Under x rays such a condition will show a filling defect, and fixation of the colon. Recently at operation on two cases of this type there was found in both a section of the colon wall, several inches in length, acutely inflamed and densely thickened, with the minute diverticula bulging through the peritoneum. The condition resembled a sort of cellulitis of the bowel wall.

When there is a simple diverticulosis we have noticed that if photographs taken at different periods are compared fresh diverticula can usually be seen, and that some of the previously existing ones are larger. From a study of a number of such cases it seems that, while inflammatory complications may be held in check and often made to retrogress by appropriate treatment, it is not possible to prevent the diverticula from becoming larger or to make them decrease in number unless the faecal contents are deflected from the affected area by a colostomy or short circuit. From the patient's point of view the diverticula do not cause any symptoms as long as inflammatory complications can be avoided. Unfortunately, patients tend to get careless when they have no symptoms, and they think they are quite well and need not continue the treatment. This too often results in acute complications, which may be disastrous. One patient, after being quite well for several years, perforated suddenly while travelling from Paris to London, and died from acute peritonitis. Another patient suddenly developed a perforation into the bladder.

CANCER AND DIVERTICULITIS

Occasionally cancer undoubtedly occurs as a direct result of previously existing diverticulitis. It is, however, a rare complication, and in the only cases met with in which diverticulitis was a direct causal factor there had been extensive inflammatory complications lasting over very many years. One patient had septic sinuses communicating with the colon on the abdominal wall and in the perineum; cancerous changes had occurred in two of the sinuses and had spread to the skin. In another case, an adenocarcinoma had developed and spread on to the abdominal wall along the track of a septic faecal sinus. In a number of patients carcinomatous growth has been found associated with diverticula in the colon. There has been nothing to prove, however, that one condition was a causative factor of the other, and it is more probable that the association was accidental. We have to remember that both diverticula and carcinoma tend to occur at about the same period of human life, and it is therefore not improbable that they may occasionally be found associated in the same individual. The only fact of importance that seems to be established in this connexion is that cancer may sometimes supervene upon a neglected and chronically septic diverticulitis.

TREATMENT

We do not propose in this paper to discuss at length the treatment of diverticula of the colon. We have still very much to learn, and there is ample room for improvement of our methods to-day. At present we can do little, if anything, to prevent the development of diverticula, or to stop their increase either in size or in number. Nevertheless, careful treatment will do much to prevent complications, or, if complications have occurred or are obviously threatening, to obviate such fatal or serious results as perforation into the bladder or peritoneal cavity. Operations for diverticulitis, the inflammation from which has spread to other organs, or has resulted in obstruction, are among the most difficult of surgical procedures. Results are often excellent, however, and patients who have become hopeless invalids can be restored to good health and usefulness. A chronic septic condition, as

exists in diverticulitis, is apt in time to be a source of similar trouble in other parts of the body not within the immediate neighbourhood of the lesion. Cases of chronic septic arthritis, phlebitis, thrombosis, empyema, and meningitis, have all come within our experience, while chronic sepsis of the urinary tract is quite commonly associated with the condition.

The best results have been obtained in cases in which the affected portion of the colon could be completely resected and the bowel joined up again; this is the one condition which can be completely cured. We have had several such cases, but unfortunately it is rare to find the lesion sufficiently localized at one portion of the colon to render resection possible.

CONCLUSIONS

1. Diverticula of the colon are not the result of inflammation, and inflammatory conditions of the colon wall which often follow diverticula are always secondary.
2. In spite of treatment, diverticula of the colon, once formed, tend to increase both in size and in number.
3. This increase is often very slow, and may be unaccompanied by symptoms.
4. In the absence of secondary inflammation, diverticula do not, as a rule, cause trouble, but we have reason to believe that exceptionally sudden perforation from the giving way of a diverticulum may occur, apart from any complicating inflammation.
5. A definite diverticulitis is always a very serious lesion, with grave possibilities.
6. A sudden disaster from perforation is not so uncommon as is often supposed.
7. Suppurative diverticulitis is occasionally followed by carcinoma, but it is not a common causal factor of cancer.
8. Operative treatment should not be left until after some grave complication has occurred, but should be timed so as to anticipate and to prevent such disaster.

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THE KELOID OVARY*

BY

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The uterus is the passive partner in the group of sexual organs. Its function is not to originate impulses nor to elaborate hormones, but simply to be *sensitive* to impulses or hormones originated by the more active partners, of which the most prominent, if not actually the most active, are the ovaries. It is important, I think, when considering diseases of the uterus, to bear in mind its state of physiological dependence. It is important because it may possibly lead to the view that symptoms of disease of the uterus—for example, uterine haemorrhage and dysmenorrhoea, associated, perhaps, with definite pathological changes in the endometrium—may be due to a disease or disorder of function originating in the ovary. For instance, the ovary may fail to send any message at all to the uterus, or, on the other hand, it may send so disordered a signal that the endometrium is left, as it were, in a state of uncertainty how to react to it.

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In this paper I shall attempt to discuss the part played by ovarian defect in the etiology of excessive menstrual loss associated with little or no gross change in the uterus that can be made out by bimanual examination. This is the condition that used to be called chronic endometritis, or, when there was excessive hyperplasia of the endometrium, *endometritis tuberosa vel fungosa*; curettings from these cases, if they show anything more than normal premenstrual endometrium, show more or less hyperplasia without the characteristic appearances of chronic inflammation.

Wilfred Shaw, in his important contribution to the literature of uterine haemorrhage, discusses this type of case, and mentions that it is frequently associated with atrophic changes in the ovaries. He considers that this change in the ovaries is due to an influence exerted on them by the diseased endometrium. I have noticed changes in the ovary in this type of case, and I want to suggest a different and, as it seems to me, more likely explanation.

The ovary is unique among the organs of the body in two respects—one physiological, the other pathological. The physiological peculiarity is that it is the only organ which is normally subject to trauma and consequent reparatory changes. The trauma consists of the lesion of the surface that occurs at the time of the rupture of the follicle and the formation of the corpus luteum. The reparatory process is the little scar, consisting of fibrous tissue, by which the surface lesion is repaired. Nowhere else in the body, except in the umbilicus, is there such a thing as normal scar tissue. The process of healing of this little normal lesion is the same as that in any other wound.

KRUKENBERG TUMOURS OF THE OVARY

The pathological peculiarity of the ovary is the occurrence in it of the remarkable bilateral carcinomatous growths known as Krukenberg tumours, found nearly always, if not invariably, in association with carcinoma of the stomach or large bowel. Consideration of these tumours will lead me into a digression from the main argument, but I would like to make a suggestion as to the origin of these tumours.

We may, I think, regard it as extremely probable, though not inevitable, that, if in any organ, or indeed in any object at all, we find two unique features, these peculiarities will be found to be connected in some way. One will depend on the other and could not exist without the other. If this argument is accepted as logical, we may seek to find some association between the two features of the ovary which make it unique—its normal liability to trauma and the occurrence of these remarkable tumours.

There are two current explanations of the Krukenberg tumours. One is that cancer cells become detached from the primary growth and are grafted on the ovary after falling through the peritoneal cavity. This does not explain why the ovaries should be the only organs in the abdomen to be affected, or why cancer cells are found in the deeper parts of the ovary and not on the surface. The other view is that the ovary is invaded by lymphatic spread via the lumbar glands. This also is difficult to accept because it does not account for the absence of infiltration of other lymphatics (for even glands in close relation to the primary growth are often not involved in these cases), or for the fact that the tumours are bilateral.

The first hypothesis, however, becomes more easy of acceptance, and indeed, I suggest, affords an entirely satisfactory explanation, if we employ the logical argument that this unique pathological phenomenon is likely to be connected with the unique physiological one—namely, the periodical occurrence of an open wound on the surface of

the ovary. Cancer cells are delicate weakly cells, more vulnerable than other cells (for example, to α rays and radium), and they will perish if they fail to find a suitable nidus. The surface of the peritoneum does not afford a suitable resting-place, nor would the ovary, if its surface were unbroken. But if it should happen that cancer cells become detached (and it must be a rare occurrence, corresponding to the rarity of these tumours) and fall into the cavity formed by a recently ruptured follicle, they will find a place where conditions are favourable for survival. The wound is quickly healed over by scar tissue, which forms, as fibrous tissue always does, an insuperable barrier to the spread of cancerous growth. The path of least resistance (always taken by cancer cells) is downwards into the substance of the ovary, along the track taken by the ripening follicle in its ascent to the surface. Hence the growth takes place in the substance of the ovary, not on the surface.

The hypothesis that Krukenberg tumours originate from the implantation of cancer cells in a ruptured follicle was suggested by Schauta. If it is valid it will affect our view of the prognosis of these cases. We shall not regard them as having the same significance as secondary growths spreading by lymphatic permeation. If they are removed before the growth has spread beyond the ovaries, and if the primary growth can be removed entirely, the prognosis should be good compared with that, for instance, after removal of a growth of the breast and affected axillary glands.

I will conclude this digression by recalling that there is a third peculiarity of the ovary: it is the only organ in the abdomen that is uncovered by peritoneum. The connexion of this peculiarity with the function of ovulation and also, if this hypothesis is correct, with the occurrence of the Krukenberg tumour, is obvious.

To return now to the normal ovary and in particular to the process of repair of the lesion made by the rupture of the follicle. This little wound, like other wounds, is repaired by the growth of connective tissue cells in the form of scar tissue. In course of time the smooth surface found at the time of puberty is gradually changed to a more or less corrugated surface, patchy at first, but later becoming uniform. Normally there is no marked change in the consistence of the ovary; the fibrous scar tissue is thin and does not convey the impression of hardness. Quite often, however, one finds here and there, on the surface of the ovary, areas or patches of exceptional hardness, suggesting that in these areas fibrous tissue has been laid down in a greater degree than usual. Less often the hard fibrous patches are so numerous and continuous that the ovary has almost the hardness of wood, though there are generally softer parts where cystic dilatations of follicles project on the surface. Microscopical sections of these ovaries demonstrate the excess of fibrous tissue.

The process of repair in all tissues depends on the existence of a regenerative capacity. This function of regeneration varies in different tissues and in different individuals. Some tissues have a very perfect capacity of regeneration, for instance the skin and mucous membranes generally. Lesions of these tissues, when they heal, show little or no evidence of the former injury. On the other hand, lesions of highly organized tissues, such as muscle and the cells of the central nervous system, show little or no evidence of any regenerative capacity; injuries to these tissues are repaired by connective tissue. But the regenerative capacity varies not only in different tissues but also in different individuals. A good example of this is seen in the repair of fracture of bones; in some individuals repair is delayed far beyond the normal; in a few there is practically no reparative process at all. Even in the simplest form of the regenerative process, scar formation from growth of connective

tissue, there are individual variations. Thus it is not uncommon to find, especially in slowly healing scars, a tendency to overgrowth of fibrous tissue. This condition we speak of as keloid.

The excessive formation of fibrous tissue found sometimes in the ovary may be looked upon, I suggest, as a form of keloid overgrowth of fibrous tissue. It is a keloid scar formed as a disordered or exaggerated regenerative process in the healing of the scar left by the ruptured follicle. It is not a disease in the sense that it is a reaction to a morbid extrinsic factor, such as bacterial infection. There is no evidence of inflammation, and it is incorrect to speak of the condition as chronic ovariitis. It is a disease, however, in the sense that it is the cause of symptoms of disease. I shall endeavour to show how such a condition is able to produce pathological manifestations.

Whatever theory we may hold as to the interaction of the various sexual hormones in the initiation and regulation of the menstrual cycle, it will, I think, be agreed that the liquor folliculi contains some substance that is essentially and perhaps solely concerned with the activation of the endometrium. It may or may not be necessary for an ovum to be discharged; it may or may not be necessary for a corpus luteum to be formed; but without the hormone elaborated in the liquor folliculi the endometrium remains inactive. Perhaps we may go so far as to say that the complete cycle of ovulation, including the formation and atrophy of the corpus luteum, is necessary for the normal cycle ending in complete shedding of the menstrual decidua. If the ripe follicle comes near the surface of the ovary, but, for some reason, does not rupture the surface, then no corpus luteum is formed, or rather an incomplete or abortive corpus luteum results, projecting, as we sometimes see it, on the surface of the ovary as a sort of polypoid excrescence; no haemorrhage has taken place into it.

It is most unlikely that such an abortive corpus luteum will function entirely like a normal one; it may fail to function at all; but it is more likely that the granulosa cells, formed for the special purpose of secreting a hormone whose object is to maintain the endometrium in its thickened state, to prevent its disintegration, and so to "prepare a bed" for the ovum, should at any rate make some attempt to exercise their function. We are justified, I think, in making the assumption that the signal sent by the abortive or imperfect corpus luteum to the uterus will be an imperfect signal; it will be transmitted to the endometrium, but, if I may put it so, it will not be so readily understood, with the result that the endometrium is left in a state of uncertainty as to whether it is expected to disintegrate or to persist. It may happen, therefore, that the discharge of the menstrual decidua is incomplete, and though haemorrhage ceases at the normal time, some cells that should have been discharged remain when the endometrium returns to its post-menstrual condition. The result will be that in course of time there is, by repetition of this process, a considerable hyperplasia of endometrium, which never returns to its normal state. The hyperplasia may proceed so far that the endometrium is brought to the condition we are familiar with as endometritis tuberosa vel fungosa. Long before the changes in the endometrium have advanced to this degree of hypertrophy there will be increased menstrual loss, becoming continuous in those cases where the hypertrophy is so marked that the polypoid or fungous overgrowth has practically the effect of a tumour of the endometrium.

It is in cases of this kind, where excessive menstrual loss, sometimes prolonged or continuous, is associated, not with gross enlargement of the uterus, but with hypertrophy of the endometrium, that I have found the marked degree

of induration of the ovaries described above. Where the induration is extreme, it seems impossible that the wonderful mechanism whereby the ripening follicle is conveyed from the deeper parts of the ovary to the surface can be effective. The dense fibrous tissue offers an insuperable resistance. The liquor folliculi is there, and its hormone is presumably absorbed, for menstruation goes on, but ovulation does not take place and no corpus luteum is formed. In the less-marked cases the follicle may get near the surface but fail to penetrate the surface layer of keloid tissue, though there may be parts of the ovary where there is little or no thickening and ovulation can take place normally in these parts. So it would seem to depend on chance, in these cases of patchy induration, whether ovulation is normal or incomplete.

I suggest that it is wrong to regard the sclerosis of the ovary as secondary to changes in the endometrium, and that such a view of the pathology of the condition is at variance with the teachings of physiology that it is the ovary which influences the uterus, and not vice versa. The primary change, I suggest, is in the ovary, and it consists of a failure of the regenerative process, whereby an excess of fibrous tissue, in other words keloid tissue, takes the place of the small fibrous scar that normally closes the stigma.

TREATMENT

It is a sound general principle of treatment to seek for the cause of the disease and remove it. In the type of case we are considering, however, there are obvious disadvantages in adhering too closely to this principle; for assuming for the moment that the condition I have described is the actual cause, we should, logically, be forced to advocate removal, on inactivation, of the ovaries—or, at least, of the most obviously affected ovary. There is the possibility, certainly, that in cases where the fibrosis is not too far advanced a course of treatment by thyroid extract might have the effect, as it undoubtedly has in some other situations—for example, in Dupuytren's contraction—of softening the excess of fibrous tissue and facilitating its absorption. Though I have no proof that thyroid extract can act in this way on the sclerosed ovary, I suggest that the beneficial results of thyroid therapy in some cases of uterine haemorrhage in young women may possibly be due to the fibrolytic action of thyroid extract. Most of the cases in which this type of haemorrhage is met with, however, are not those of young women. I have come across a few cases in young women where small patches of dense induration in an ovary are associated with dysmenorrhoea and considerable but not excessive loss, but much more often the patients are over the age of 36. At this age it is probably hopeless to attempt to deal with the cause of the disease, and removal of the ovaries is hardly to be considered. Nor am I in favour of inactivating the ovaries by α -ray treatment. It remains, therefore, to treat and, if this is unsuccessful, to sacrifice the innocent partner in the pathological complex—namely, the uterus—to treat the symptoms rather than remove the source of the disease, because almost any sort of ovary is better than none at all. I believe that on pathological grounds hysterectomy is the best treatment in all these cases, but there are other factors to be considered besides pathology.

The uterus deserves and receives more consideration than the appendix or gall-bladder. Therefore one's first thought, as regards treatment, is either curetting alone or curetting followed by radium. I think curetting is worth doing, not only for its diagnostic usefulness, but because the removal of hyperplastic endometrium is generally followed by temporary improvement, and occasionally the relief is permanent. The curette removes the

excess of endometrium which should have been removed by the disintegrating effect of menstruation. As the accumulation of menstrual decidua, due to ovarian disease, is generally a slow process, it may happen, in women approaching the menopause, that relief from symptoms after a single curetting lasts until the menstrual function comes to an end.

In many cases, however, and probably in most, the relief obtained by curetting is only temporary, not more than a few months. If serious haemorrhage recurs after a single curetting I believe hysterectomy should be performed. All due consideration has been given to the peculiar position of the uterus, and it should no longer be regarded as differing from a diseased appendix or gall-bladder. It should simply be regarded as a source of ill-health and perhaps danger, and should be removed. I would go further and say that, even apart from ill-health and danger, if her uterus has become a nuisance to a woman, on account of losses prolonged and severe enough to interfere with her enjoyment of life and with her career if she is in business, even though the general health may not be greatly affected, hysterectomy is justified if curetting has failed. I have no experience of radium in these patients, though I should be inclined to use it if hysterectomy is, for some reason, inadvisable. In parous women without gross enlargement of the uterus there is a good deal to be said for vaginal hysterectomy in this type of case. Though the operation is undoubtedly more difficult, I believe the shock is less, and the period of convalescence shorter.

In conclusion, I should like to refer again to Mr. Wilfred Shaw's valuable paper on uterine haemorrhage,¹ and especially to the section on metropathia haemorrhagica, and to criticize, not his findings, pathological or clinical, but certain of his deductions. He refers to the association of a polypoid condition of the endometrium with atrophic ovaries, one of which is generally cystic. He regards the hyperplasia of the endometrium as evidence of over-activity of the endometrium and the shrunken condition of the ovaries as evidence of diminished activity of the ovaries. He concludes that the polypoid endometrium reacts on the ovaries to produce an inhibition of follicle ripening, or of ovulation, or of full formation of the corpus luteum; the ovaries, in turn, responding by producing continuously a toxin, identical in type with that causing the disintegration of the premenstrual endometrium in normal menstruation. And he regards as proof of the inhibitory action of the polypoid endometrium the fact that, in some cases, curetting is followed by a return to normal menstruation.

In criticism of these findings I would say: (1) We have strong evidence that the ovaries control the uterus, but none at all that the uterus can influence the ovaries, or that the uterus has any action except to react to hormonal signals from the ovaries. (2) Hyperplasia of the endometrium is not evidence of activity of the endometrium. It is much more reasonably to be regarded as evidence of a defect in its response to ovarian signals, or, equally logically but more probably, a deficiency in the hormone itself, either in quality or in quantity. The thickening of the endometrium is, I maintain, to be regarded rather as a failure of premenstrual endometrium to be completely shed than as a true hypertrophy of endometrial tissue. (3) Curetting acts by removing the excess of endometrium, and in that way imitates the action of the corpus luteum, or of whatever hormone is responsible for disintegration. As normal ovulation may be possible in unaffected parts of an otherwise diseased ovary, menstruation may go on normally once the endometrium has been made smooth by the curette. (4) The size of the ovary is no criterion of the activity of the function of ovulation. The presence of a ripening follicle will ensure the first part

of the menstrual cycle—namely, the thickening of the endometrium. Incompleteness of ovulation—that is, failure or delay of the stage of corpus luteum formation—will be followed by incomplete disintegration, and consequent retention of degenerate endometrial cells. In some cases I believe this incompleteness of ovulation is due to the dense fibrous (keloid) covering of the ovary.

References to the connexion between pathological conditions of the ovary and menorrhagia are not infrequent in the literature; the earliest of which I am aware occurs in a paper by C. J. Bond,² in which the connexion is very convincingly established.

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THE INTERPRETATION OF PAIN IN INFANCY*

BY

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Of all the symptoms from which man suffers none is so insistent of recognition as pain, nor is any of greater value to the doctor in helping him to make a diagnosis. The adult is in the fortunate position of being able to give an accurate description of his pains, and to indicate their locality with sufficient precision. The young child, once he has acquired the power of conversation, can also tell us when he feels pain, although a fuller analysis may be beyond him, as, for example, when he complains of headache but localizes it at the umbilicus! Often in the baby the presence of pain can only be suspected after careful observation, and if a parent is convinced that a child is suffering in this way it is generally foolhardy of the doctor to cast such testimony aside. By taking a careful history, followed by an attentive watch and thorough examination of the infant, the characters of its pains and the site of their occurrence can be determined.

CHIEF EVIDENCES OF PAIN

The commonest effect of pain on a baby is to make him cry, and much help is to be gained from a study of the characters of this expression. Pain is by no means the only cause of a baby's crying; hunger, cold, a soiled napkin, or simply a state of boredom, are also contributory factors. It is not until the child exercises his will that crying due to temper makes its appearance; this is a cause which is exceptional in those under 6 months of age.

The features which should always be noted are whether the cry is intermittent or continuous, whether it is a piteous wailing or a short, sharp scream, and whether it occurs at any particular time of the day or night. Does it bear any relation to the times of feeding, being relieved or aggravated by food? Does it cease after the belching of a good deal of wind? The history may show that the pain only accompanies certain physiological performances such as sucking, micturition, or defaecation. Under the stress of severe pain the infant may not cry because he is holding his breath, as frequently happens in severe colic.

The baby's posture during the bouts of pain should be carefully noted. It may be that the hips are flexed to bring the thighs up on to the abdomen, while the arms may be bent, with the elbows firmly pressed against the sides. In other cases, the infant may become rigidly extended, with arched back, so that he supports himself

* Abstract of an address delivered before the Hampstead Division of the British Medical Association.