there may be dysmenorrhoea, for, though not actually obstructing the cervix, they may give rise to endo-metritis and cervicitis and so act indirectly.

Most authors admit they are causes of dysmenorrhoea.

Having considered the chief pathological conditions of the Cervix associated with dysmenorrhoea and likely to act as obstructions to the flow, it but remains to point out that a cervical canal which is free enough for a normal flow may be more or less completely blocked by an abnormal discharge.

F. Obstruction due to abnormal character of discharge

In the after pains following labour we very often are told that considerable distress has been at once relieved by the passage of a clot or shred of membrane. There seems no reason to doubt that the clot has temporarily obstructed the discharge and given rise to the pain in the pelvis by inducing uterine spasm.

And in membranous dysmenorrhoea the character of the discharge is so striking as to be accepted as the cause of the pain which so constantly occurs, at least during the passage of the membrane.

Now, there is no agreement as to the cause of membranous dysmenorrhoea, but there is wonderful unanimity as to the hopelessness of any treatment that does not make an energetic attack on the mucous membrane of the uterus. It has been alleged by some that it is simply a series of monthly abortions but the first case, reported by Morgagni, is sufficient to prove the contrary. (Champneys, p. 30). McNaughton Jones in his textbook, remarks that chronic inflammation has been often found to accompany it. Schröder thinks it is so often

accompanied by chronic inflammation of the uterus as to suggest a causal relationship. Wyder and Pozzi have no doubt of this.

And there are observations which have a direct bearing on the formation of such a membrane. For instance Lewers (textbook 1891, p. 158) in describing a corporeal endometritis following a bad confinement mentions the escape of 'yellow, leathery, offensive masses,' a month after the confinement, which he was not sure whether to look on as sloughs or clots. Again Jones (textbook 1891. p. 522) tells us that "in patients "whose general health is impaired and who contract vaginitis "through the irritation of purulent discharges from the uterus, "membranes may form on the vaginal mucous surface, of a diph- "theritic character."

After the introduction of a small piece of Silver Nitrate into the uterus for subinvolution, a woman passed p.v. after much pain a membranous looking body on the third day. In the Lancet (March 11th 1893, p. 546) a similar case is reported after Sulphate of Copper. Four days after there was uterine colic and vomiting and "a quantity of false membrane was passed in which microscopical examination showed all the elements of the uterine mucous membrane." This seems to be artificially induced membranous dysmenorrhoea.

Exfoliation of uterine mucous membrane is known to occur sometimes in Cholera and in phosphorous poisoning. (Champneys p. 20)

In a case of intermittent hepatic colic with Jaundice and sometimes with feverishness lasting a day (101° to 103°), the most careful examination failed to discover gall-stones once in many attacks. But several times we found long, tough, gelatinous strings which the patient had taken for tapeworms. These suggested an origin for the jaundice in inflammation of the bile-ducts, leading to formation of casts, which in their passage caused colic and obstruction. Dr Ralfe has reported another such case quite recently (Clinical Journal Nov 9th 1892). And it must be remembered that in many of the cases of membranous dysmenorrhoea (Champneys' tables) the membrane is fibrinous. Further, in membranous dysmenorrhoea the process of membrane formation sometimes extends into the vagina (Champneys p. 49). Then it is only necessary to mention diphtheria and croup, and fibrinous Bronchitis. There seems no reason to doubt seriously the connection between Chronic inflammation of the uterus and membranous dysmenorrhoea.

A full account of the Metritis theory of membranous dysmenorrhoea and references to Schröder, Wyder and others is given in Pozzi's chapter on Metritis in Vol.I of his textbook (Syden. Soc. T.). He defines it as "a chronic metritis with acute exacerbations and inflammatory shedding of the mucous membrane during menstruation." Sometimes only shreds are eliminated, sometimes the mucous membrane is expelled entire. Membranous dysmenorrhoea dates from a confinement or miscarriage, or, more rarely, from the establishment of menstrua-

tion - the usual origins of endometritis. It tends to go on to the menopanse, if not treated. It tends to sterility, but pregnancy may intervene and the disease return after delivery. Curetting, then Iodine applications, give good results. Such is a short summary of Pozzi's views. Even Champneys who thinks the dysmenorrhoea spasmodic recommends curetting for its treatment.

No doubt the pain often begins a few days before the membrane is expelled, as Champneys points out, but the endometritis is the key to this. The menstrual hyperaemia will increase the congestion of the inflamed uterus and cervix, and the canal may be still further obstructed by tough mucus. In short there will be an ordinary obstructive dysmenorrhoea with a membranous dysmenorrhoea added to it.

But whatever the pathology of membranous dysmenorrhoea, it is conceivable that similar causes, acting in a less degree, may give rise to clots, shreds, and altered character of the flow, instead of a membrane. Such a discharge may not get readily away, especially if it arise in connection with an inflamed uterus, but, gradually accumulating, may give rise to painful uterine contractions from time to time - in short, to an obstructive or spasmodic dysmenorrhoea.

In this light it is interesting to read the usual description of spasmodic dysmenorrhoea. Lewers (textbook p. 76) observes that "the pain begins a few hours before the flow "and lasts for the first day or two of the period. The flow "then usually increases in quantity and the pain diminishes or

"ceases. The pain is at its worst while the flow is scanty.
"The blood may contain clots or shreds."

This is very similar to Champneys' description. He thinks it almost always is primary, i.e. begins with the onset of menstruation. And "the flow is scanty, especially during "the time the pain is most intense. The blood is often mixed "with clots,which on examination are found to be the membranes "of membranous dysmenorrhoea." Further on (at p. 81) he holds it is established that (1) Sapsmodic dysmenorrhoea is pre-eminently an affection of the immature uterus; (2) the pain is certainly colic, and often precedes the flow, and sometimes occurs without any flow at all; (3) it is often associated with nervous phenomena in other parts of the body. Then he begins to theorise: "It is essentially a neurosis, and has motor "phenomena (colic), and vaso-motor phenomena (scanty or greatly "varying flow); some would go further, and add secretory "phenomena, such as catarrh, and quote the analogy of asthma."

This neurotic theory has been criticised in my introduction and I need not again refer to it. Nervous phenomena are very common in connection with menstruation, painful or otherwise.

We must accept the clinical facts. But, surely, another explanation may be suggested, viz:- The Menstruation begins with a slight oozing of blood which as it slowly fills the uterus becomes mixed with flakes of mucosa. It is retained

longer than when the discharge is free, and this delay leads to clotting (which is favoured by the epithelial debris, perhaps). The clots prevent free egress of discharge and so bring on uterine colic - In short, we get spasm as the result of obstruction. After a day or two the flow becomes freer, and the more copious discharge does not linger long enough to clot, but washes out the uterus and puts an end to the trouble.

That immature uterus is not essential is shown by the occurrence of the dysmenorrhoea in multiparae. Champneys (ib. p. 59) explains that in women who have rapidly borne many children the uteri are worn out, exhausted, and subject to after pains. In the same way he thinks they are subject to spasmodic dysmenorrhoea. I would suggest that the frequent occurrence of labour in these uteri has brought on chronic metritis and that the dysmenorrhoea is due to the catarrh and, perhaps, alterations in the discharge - the result of the metritis.

Champneys observes (on the same page) that 'in some cases 'the pain may not precede the flow, and may not be relieved 'by the flow." I have met with this condition in a married woman with one child five years old. The dysmenorrhoea was severe, began with the flow and lasted throughout. There was nothing but chronic metritis and tough catarrh. One application of Carbolic cured it and in three months she was pregnant. Theoretically this is what we would expect in a typical spasmodic dysmenorrhoea - pain not preceding flow.

But it is uncommon in practice. And I think this is because we have not often chronic metritis without ovarian congestion or slight salpingitis.

In this connection I would quote Pozzi (Vol.II p. 329) "even when the metritis is the predominant condition, it is "very frequently accompanied by a slight amount of Salpingitis "quite sufficient to lead to tenderness of the appendages." On this account pain precedes the flow in most cases and is due to hyperaemia of the congested appendages.

Again Champneys says "the character of the pain is not "always intermittent, it may be continuous; perhaps, because "one spasm overlaps another." But a continuous pain can not well be called spasmodic. Further, continuous contraction must end in fatigue. It is interesting to note that Champneys explains the action of ergot in dysmenorrhoea by supposing that it converts colicy or clonic contractions into tonic contractions and so gives relief. There seems contradiction in this. Personally, I give ergot to stop the flow in dysmenorrhoea. And I find that the most effective prescription I have for spasmodic dysmenorrhoea is ergot and belladonna in fairly large doses. The flow usually stops soon. Appropriate treatment of the diseased condition can then be tried during the intermenstrual period. It is probable that continuous pain means that there is something more than spasmodic dysmenorrhoea. More than one form of dysmenorrhoea may be present.

"In rare cases" says Champneys "the pain is intermenstrual "or occurs where there is no flow at all." Again (ibid p. 82) he quotes Vedeler's observation that dysmenorrhoea may occur monthly without any flow at all. There can be no question of (obstructive or) spasmodic dysmenorrhoea here. But ovaritis would have to be excluded. And we know from experiment that irritation of the ovarian, crural or sciatic nerves produces reflex uterine contractions (Lander Brunton's Pharmacology p. 454, 3rd edit.). And it has been observed that in ovaritis pains sometimes ocur with evacuation of muco-pus from uterus. These Pozzi thinks (Vol.II p. 326) are reflex uterine contractions. Ovaritis is not common - and these "pains" are not common in ovaritis, but "there are often prolonged periods of amenorrhoea in ovaritis. (Pozzi Vol.II p. 327)."

Rheumatism, Gout Anaemia predispose to catarrh & ∴ to dysmenorrhoea — The dysmenorrhoea depending on congestive obstruction of the cervix, or changes in character of flow due to endometritis combined with endocervicitis. Spasmodic dys. in a rheumatic girl, aged 19, of two years standing that followed a chill with suppression tonsillitis I found yielded completely to Soda Salicyl & Iodid of Potash taken for 6 weeks. There was a little pain for a day before the period, but the loss greatly increased the pain which became typically spasmodic & lasted till the end of the period. There was slight leucorrhoea between periods.

DYSMENORRHOEA DUE TO ENGORGEMENT OF DISEASED ORGANS — ~~DYSMENORRHOEA~~ DUE TO HYPERAEMIA AT THE PERIOD

In the introduction it was stated that it was difficult to distinguish more than two kinds of dysmenorrhoeal pain. It was suggested that one was due to obstruction - this one has now been considered; and one to the hyperaemia that precedes and attends the period. The former has its origin in the uterus. Congestive dysmenorrhoea has its origin chiefly outside the uterus, - in the ovaries and tubes. It has been pointed out that the characteristics of the congestive form are: pain preceding period, of a dull, aching - not paroxysmal character, and relieved by the flow. It remains to be considered how this pain arises.

In order to understand this it will be necessary to consider the structure of the appendages, the effect of inflammation on them, and the changes they are subject to at the periods.

The tube is a hollow cord, the walls of which are made up of peritoneum, connective tissue, unstripped muscle and mucous membrane. In cross section the lumen of the tube is seen to be almost filled up by folds of mucous membrane. There is a very rich arterial supply to the mucous membrane, whilst veins and lymphatics are abundant in the walls of the tube. The fimbriae, one of which is attached to the ovary, contain involuntary muscle and are very vascular. The vas-

cularity of the tube and the abundance of veins and venous sinuses in its walls show that it is an erectile organ - such is the opinion expressed, after special anatomical examination of the tube, by Mr Milroy in his paper in the Glasgow Medical Journal for June 1891. The effect of the menstrual hyperaemia is probably to increase the length and thickness of the tube, and so bring it into a closer relation to the ovary.

Freund (Pozzi Vol.II p. 313) distinguishes two kinds of tubes in healthy women: one nearly straight and of normal calibre; the other contorted and constricted in places, a remnant of the infantile condition. In the first class tubal affections run their course rapidly and may be recovered from without treatment. In the other suppurative inflammations necessarily terminate in the formation of cysts from the narrowness of the oviduct. This conformation may be suspected when the patient has a delicate complexion, and has had dysmenorrhoea from the very commencement of menstruation.

But without malformation we can understand the havoc an acute inflammation will work in the tubes.

On account of the continuity of the mucous membrane of the tube with that of the uterus, and the frequency of endometritis, one would expect that Salpingitis is common. In a series of 100 female bodies, Lewers found grave tubal disease in 17. And although this would be very excessive for private practice, it shows how common is tubal disease. In almost all cases salpingitis is secondary to endo-metritis. And

reference has been made to the opinion of Pozzi that more or less Salpingitis commonly accompanies an endo-metritis, The more intense inflammations usually arise after gonorrhoea, or sepsis following abortion or confinement. These spread through the uterus to the tubes setting up acute purulent inflammation, which almost always obliterates the abdominal ends of the tubes by matting together the fimbriae. This shuts the pus out of the peritoneal cavity, although the ovary is practically always involved in the wreck of the fimbriae. The inflammation may stop here, and, gradually passing into a chronic form, leave a more or less thickened and distorted tube adhering to an inflamed ovary. Or strictures may arise in the tubes at the narrow uterine portion and pus may be shut in between the two ends of the tube giving rise to pyosalpinx. The dilated tube, fixed to the ovary by adhesions, commonly sinks into Douglas' pouch where it contracts other adhesions.

Now what is the effect of menstruation on tubes like these? The hyperaemia comes on, and the thickened, adherent, distended, distorted tubes cannot accommodate themselves to the turgidity as they did. Erection is interfered with, adhesions are dragged on, and the period is preceded by pain as it is by hyperaemia.

In tubal gestation there may be little uneasiness even when the tube is distended to the size of an egg, and the irregular menstruation that attends it may be painless.

Indeed no symptom may precede rupture of the sac. It is, therefore, the adhesions and dragging that occasion the pain rather than the dilatation of the tube. Indeed, this pain seems altogether comparable to chordee.

It is generally believed that erection of the appendages attends coitus. In cases of haemorrhage from the uterus, the haemorrhage is sometimes observed to start afresh after coitus. Women have been known to menstruate for the first time shortly after coitus. I have met one case where a woman over twenty who had never been unwell began to menstruate shortly after her first coitus. Certainly women have been known to conceive without menstruating at all. In fact, so far as the sexual organs are concerned, coitus may be looked on almost as transient menstruation without sanguineous discharge. We are, therefore, prepared to find that dyspareunia is a very constant symptom of tubal disease. Women with tubal disease have often for months to give up marital relations on account of dyspareunia. Although tenderness of the internal organs to contact is present in some cases, in others the act is not painful but the pain sets in soon after it. This tells against its arising from tenderness of the organs to contact in all cases. The explanation in some cases seems to be that the tubes are unable to expand properly when flushed with blood but drag painfully on their adhesions. The great sensory excitement may mask the pain during the act, but soon after the aching sets in and a neuralgic condition once started is

likely only gradually to pass off.

The natural history of tubal disease indicates that a tubal is almost always mixed up with a uterine dysmenorrhoea, because a condition of uterus sufficient in itself to give rise to obstructive dysmenorrhoea often precedes tubal disease. Indeed, an endometritis may go on for some time without affecting the tubes to any extent, and there will be only obstructive dysmenorrhoea. After a time a granular condition of the uterine mucous membrane may come on and the more profuse mucous discharge will get less freely away. Then the tubes are invaded just as are the Eustachian tubes in adenoid disease of the naso-pharynx. And in this way tubal disease may more slowly develop till a condition as disastrous as the result of an acute inflammation is arrived at. The dysmenorrhoea will now be composite and the premenstrual pain be as pronounced as the menstrual.

It is certain that in tubal cases, after curetting and the daily use of glycerine tampons and the vaginal douche, there is often great relief and permanent improvement in the dysmenorrhoea and other symptoms. In short, treatment of the uterine condition alone is often followed by excellent results. Martin says that only one-fifth of his tubal cases require the major operation (Medical Annual 1888, p. 443).

Again it is just as certain that extirpation, though it generally relieves and often cures, not infrequently does little good. And this we might expect. For the condition

of uterus remains very much as it was. And the ovaries, existing as little masses of connective tissue or bags of fluid, have long been to a great extent eliminated. Indeed, the removal of such obsolete organs must often have little effect on the recurrent haemorrhages from a granular surface developed on a mucous membrane prone to bleed. We have here an explanation of the failure of many extirpations. In these cases treatment of the endometrium should always be practised. Pozzi (Vol.II, p. 364) says "every operation on the tubes should be followed by curettage to modify the concomitant endometritis."

In regard to menstruation after extirpation, Pozzi thinks the changes in the mucous membrane in these cases are favourable to it. He quotes Sänger as having only two cases in which the catamenia persisted after extirpation. In one of these menstruation stopped after curetting, in the other menstruation probably depended on endometritis attendant on multiple myomata. Pozzi also quotes Czempin's opinion that the cicatrix in the broad ligament left by the operation may cause passive congestion and favour bleeding. (Pozzi Vol.II, p.275 and note).

I have been closely associated with the treatment of two cases by castration. In one the operation was for menorrhagia attendant on multiple small myomata; in the other for excessive menorrhagia and dysmenorrhoea, recurring every fortnight, in a case of chronic salpingitis and cystic ovaries.

In neither case was there the slightest loss after convalescence, although in both menstruation began, as is usual in these operations, soon after castration and lasted for a few days. In the latter a rather profuse leucorrhoea with bearing down pains showed that her endometritis was still active - indeed, I had to dilate and treat the endometritis a few months after the major operation. The menorrhagia in these two cases ~~was~~ had been so excessive that it was hardly expected that castration would be entirely successful in arresting it.

Lewers reports (textbook 1891 p. 282) two cases in neither of which menorrhagia was remarkable, in which after castration the catamenia still continued. Indeed, in one case the loss seems to have been greater after the operation. Both were married women.

In the two I have mentioned above one was unmarried, the other was a widow. It seems probable that after castration a married woman may have lossess determined by the hyperaemia attendant on marital relations.

Ovarian dysmenorrhoea

Ovaritis, though very uncommon, perhaps, is admitted by most authors to exist as a disease by itself. Pozzi allows (Vol.II p.330), while insisting that ovaritis is almost always due to and associated with tubal disease, that fibro-cystic ovaritis may exist alone. And most writers take this view. The association of ovaritis and mumps has been long admitted.

It sometimes ocurs in the course of fevers. Matthews Duncan has ascribed its origin in some cases to indulgence in alcohol. It is generally believed that a chill at the period by stopping the flow may lead to congestion of the ovaries. The symptoms of ovaritis are usually stated as (1) pain present more or less during the intermenstrual period, but greatly increased as the period approaches. (2) This pain is often accompanied by sickness. (3) Tenderness in the ovarian region and exceeding tenderness on vaginal examination.

Now if there is such a thing as simple ovaritis, it does not seem unreasonable to infer that a condition short of ovaritis may exist which only gives trouble during the engorgement attending the flow. And the flow may relieve this pain.

As already mentioned, the ovary is known to be enlarged at the menstrual period. And a pain, especially in the left side, of no great severity is a frequent forerunner of a normal period. In a large proportion of the dysmenorrhoeas of young unmarried girls the pain is experienced during the two days preceding the period, and the flow brings complete relief. The pain is described as an aching pain in the side, especially the left side; and sometimes it is intensely acute. Sickness sometimes accompanies this pain. And the patient refers the pain to the ovarian regions which are often tender to pressure. It is evident that this form of dysmenorrhoea has in it much that reminds one of ovaritis. But there is no uneasiness at all during the intermenstrual period. It seems probable that

it is the result of congestion of the ovary. In many cases one gets a history of a chill at the menstrual period leading to suppression and pain at the next period. The chill may act on the ovary, or it may cause a slight endometritis with ~~tenderness~~ congestion of the appendages. In the absence of stenosis and flexion there is not likely to be the least obstruction, and the tenderness of the appendages merely gives rise to pain preceding the period. Further I have often known a case of this kind at the beginning, that afterwards had pain during the flow added to the pain that had preceded the flow. I have also seen a case of this kind alter soon after marriage, so that there was pain more or less during the intermenstrual period. In short, marriage converted the congestion into inflammation of the ovary.

In reference to intermenstrual pain it may be mentioned that some writers would go the length of naming an 'intermenstrual dysmenorrhoea.' This, they suppose, depends on ovulation, painful ovulation occurring apart from menstruation. There is no proof that ovulation is ever painful. And Pozzi has no hesitation in referring the symptoms to inflammation of the uterus or appendages. (Vol.II, p. 288) I could imagine something of the kind arising in endometritis from pains induced by retention of the mucus in the uterus. And in the less grave forms of tubo-ovarian disease there is often pain, exactly like the dysmenorrhoeal pain, brought on by coitus and lasting for many hours after.

Lastly it may be mentioned that ovarian varicocele has been said to cause dysmenorrhoea (Pozzi Vol.II p. 287).

Prolapsed ovaries may suffer from passive congestion owing to the bend of the broad ligament and give rise to pain at the periods.

So far as treatment is concerned it may be said that ovaritis is not of much importance as it is rare. And knowing that the common dysmenorrhoea of young girls is quite relieved by the flow, our object is to encourage its appearance. We cannot depend on doing this. But as the flow probably acts by relieving congestion we can always accomplish this by lowering the general pressure. For this reason a warm bed, warm drinks, sweating powders, spirit of Nitrous Aether, Nitroglycerine and Nitrite of Amyl, & antifebrin &c. act well. The reputation of gin and hot water is surely thus to be explained, but there are better remedies in the preceding list.

The narcotics are not so often required here as in obstructive dysmenorrhoea.

This ovarian dysmenorrhoea is commoner than practice would indicate, because its treatment is ~~easily~~ often within the power of the matron who has the people's pharmacopeia at her finger ends.

Antispasmodics are valuable in dysmenorrhoea because they lower vascular tension, not because they relieve uterine colic.