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ON PATHOGENESIS OF ENDOMETRIOSIS

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THE PATHOGENESIS OF ENDOMETRIOSIS

(A historical-critical treatise of the discovery by Freund and Recklinghausen to the traumatic theory with extensive discussion)

(Following is the translation of an article by Dr. Harald Petri, published in the German language periodical Zeitschrift fur Geburtshilfe und Gynakologie, vol 138, 1953, pages 16-34. Translation performed by C. L. Lust.)

A historic-critical treatise of the various theories on the pathogenesis of endometriosis is very appropriate now because very little doubt exists about the clinical picture of the illness. Symptomology, diagnosis and therapy are largely well characterized, especially so since the last report of Bonordon. The present pathogenic view of andometric heterotypes is established and is associated with no uncertainties. Now and again one does hear the serosaepithelial metaplastic view, but generally the view is accepted of implantation-genesis, for which Philipp and Huber showed via cuts through the andometrically altered tubes and further proved the hematogenic embolic way. This has been referred to since 1937 as traumatic genesis.

It is therefore a stimulating task to elucidate the historic developments of the pathogenic views of the discovery of "Adenomyome and Zystadenome of the uterus-and tube change" by Freund and Recklinghausen and up to the present. I have already referred to this in "On the traumatic pathogenesis of Andomet" in Z. Geburtsh (1949) vol. 131 #3, page 268.

Seven theories evolved to explain the appearance of andometric proliferation: 1) dysontogenetic theory of Freund and Recklinghausen about fetal organs; 2) vessel-endothelial theory; 3) lymphogenetic metastosis after Halban; 4) derivation of serosaepithilium (Zoelo epithelium) from metaplasia (Meyer, Lauche, Tobler, Hueter); 5) implantation of Andomet fragments via retrograde transport (Sampson); 6) Philipp and Hubers implantation theory with source of interstitial tube constituents and their hematologicembolic view; 7) traumatic pathogenesis (Petri).

Freund and Recklinghausen believed (1891-1896) that Andomet was a dysontogenetic phenamenon. This was shortly after the first report about this topic. Under certain conditions fetal organ rests should proliferate in the adult. Kidney tissue was thought to be especially important. The authors differentiated among three groups; Adenomyomen, which referred to the fetal kidney; and two others Wolff-duct and duct of Mueller. The position of Adenomyosis and the dorsal wall of the uterus and on the dorsal side and outer layer of the tube angle the authors contend that a crossover occurrs between duct of Mueller and the fetal kidney. In this way these latter two parts could merge. The tube endometriosis lateral to the tube angle they saw as a destroyed portion of the kidney system. They further speak of a paroophoral andonomyiosis of the uterus. They recognized inguinal endometriosis

and connect it with the capsule of the fetal kidney. From the lower angle of the Wolff-body a band stretches to the inguinal canal, which is known to become the Gubernaculum Hunteri (responsible for descending of the testicals) in men and the Liq. ovari and Liq. notundum uteri in women. The liq. rotundum, inserted outside on the outer wall over the tuberculum pubicum, can drag along the Wolff-body from the kidney tissue, and they can then serve as a starter for new formations. These authors also connect the endometric proliferations from the membrane of the Mueller duct. This is also clearly reproduced in "Pathologic Anatomy" by Kauffmann. Recklinghausen also mentions retrocervical adenomyoma, which he feels are from seroscepithelium. The authors assumed that fetal kidney remains react the same in the adult female in the menstrual cycle as the normal endometrium. Others followed the views of these authors. Pick believed that peritoneum of mature women has the ability to proliferate due to endometric infiltration from fetal tissue remains. Franque describes an endometrosis retrocervicalis and explains the proliferation either directly due to the Douglasserosa or from remains of the duct of Mueller. A similar view is held by Lahn. He sees the reason for the proliferation because of a dysfunction of the ovary and postulates a hormonal theory. R. Meyer referred to this view as a "fashionable observation". Kanther believed a tumor in the liq. rotundum is the germinal layer of Mueller-duct epithelium. Sulzer supports the dysantogenic zoelomepithelial histogenesis, especially the influence of ovary-hormones, for intraabdominal and other endometrioses. Even Seitz noted that internal endometrioses could be caused by residues of the duct of Gartner. Schaerr and Scheidegger also believe and onet cannot be cleared up yet unequivocally; a fact which is similar in many ways to the germinal kidney theory. R. Meyer stated that he did not agree with the hypothesis presented by the discoveries since neither macroscopic or histological tests have produced unequivocal findings. Extensive developmental studies now lead one to reject the kidney theory.

The so-called vessel-endothelial theory has always played an unimportant role. When the germinal-kidney theory became under fire, new explanations were sought feverishly. This "new-found" theory was based on the fact that endometric tumors contain many blood vessesl. The fact that this was well grounded for andometrioses was not known widely at that time. This theory further stressed the dependance on ovarian hormones. Manzi thought he had evidence against the new theory. He made transplants from uterus tissues into veins and also injected ground-up membranes into arteries. He always obtained negative results, therefore he said that no basis existed for epithelial elements from uterus in the renalendothelium. Spiritos's work was also inthis vein. He placed fine elements of membranes into the peritoneal cavity of rabbits. He did not obtain cytologic or adenomyotic formations. The membranes were changed into a fibrius-like mass infiltrated with leukocytes. In this way he meant to prove that endometric particles in the blood and lymph canals were not capable of developing further; and that they degenerated which then made a hematogenic or lymphatic metastasis impossible. He found that the tissue placed in the peritoneum implanted itself well and proliferated. According to Spirito the peritoneum and the genitalia are excellent places for proliferation to occur.

These experiments also cast doubt on the lymphatic genesis, even though Halban, its originator, was well respected. He conceived the expression: Hysteroadenosis metastatica! He meant that groups of cells loosened themselves within the uterus and were transferred via lymphatic channels. The cells, however, retained their activity. Halban's theory was not accepted by Mastitz. Seitz was also opposed; he thought that a passive transfer of endometrium in lymph ducts was important. Halban's work was later generally not accepted. It was then emphasized that a close histological relationship was never established between tumors and lymphatics for this disease; furthermore in lymph nodes no endometric tissue has been found.

Today we must say that the thoughts of the doubters of the vessel-endothelial and lymphogenic theory lacked only histological evidence. They were apparently on the correct path. Their theories have to be recognized today with some reservations; more about this later.

The thesis of R. Meyer represents an important turning point. He showed that all heterologous epithelial proliferations against the body were common, and that the special hormonal influence and sexual related special forms of paritoneal-epithelium heterotropy do not lead to their exclusion from independent heterotopies. He thought that the proliferations on the epithelial serosa originated there. He thought that inflammation alone was responsible for initiation. This is also important later for spreading and epithelial proliferation as well as the nature of the stroma. The specific hormone influence of the ovary is not the cause of the growth, but is the cause for the peculiar, special, endometric nature of the membrane in the proliferating tissue. R. Meyer said that the cause of proliferation is solely inflammation. R. Meyer discredits Sampsons retrograde implantation-theory as an "adventurous explanation". As for the tube endometriosis, he said that the accumulation is not likely, especially since Novak showed that the ostium uterinum tubae were often much too small to pass these particles. Via inflammatory metaplasia a heteroplastic membrane results at these places. This heteroplastic, endometric condition of the tubes has no practical value in the Meyer's view. The ovum will still implant itself in the tube of its designation when the time is ripe. I pointed out in 1937 that a tube, whose wall is the same as the endometrium, the normal growth implantation dodium for the ovum, was in effect an unchanged tube (Philipp and Huber). Endometriosis according to Meyer originates primarily from the serosa; the causative factor is inflammation and only later the influence of the ovarian secretions. This view became widely accepted, because it was felt this was an enlightened solution of this problem. Vassmer didn't believe on transfer of uteralmucosa in laparotomy-grains, but thought the origin in normal serosa. He also spoke similarly of andenomyosis in Douglas and generally refers to the frequent inform epithelial dystopy in women. R. deJosselin de Jong believed all proliferation to deviation of the Zoeloepithelium and thus comes closer to the primal-kidney theory because he believes these divations are capable of forming Mueller-duct type tissues. Stueblor and Haeuber decline Sampson's view. Ovarian hematomas are considered formations on the bottom of the adenofibrosis which did not originate via implantation. of endometriosis, but surface epithelium of ovary itself. Artusi discusses

a non-uniform causal genesis and dicusses the cause of inflammatious, infectious, toxic, malformed, and hormonal metobolic. Fischel supports Meyer and emphasizes the derivation of the peritoneum and mucosa from the same germinal layer. He talks about hormonal dysplastic irritations. Berkheimitsch supports Meyer. Ochlecker classifies Meyer's view under the Lauches' modification. The proliferations start in the peritoneum. The sweat-gland in this area alter themselves into tumor like fibromycoadenomas under the increased ovarian hormones. The question of etiology for vesiculat endometriosis he does not resolve: Perhaps one can assume germs or fetal organ residues. Kitai emphasized the common genesis for extra -+ intraperitoneal hord in the sense of Meyer. Seitz assumes the serosa epithelial theory for endometriosis. The parts from the zeolomepithelium also form a endometric character in the post menstrual life of a woman.

Heim believes in the serosa epithelial theory. The heterotopes arise on the spot from mesenchymal tissue elements. He thought on the mesenchyme in the region of cloacal cut of the earlier embryonic zeolom cavity. With Heim a new era of Meyer's theory begins; now all heterotopes can be explained. In recent times the very rare extremes - further endometriosis - can be explained with an expanded Heim theory. Mankin, Mavratil and Kramer and Sictl could find no explanation (1935-36).

At the beginning of his work, Sampson's view was not accepted. The serosa epithelial theory was not tenable based on the personality of the originator, etc. Sampson was bothered by the fact that endometriosis interna, a growth of normal endometrium, and the externa (equivalent clinically, physiologically and anatomically) showed originals in the same way. He sought to find a connection between the endometriosa externa and normal endometrium. He concluded that the adeno-fibroses that are not in contact with membranes can originate from endometric particles. These particles reach the peritoneal cavity via the tubes during menstration and become implanted there. It was essentially the theory of menstrual retrograde progression. This he thought explained endometrosis in mature females, while Meyer's theory was the reverse. Why an inflammatory heteroplasia does not appear in young girls and old women? It comes from the uterus if any normal drainage is blocked. It flows back into the peritoneal cavity. Borsten also causes the seeding of menstrual endometrium on the surface of ovaries. He said the menstrual blood contains viable endometrium which is capable of implanting. The peritoneum and ovum are particularly suited. Sampson further postulates whether gynacological operations or investigations could injure the membranes of the uterus (forceps, ventrofixation). Forceps could relocate material so that it could develop into endometric heterotopy. I myself substantiated this phenomenon once in the uterus. Also rests of suture material could be demonstrated in the mucous endometriosis (not to be confused with foreign-body granulomas). The teaching of operative transfer and seeding brought Sampson recognition. He is today still recognized as a pioneering spirit in this area. Wielod recognized retrograde transport of cellular material in menstration. Rosenstein expounds the view of implantation of scar endometeri. Christopher observed an inguinal endometriosis and thought

that normal endometrium had been transported via tubes to the stomach wall and reached the inguinal area. Katz and Szones saw the cause in the tubes. Rieck said the proliferations are dependent on the cyclic conditions of the ovaries, the hormones are specific and affect no other membrane in cyclic fashion. It concerns implants of uterus. Rieck said, "Without endometrium, no endometriosis." Baer succeeded in seeing free uterus-mucous-membrane in the lumen of tubes during operations (objective demonstration). Frankl believes in mechanical, genetic moments for internal endometriosis. Harbitz thought that implantations occur after hysterectomy during early pregnancy. Albrecht believes seeding. Dietrich-retrograde movement via tubes. Hackermann observed a scar end after perfubation. Rieck and Hueter saw scar end after extra peritoneal cuts.

Sampson received a big boost in his teachings when R. Meyer supported him. At one time Meyer called his ideas "adventurous"; now he says it is right. He declined the genesis of the serosa and believes the implantation via the tubes (of transported uterus membrane). He still believes his serosa epithelial theory for extraperitoneal endometriosis. Vogt supports Sampson. He notes that the formation of the illness could also be of importance for the artificial abortions. This may be why endometriosis is more widespread than in Germany. He calls it a "cultural disease". Molinengo recognizes the implantation theory. Sauer does also. Seitz decided partly for Meyer and partly for Sampson, and partly for Hulban's kidney theory. Joseph also believes implantation for bladder endometriosis. Haselhorst believes Sampson. In regards to scar endometriosis he decides for transfer or invasion. The scar disease appears only after gynocolic operations, after the uterus was opened, or a tube was excized. Scars don't always heal well in the uterus and sometimes sores occur and membrane-pieces are embedded into surrounding tissues during uterine movement. This is active transport. Gland epithelium may also proliferate along the fistulas by itself. This may be invasion as may occur in endometriosis interna. After abrasions a passive transport of endometrium into myometrium must be considered. Haselhorst recognizes three types of transfer: invasion, implantation, transplantation. Matyas reported an endometriosis interna of proliferation of endometrium in the musculature. This may go through the peritoneum and can lead to various heterotopes of the cavity. Ottow observed statistically that of 353 cases of ventrofixation operated as per Leopold-Czerny, 16 adenomyosis occurred: of 1000 other cases of gynocologic operations only one (1) endometriosis occurred.

Experiments in animals supported Sampson. Fuchs, Harbitz, Zaleckd are to be mentioned here. Harbitz made endometric transplants (autotransplants) in rabbits and always obtained positive results. Fuchs had one negative results. He did not get implantation in his one case. He did observe that in castrated animals the transplants did not take. The well-known trials of Fuchs and Schmidt of monstration after total exterpation are widely recognized.

R. Meyer's later view of the recognition of implantation in IP heterotopy but not for extra paritoneal endometriosis was seriously jeopardized with scar disease, which is extra paritoneal. This created an extremely infavorable picture since much confusion existed here, since operations

in the peritoneal cavity without involving genitalie epithelial scar disease never develops. Sampson's view is also on solid ground in microscopic examinations. Meyer should find microscopic evidence of proliferation and differentiate between normal serosa and cyclic tissue. If operated on in dysmenorrhoic difficulties one finds completely formed endometrioid tissue. Such a metaplasia cannot be formed overnight according to Sampson. We definitively find the beginning of endometriosis around 25 years. The further removed from the uterus, the fewer (seldom) the endometriosis. The tubes themselves and their immediate environment are most often involved.

The discussion up to now, although not exaustive, I have presented the history of endometriosis research in its broadest aspects. It seemed useful to compare side by side the writings of several of the older authors, especially if their contributions are still recognized. Now I should discuss the "traumatic" theory, which I first reported in 1937 before I had knowledge of the epoc-making work of Philipp and Huber (1939). To start: the implantation theory of Philipp-Huber, I can probably be short here since I assume that most experts in the area are very keenly acquainted with many details of this. In Zbl. Gynack. vol 1+9 (1939) Huber and Philipps reported histological sections of tubes. Over 20,000 seral and stepwise sections provide the evidence for a connection of adenomyosis tubae and Endometrium corporis. Both authors are displeased with the Sampson theory. They admit that a small part of the endometriosis are free in the intestitial spaces of tubes. These represent women with intrauterine problems at admission. This may have been effected by mechanical transportation via various operational procedures. This explanation is still fraught with difficulties had it not been for a fortuitous occurrance. They found in their sections, pictures that clearly presented evidence for a hematogenic transfer. Endometric tissue reached blood vessels and was carried off. To implant these endometric particles a minimum level of hormone is needed. In the old literature all cases were represented by a cessation of pregnancy (abortion). It developed neither because of proliferation in the uterine region or because of implantation. In the remaining 80% of cases, endometric tissue is transported away and implanted. Sometimes heterotopic colonies result in the peritoneum without affecting the interstitial parts of tubes. The normal carrying path is as always. Spread may occur via hematologic ways. A lymphetic spread is very likely also particularly in light of modern theory and research.

During a critical review of the total endometriosis literature, I recognized in 1937, that Sampson's implantation theory had no general validity. Above all, I recognized that eteologically a trauma in a very general sense must be assumed in order to bring the various disease-pictures into a common frame work. I did not believe the so-called far-distribution; the Huber-data was still forthcoming. On this theory the postulation that the desquamous, menstrual endometrial residues are starters for heterotopic proliferation was difficult to understand. However, based on experimental work of Fuchs, Schmidt and others, I recognized the implantation theory, also evidence of endometric growth in laparotomy scars after operations on the uterus or tubes. Here intact, viable tissue was transferred, grew and reacted as the endometrium. Workers prior to

Sampson repeated this argument much too little. The generally accepted post operative implantation appeared to be the bridge to all other heterotopies. Thus as in scar-endometriosis, the other endometriosis had to be explained similarly.

a) Into the group of operative-caused endometriosis a very common occurrance must be put; the appearance of intraabdominal foci after uterus perforations. This may lead to transfer directly, or if the perforation is not closed (not noticed or bleeding stopped) or purposely leaves it open, this can lead to secondary effects on the peritoneum. An explanation for instrumental transfer.

b) For the genesis of endometriosis interna, under heterotopy, I came to the following view (with Kleine). Active implantation based on disturbances. Normally the endometrium cannot penetrate into the myometrium. The myometrium is naturally resistant so that this resistance must be broken in order to allow proliferatory or regenerating endometrium to enter. Myometrial resistance is weakened by abrasions, scraping, etc. If interna breaks through serosa it becomes externa. In this way endometriosis can develop of intestine and bladder.

c) During operations heterotopy can occur via transplantation. During operative removal of chocolate-cysts, often the cysts burst during the operation and the contents spill out. In this way endometrium could come into the peritoneum, eg. during operations of the lower stomach.

d) In scar endometriosis, the scar must be injured. I proved this clinically in several cases. Of course, it remained unclear whether this was primary or secondary. It may be that accumulated endometrium, even if it is small, may represent a foreign body in the region and cause a loosening of tissue. This is probably similar in heterotopy. In fresh scar tissue a hernia may result. This is of general importance in this area of work.

e) For septum endometriosis, which is mentioned seldom, I saw the so-called traumatic-birth-damage as etiology much as was prescribed by Hilgenfeldt for another disease. In all septum endometriosis, births preceded it or gynecologic procedures were done. The birth or the gynecologic tests may cause rips in the membranes and this may lead to implantation, which can eventually become very prolific centers and then septum endometriosis.

At the center of my thoughts was always the work of a clear proof that endometriosis did not develop from menstrual desquamous rests, but that transfer occurred instrumentally in the general sense. Many factors are grouped together under the general heading "traumatic pathogenesis". My views of 1933 have been more firmly established because of Philipp-Hubers work about implantation. The so-called extraperitoneal endometriosis can now be explained readily. Meyer believed on metaplasia, Sampson expounded his own theory, Hilgenfeldt proved some work experimentally, but the implantation theory must be accepted. But, why are the peritoneal wall, navel and groin involved? It is an old surgical fact that foreign bodies of the peritoneum are expelled (perforate) in the area of navel and groin.

The wall is thinner here than in other areas. Endometriosis acts as a foreign body in peritoneum. I could prove some of these facets in experimental fact. In navel endometria, I found long stringy threads from the proliferation to the peritoneum. Hilgenfeldt and other authors also reported connecting tissue of this type.

The fact of implantation was firm; the manner was problematical. Only fresh, viable endometrium is transferred and implanted. Also endometriosis is recognized as a modern illness (cultural disease). Without doubt, the disease is more prevalent after 1900. Very likely post operative endometriosis and spontaneous metriosis were there earlier, but not to the extent of later. The methods of gynecology introduced new facets. For the same reasons the interna-disease was rarer. The distribution to arm, leg and thorax can be easily explained on the basis of hematologic transfer. As a precipitating factor trauma plays an important role. Abortion or interupting pregnancy may be one severe trauma.

The fact that our knowledge of the traumatic genesis of endometric heterotopy may be used prophylactically can be ascertained from the recent literature.

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