coalescing or amalgamation of these nodules to form larger and more invasive growths. Then would follow the secondary changes in the muscular walls of the uterine or ovarian arteries, or both, to which I have elsewhere drawn attention; the thickened tissue causing such an amount of obstruction to the flow of the arterial blood as produces increasing tension, and therefore demands increasing strength on the part of the arterial circulation to overcome this abnormal resistance. Then this increasing arterial tension will account for the tendency to ovarian disease, and the increasing obstruction to the blood supply will account for the degenerative changes, which are so frequently found in the fibroid growths themselves; and finally that the ovarian disease is most common when the fibroid growth affects the upper part of the uterine body, that is to say, the area into which the ovarian arteries run, whilst ovarian disease is, for the same reason, either less marked or altogether absent when the fibroid change chiefly or entirely affects the lower half of the uterus.

It follows from this theory of the genesis of fibroid growths that they may be found in any part of the uterine wall, as indeed is well known to be the case. Thus they are usually divided in text-book descriptions into three classes: those that are *sub-mucous; interstitial;* and *sub-peritoneal;* the first class being those which project into the cavity of the uterus; the second variety being those which are embedded in the wall of the organ; and the third including those growths which are projected out under its peritoneal covering. For clinical purposes, this is a useful and practical classification; but it is well to remember two facts. First, that it is comparatively rare in any woman over forty years of age to find only one solitary fibroid growth, whatever its position may be; and, secondly, that the position of the growths varies from year to year and, perhaps, even from month to month. I am inclined to believe that, in the great majority of cases, if not in all, myomatous nodules arise deeply in the substance of the uterine wall, and that it is merely a mechanical factor which determines whether the growth will move inwards, or outwards, or remain in its original position. It would be, indeed, only in accordance with a well-known physical law to argue that fibroid growths must move in the line of least resistance. Such a growth at first, then, is enveloped in a muscular wall, amongst which it is more or less of a foreign body, and the more it takes on a fibroid character, and the harder it becomes, the more must it act as a definite local irritant. It will then effect one of two changes; it must either cause similar degeneration in the muscular tissue around it, and thus become the focus of a continually increasing fibroid growth, or it must yield to the muscular compression set up and maintained by its constant irritant effect, and be squeezed either in, to the uterine canal, or out, to the external surface of the organ, whichever may be the nearest to it; or, in other words, towards that part of the uterus which has, at first, the least muscular wall and resistance between the growth and an open cavity. If, in short, the myomatous nodule first forms near the uterine canal, it will tend to become a *sub-mucous* growth; if it forms nearer to the outer surface, it will tend to become *sub-peritoneal*, or be forced between the layers of the broad ligament on one side or the other; and if it happens to be originally placed almost exactly in the centre of the wall, with an equal strength of muscle on both sides and above and below it, then it will tend to increase in size in that position, until in due course it approaches sufficiently near to the internal canal, or to the external cavity, to determine the direction of least resistance in its future movements. Whether this theory be true or only plausible, it, at any rate, explains every condition with which we are familiar upon dissecting uteri containing fibroid growths of varying sizes and in different positions in the organ. There remains another and very important consideration from the pathological standpoint.

I have often observed that when patients, who have had more or less large fibroid growths either in the anterior or posterior uterine wall, have become pregnant, active changes appear to be set up in the tumour itself which appear to me to be capable of a simple and common-sense explanation on the theory I have just advanced. If such tumours are of moderate size, say that of an ordinary orange or smaller, neither pregnancy nor parturition are, as a rule, materially interfered with; but when the latter is completed, definite changes seem to occur in many instances. In some, the tumour appears to increase considerably in size; in others, again, it seems almost to disappear; and in yet a third class, degenerative changes are induced in the substance of the growth, to which reference may hereafter be made at greater length. When an increase in the growth takes place, it appears to me that this is easily explainable on the supposition that the greatly enhanced blood supply of the organ during pregnancy increases the nutrition of a soft myomatous tumour, and therefore leads to its rapid growth and development. On the other hand, the greater pressure exercised by the more active muscular wall upon a myomatous mass can easily be understood to cause its compression, and thus a more or less rapid shrinking and diminution in size. And in the third event, the increased pressure of the greatly increased muscle, combined with the disturbance in the usual blood supply to the tumour, provide at once both the causes which are generally recognised as tending either to the death or the degeneration of new growths. In any case, therefore, it may be asserted that pregnancy causes more or less active changes in any fibroid tumour contained in the gravid uterus.

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