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(9)

ORIGIN — CANCER AND SARCOMA ONE; IDENTICAL IN CYTOLOGY. FINDING THE PURE CANCER CYTOLOGY, ONLY, IN A REPORTED CASE OF UNDIFFERENTIATED ROUND CELL SARCOMA. REVISION OF MANY CASES OF SARCOMA YIELD PURE CARCINOMA ON RESTUDY. DOES "CURE OF CANCER," 10-18 YEARS, POST OPERATION WITH NO RECURRENCES, THEN FATAL RECURRENCES, SUGGEST A DORMANT ABEYANT CHRONIC FORM OF CANCER — A BENIGN FORM OF CANCER? VARIOUS (30) CASES IN POINT OF DISCUSSION. THE CAUSAL DIMINUTIVE CANCER CELLULETTES, DORMANT OR ACTIVE, MUST BE NEUTRALIZED OR DESTROYED FOR ULTIMATE CURE. A FURTHER AMPLIFICATION OF THE SPECIFIC CANCER CELLULE AND ITS CONDUCT. TRANSMUTATION.

CELL DIVISION.

By
FRANK A. STAHL, M. D.
Rush 1887
Chicago

With Compliments of Author

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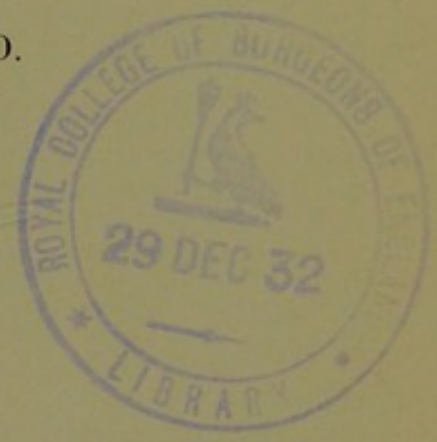

One Time

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Chicago, Illinois.

Contents of Previous Researches

The present publication is the seventh of a series of seven researches concerning the histology and physiology of the early ovum; 2-8th week.

They are in order of appearance as follows:

First—The Syncytium; (contents).

1. The syncytium wholly foetal in origin; (forensic)
2. No characteristic decidua in extrauterine pregnancy; (forensic).
3. The amoeboid-digestion of ovum environment function of the syncytium furnishing sole source of nutrition to primal ovum, except that of the minute gestation cyst and the vitellus pabulum.
4. Origin of first blood corpuscle and plasm in chorion.
5. An origin of cancer from uncontrolled proliferation of embryonal cells — primal and included.
6. Demonstrating Cohenheim's theory for the origin of tumors.
7. The Syncytiomata; benignant and malignant, etc.

Published in Amer. Jour. Obstet., 1902.

Second—The Crescent Erythrocyte; Chorio-decidual attachment; contrast in decidual cell and roving amoeboid syncytial nucleus-cells.

The difference between embryonal primal nucleus-cell and the regular cell as an epithelium cell or an erythroblast blood cell; etc.

Published in Amer. Jour. Obstet., 1906.

Third—The Crescent Erythrocytes in normal and abnormal blood conditions. Explanation for deformity of red blood cells in the anaemias.

Published, personally, January, 1930.

Fourth—Concerning Origin and Development of the Chorion, Amnion and Yolk Sac. The great importance of the Corona Radiata Cells. The Zona Pellucida Ovum Theory vs. the Zona Pellucidaless Ovum, the latter seemingly to enjoy popular embryologic opinion. Edgar Allen's multiple recoveries, 1928, of unfertilized ovules in human tubes — iconoclasting Zona Pellucidaless Ovum Theory, etc.

Published, personally, October, 1930.

Fifth—An extended amplification and résumé research concerning Origin of Blood; First Blood Corpuscle, First Blood Plasm, First Blood Space and First Blood Vessel. Blood corpuscle differentiation from the primal uncolored multinucleolated blood corpuscle to the non-nucleated red blood corpuscle, the erythrocyte, of maturity; i. e., blood corpuscle maturity, circa 8th week; the blood plastid of Minot. First blood spaces and blood vessels; The Three Blood Circulations; (1) the primal uncolored white blood circulation; (2) the first red blood circulation of the transient red erythroblast; finally (3) the permanent red blood circulation of the erythrocyte, etc. The uncolored multinucleolated nucleus-blood-corpuscle of the primal white blood circulation, is the primary blood cell in the blood; not the erythroblast.

Published, personally, March, 1931.

Sixth—Origin of cancer. The specific cancer cell of carcinoma, contrasted with the normal matrix embryonal cellule of primal ovum days, to 8th week; from which the cancer cell is directly descended. Cancer not epithelial. Sarcoma not connective tissue.

Published, personally, January, 1932.

Seventh — Origin — Cancer and Sarcoma one; identical in cytology. Finding the pure cancer cytology, only, in a reported case of Undifferentiated Round Cell Sarcoma. Revision of many cases of sarcoma yield pure carcinoma on restudy. Does "Cure of Cancer," 10-18 years, post operation with no recurrences, then fatal recurrences, suggest a dormant abeyant chronic form of cancer — A Benign Form of Cancer? Various (30) cases in point of discussion. The causal diminutive cancer cellulettes, dormant or active, must be neutralized or destroyed for ultimate cure. A further amplification of the specific cancer cellule and its conduct. Cell Division.

Published, personally, December, 1932.

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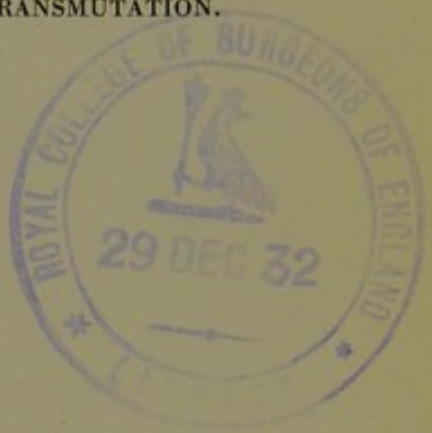

27 Plate Illustrations
21 Personal and Original
With Explanations

In "Origin of Cancer" the assertion is made that cancer and sarcoma are one; in origin; descended from the primal matrix embryonal syncytial cellule of primal ovum and subsequent days; identical in cellular form, identical in cellular cytology; similar in conduct of cellular divisions and similar in types, form and appearance in offspring of such divisions; the cancer and sarcoma cellules are similar in aberration of normal conduct in cellular division, normal evolution ceasing in proliferation; both cancer and sarcoma are similar in intense uncontrolled hyperproliferation; in both no further cellular differentiation into further normal cellular ultimates; both are similar in growth conduct, such growth being due to the amoeboid destruction and digestion of environmental contact tissues, either soft or hard; both are identical in replacement of destroyed and amoeboided environmental tissues by a new tissue characteristic of its own proliferation of cancer and sarcoma cellules; having no features or functions common to normal tissues; similarity in malignancy, similarity in malignant metastases; simi-

larity in diminutive cancer and sarcoma cellulettes, the cause of destructive metastases and recurrences; however perfect may occur removal or destruction of autosite cancer or sarcoma, until today, cure can only be positive when cancer cellulettes are neutralized or destroyed, recurrent metastases recurring even after ten years of apparent cure, 10 years (4) cases — 18 years (1) case; because of the potential powers in the diminutive cancer cellulettes remaining latent or dormant in the tissues during these periods of apparent cure; similarity in cancer and sarcoma termination, death of host and cancer due to exhaustion from malignant process.

CANCER NOT EPITHELIAL: SARCOMA NOT CONNECTIVE TISSUE. THE CAUSATIVE CELLULE OF CANCER; THE SPECIFIC CELLULE OF CANCER (AND SARCOMA).

Though today, origin of cancer (and s) has been traced to the primal matrix embroyonal syncytial cellule of primal ovum and subsequent days, popular opinion, however, seems yet to cling to the theory of "Transmutation of one cellule into another"; atypical proliferations, as epithelium into cancer cell, connective tissue cellule into sarcoma.

Many names of weight have inclined to this theory (Thiersch, Waldeyer, Adami and others).

And just here belong Anaplasia and Dedifferentiation, synonym terms covering practically the same thoughts.

A few excerpts:

"Sarcoma is a malignant tumor of mesoblastic derivation composed of rapidly multiplying cells resembling those of connective tissue."

"It is held by some of the best authorities that sarcoma arises through Anaplasia from the differentiated tissues. Jos. McFarland, Surgical Pathology, page 307, 1924."

Note: "The theory of the specificity of the three germinal layers is no longer accepted by embryologists. Wm. H. Woglom, American Journal of Cancer, 1931."

"Sarcoma a cellular tumor composed of anaplastic tissue of any of above (connective tissues) types. Jas. Ewing, Neoplastic Diseases, page 27, 1928."

YET?

Transmutation — Anaplasia — Dedifferentiation imply retrogression in cellular evolution.

A finished mature normal cell like an epithelial cell with its proliferating offspring; with identical normal characteristics in construction, form and function; in its maturity suddenly giving offspring, origin to a primal embryonal immature cellule, with entirely different characteristics in construction, form and function? A normal cell, formerly, always passively constructive, controlled in growth, now suddenly transmutes into a wholly different cellule, uncontrolled in growth; now characterized with the ens malignitatis (Teutschlaender) of the malignant cellule and hence destructive in function to normal life! A destructive reversion; Improbable.

Cancer (and s) is progressive evolution of a primal normal cellule, then aberration in the second stage of normal cellular evolution, proliferation; into the abnormal specific cancer cellule. No reversion whatever.

Then too, cancer cytology is always identical with mater cytology; transmutation cytology would always be wholly different to its mater cytology. Such transmutation theory would be direct antagonism to the law: "Like begets Like." The life courses of epithelium and connective tissue cells are diametrically opposite and antagonistic to those of cancer (and s). Cancer is not epithelial: sarcoma not connective tissue. Cancer is typical proliferation of the specific cancer cellule only, not that of another transmuted cell, as, an atypical proliferation of epithelium or connective tissue cellules.

While dwelling on the nature of cancer theories, it will be interesting also to mention that of the well known Gustave Roussy, Paris, who in the London Cancer Conference, page 15, 1928, writes in conclusion: "But above all, the embryonic theories of cancer have almost wholly lost their value in view of the acquisitions of experimental cancer."

Au contraire! it is just this primal matrix embryonal cellule descendant that becomes the instrument of cancer and its dissemination. Wandering throughout the tissues of the body, prenatal or postnatal; when wounded or otherwise injured, as by irritation, etc., this primal matrix embryonal cellule undergoes change, not in form, etc., as in transmutation, but change in cellular intrinsics, chemico-physiological, and in functions. It is then and thus that this irritated, injured primal cellule of normal matrix embryonal origin, is changed, aberrates in its intrinsics and chemics and becomes the active uncontrolled malignant specific cellule of cancer.

A normal embryological cellule gone wrong, riot, in the stage of proliferation, and continuing so; thereafter wholly uncontrolled in proliferation, ravenously amoebic and destructive to its environmental tissues and finally to itself.

In Amer. Jour. of Cancer, May, 1932, in the Abstract Division of current articles on Cancer Pathology, pages 531-537, other interesting and later theories as to cancer origin are mentioned:

1. Cancer is a constitutional disease.
2. Cancer cells of: increased cytoplasm, large nucleus with large nucleolus.
3. Cancer is not a cellular disease.
4. Cancer is not a cell problem.
5. Cancer is cell mutation.
6. Cancer is: a vibration out of harmony with its surroundings. (Rather fanciful).

To all these assertions of theory, reply, based wholly on histologic demonstration only, is:

1. Cancer is not cell; with increased cytoplasm, large nucleus with large nucleolus.
2. Cancer is cellule only, a nucleus-cellule of about 4 m. diameter, with multiple nucleoli, but without surrounding characteristic cytoplasm; control through Figs. 14 to 25.

In this series of theses on early embryological developments, the cancer cellule is shown descended from the primal matrix embryonal cellule of the primal and subsequent syncytium.

The primal matrix embryonal cellule of the new life, ovum, is descended from the corona radiata cellules of the ovule; the latter two cellules, the zona pellucida cellules and the primal matrix embryonal cellules form the great cellular connecting link between the life of the ancestor, ovule; and the life of its offspring, the ovum. The vital link which has ever interested the curiosity of the philosopher and his imagination; how explain the existence and formation of that link, via naturae, bridging, continuing the life of yesterday with the life of today.

Formerly embryologic theory had it that the all-important corona radiata cellules disappeared, died off, with the ovule in its descent through the Fallopian Tube; hence cessation of life of the zona pellucida cellules and cessation of the ovule; thus creating a void (theoretical of course and only) in the continuity of life between

the ovule and the ovum. But here the great error in that embryologic lethal conclusion. Those ovules were unimpregnated ovules, and naturally they underwent normal disintegration and lysis; and were either cast off or absorbed in materno. However, those ovules referred to, were unimpregnated ovules only, hence the great error in conclusion. Impregnation changes all those conditions and the corona radiata cellules are continued, revivified as it were, and continued on and over to the new zona pellucida-chorion of the new life, ovum; and thus, changed, by the ferment or hormone of the impregnation, they become the new, the primal matrix embryonal cellules of the primal syncytium; see Development of Chorion Ammion and Yolk Sac.

Further proof that cancer and sarcoma are one, identical, is shown in the fact that in all such cases, and however varied the details of picture of mixed cellular tissues, as sarco-carcinoma, carcino-sarcoma, osteo-chondro-sarcoma, the causative invading malicious cellule is one, and one only, the cancer cellule and its divisions, of seven forms—the cancer cytology; however caption may designate the process.

PROOF OF CANCER ANCESTRY.

The first step toward the establishment of this proof of fixation of ancestry and descent of the specific cancer cellule, is to show and prove the matrix cellule of the primary and subsequent syncytium as that ancestor. The matrix embryonal cellule is primary in time of cellule expression in the ovum. As has been explained, the matrix cellule is ovum, syncytial, extra embryonal in origin; not of the embryo proper. Cancer cellule is secondary in time of appearance to the matrix cellule, appearing first in and after the stage of proliferation. Here, in proliferation, the two cellules diverge in further parallelisms of growth and growth ultimates. The normal cellule continues on in its further normal characteristics of differentiation into normal growth ultimates and construction. The stricken, in proliferation running mate, the now abnormal or cancer cellule, continues on in growth with proliferation and hyperproliferation only, but with no further differentiation; hence destruction not construction.

These normal divisions and cancer divisions have been shown in detail in "Origin of Cancer, January 1932." They will be repeated in part here.

ILLUSTRATIONS TO SHOW NORMAL PRIMAL MATRIX EMBRYONAL CELLULE DIVISION AND MULTIPLICATION

Fig. 1. The normal double row vesicular multinucleolated nucleus-cellule of the primal syncytium 3.5-4 m; 2-8th week ovum; the ancestor from which the specific cancer cellule is descended; hence cancer cellules same in type.

Here are seen the normal embryonal types of the large round and medium sized cellules ready to break up into a cluster of infantile offspring, neo-cellules, small round cells, through their liberated nucleoli cellulettes, one offspring for every nucleolus (nothing like it in the epithelium), explaining their rapid fierce multiplication. Graduation in size is from the infantile, small round cellule not reached here but shown in other illustrations.

Here are the prototypes of the large round spinal cellule type of cancer and sarcoma; the infantile, small round cellule is the prototype of the small round cellule of cancer and sarcoma. Between these two is naturally the medium sized or transitional cellule; in cancer see Figs. 14 to 25.

The diminutive normal cellulette, the successor form of the just liberated nucleolus, in cell division; the pre-small round cellule form of the infantile small round cellule, is not shown in this illustration. It is first shown in subsequent illustrations in Fig. 7, also Fig. 8 (3). The cancer cellulettes are also shown in subsequent illustrations; see Figs. 14 to 25.

The syncytial nucleus-cellules quickly proliferate like their corona radiata forbears thus furnishing a fierce multiplication necessitated by further cellular growth.

The multinucleolated nucleus-cellule of the syncytium, Figs. 1-3-12-13, is an atypical cellule, being a vesicular nucleus only, with multinucleoli but without a typical cell surrounding characteristic cytoplasm, as seen in the case of a typical epithelial cell, or in a typical erythroblast blood corpuscle. In the epithelium or erythroblast cell, there is one nucleus with the usual mono-nucleolus, surrounded by a characteristic cytoplasm, as a rule; see Fig. 11.

The normal primal embryonal cellule, the matrix cellule of the syncytium; and the specific cancer cell, its offspring, on the con-

trary, are both multinucleolated. This as has been stated multinucleolated feature explains their fierce rapid proliferation. Compare this fierce activity of division and multiplication of the cancer cellule with the slow regular proliferation of the epithelial cell. Some difference in histologic and physiologic function. At times in the nucleus-cellules there seems a surrounding cytoplasm but this is irregular environmental plasm rather than a true regular cytoplasm.

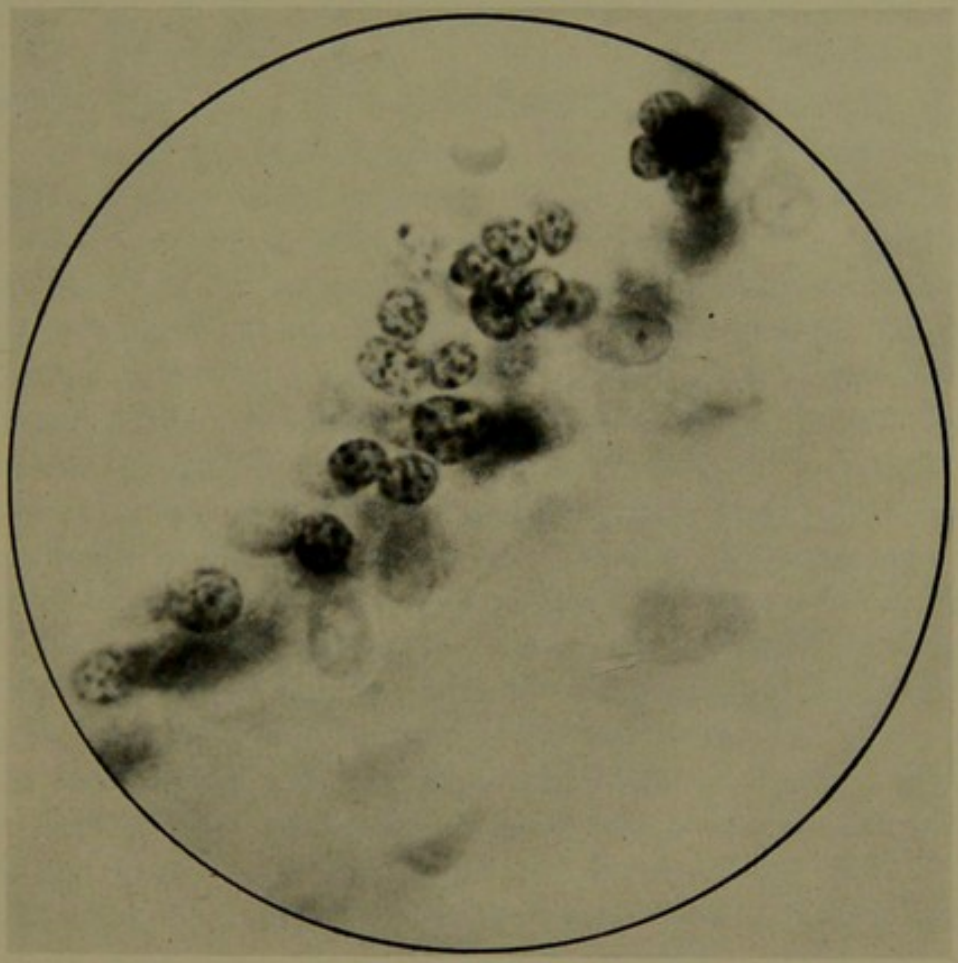

FIGURE 1

FIG. 1. The multinucleolated nucleus-cellule of the syncytium. An irregular embryonal nucleus-cellule only, without surrounding self limited cytoplasm as in a regular cell, like an epithelium or in an erythroblast blood cell. This is the type of first uncolored multinucleolated nucleus-blood-corpuscle; unhematinized; hence the term: Primary uncolored white blood circulation, where this uncolored, multinucleolated nucleus-blood-corpuscle is predominant in the blood stream; the white blood circulation continuing until about 6th-8th week. From Origin of Blood (Stahl).

THE NUCLEUS-CELLULES, OR PRIMAL CELLULES.

These nucleus-cellules, Figs. 1-3-12-13, of the primal and subsequent syncytium are the first active cellules met with and distinguishable in earliest ovum expression, 2-3 weeks growth; at earlier times tissual structures become quite indistinct for individual differentation. The term "nucleus-cellule" was hit upon for these syncytial cellules in these very early syncytial interpretations, as it seemed more correct and fitting to their intrinsic physical and functional conduct. It is cellule in that it is far smaller than the usual technical cell, as an epithelial cell; or the first technical cell encountered in the primal cytology of the ovum, the erythroblast, Fig. 11.

The diameters of the primal nucleus-cellules vary between 3.5 to 4m; that of the erythroblast between 7.5 to 17.5m. Interesting to note is that the diameters of (1) the primal nucleus-cellules; (2) the nucleus, sole, of the erythroblast; and the diameters of (3) the mature neonat erythrocytes, are about the same, 3.5 to 4m. in diameter, at 3-4-5-8th week of ovum growth.

Thus it should be recognized and be borne in mind, that the primal embryonal cellule is of ordinary nuclear size, with multiple nucleoli; or, a small cellule with multiple nuclei; but without characteristic surrounding cytoplasm. Showing the striking differences between the pure primal embryonal cellule without surrounding cytoplasm, and the maturer form of cell, as the erythroblast or epithelial cell, both these latter with a characteristic surrounding cytoplasm.

Division of these normal nucleus-cellules and division of the abnormal nucleus-cellules, the cancer cellules, through proliferation, are the same; hence these nucleus-cellules exist in both states and in ante-natal and post-natal life; ante-natal and post-natal cancer (and sarcoma) prove this assertion.

The primal normal matrix embryonal cellule is the type of the first wandering cellule, in the tissues of the body general, from the time of the earliest embryo; this type cellule continues as the wandering cellule type throughout all life, pre-natal and post-natal.

In what form do these primal cellules exist in post-natal life, that recognition may be made? They exist in the tissues of the body everywhere and somewhere.

Is the small round cellule of inflammation, repair and restoration, multinucleolated and without characteristic surrounding cytoplasm,

seen in post-natal life, especially in the process of tissue repair and reconstruction; as of wound reunion and repair; is that this cellule in post-natal life? As the descendant of the original primal constructive matrix embryonal cellule?

As remarked, these nucleus-cellules exist and flourish post-natal, for when aberrant, they are seen in all cases of cancer (and s), where they can easily and readily be seen and recognized as the aberrant normal nucleus-cellules, the cancer (and s) cellules.

As these primal nucleus-cellules are as old as Adam in their tissual origin and descent, histology and pathology may well be looked to for assistance in their recognition in post-natal life.

NORMAL AND ABNORMAL (CANCER) AMOEBIC DIGESTIVE ACTIVITY.

Fig. 2. Among the most interesting features this illustration of a so early 2-3rd week extra-uterine villus brings out, is the perfect discriminatory growth control feature, notwithstanding the intense amoebic activity; its maternal tissue antagonistic invasion for amoebic nutrient pabula; the unusual length of the villus and its offensive destructive amoebic invasional possibilities; yet with all the diverse intensive activities there is perfect growth control leading to normal metamorphotic ultimates.

Amoebic activity of the primal embryonal syncytial cellules, is the same in the normal cellules as in the abnormal cancer cellules. As mentioned, both proliferate alike through the primary stage of evolutionary metamorphosis, proliferation. Differentiation, the next stage, carries the majority of embryonal cellules on to normal ultimates.

In a few primal cellules normal development ceases in proliferation: there is no differentiation; these latter, the cancer cellules, continue existence but with increased cellular proliferation only.

Three varying expressions of amoebic activity in type, degree and results in metamorphotic ultimates, are seen in these illustrations.

1. The intense amoebic activity, as seen in this long extra uterine villus; yet normal in metamorphotic ultimates. Fig. 2.

2. The more passive amoebic activity of the intra-uterine villus; also normal in metamorphotic ultimates. Fig. 3.

3. The intense vicious amoeboid activity of the cancer cellules, to be seen in the cancer; abnormal in ultimates. Figs. 14 to 25.

CONTROLLED BY

4. Normal growth control principle guides the two former (1 and 2), to normal metamorphoses.

5. Absence of normal growth control principle, in the cancer (3) leads to destruction and death of both the host and the cancer.

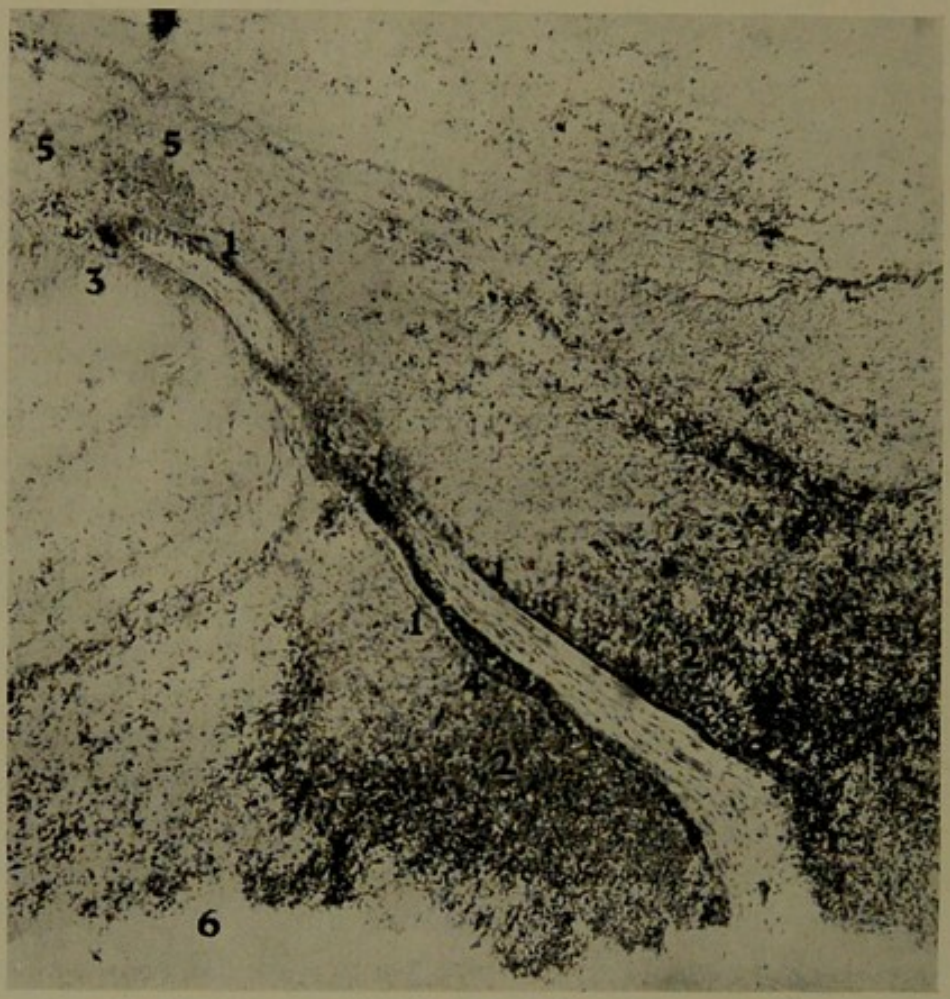

FIGURE 2

FIG. 2. A most interesting picture, an unusually fortunate find, of a longitudinal section of a long villus; from apex to broken off union with chorion; from another slide of this 2nd-3rd week extrauterine ovum; showing marked offensive invasion activity (denied by some). Note the rich inwandering and intake of nuclei and plasm, increasing from apex to ampulla; the unusually clear outline of a suggested nuclear flow; tumbling down and increasing in volume from the apex, like a great stream in and along the course of the villus; to empty at its mouth into the main stroma of the main body of the chorion. Hence now, 2nd-3rd week, already first blood-nucleus-corpuscles; already a first colorless blood plasm; the primal uncolored white blood circulation; quite before comparatively speaking, the later blood-islands, yolk sac, etc. (1) Syncytium. (2) Syncytial nucleoli proliferations conducting like tropho-blast into ovum environment. (3) Apex showing rich proliferations at (3) and (5). (4) Primal blood spaces in syncytium. (6) Chorion where villi were stripped from chorion in handling. Here in this example of active antagonistic amoebic, tunneling invasion of maternal tissues by the aggressive villi is splendid proof over against the views of: "Ovum tissues do not invade maternal tissues and aggressively so." Amoeboid activity controlled by normal growth control principle. In cancer growth control is absent, hence destruction.

Fig. 3. Another expression of this normal controlled amoeboid-digestion activity of environmental tissues by the normal syncytial cellules, 7-8th week, human ovum.

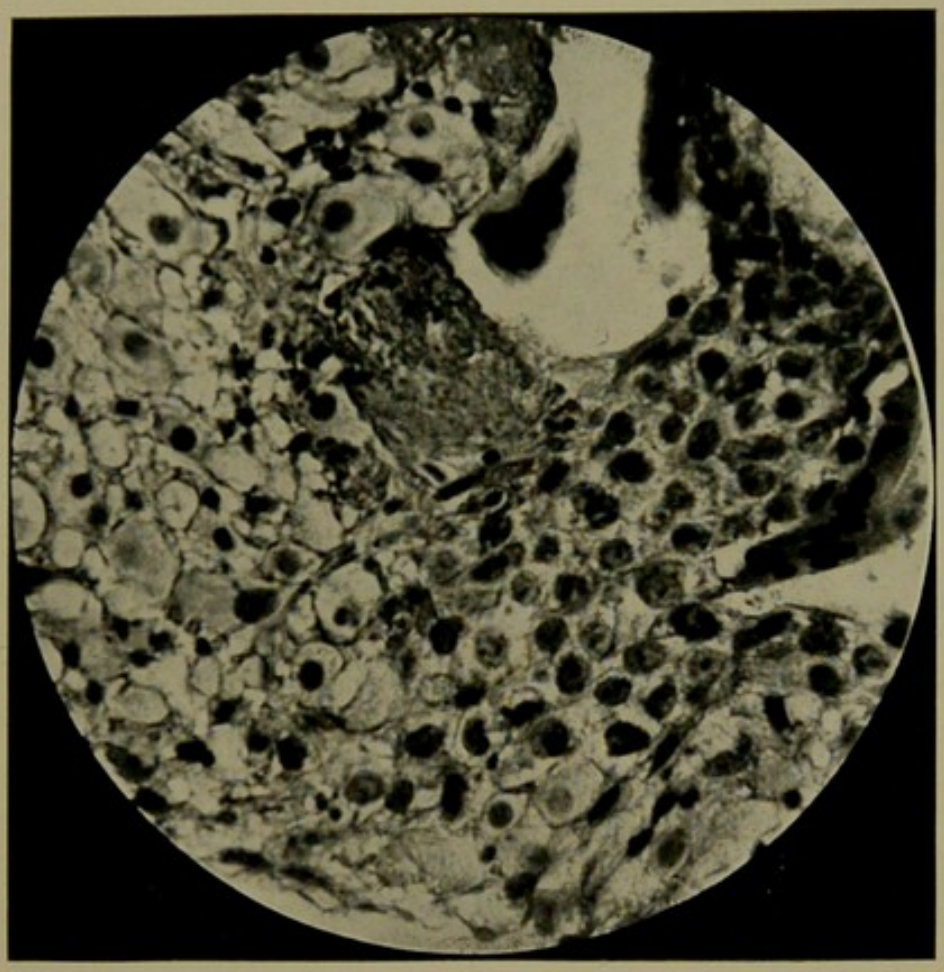

FIGURE 3

FIG. 3. To show amoeboid-digestive type of normal controlled conduct of syncytial nucleus-cellules in the process of the villus attaching itself to the decidua basalis, 8th week human ovum.

This amoeboid-digestive type of conduct is the prototype of the cancer amoeboid activity. In cancer it is uncontrolled, therefore so destructive and malignant to host tissue.

Nuclear proliferation tissue of syncytium well shown. Notice especially the invading proliferating unit is a nucleus only, there is no cell type to the invading unit. In contrast observe the perfect cell type outline of the decidual cell structure. In a previous article, the observation was advanced that there is no typical cell structure of the syncytium of the villi. This picture in itself argues most eloquently to the point in question and leaves no doubt that the invading structure is not a technical cell, but a free nucleus. Notice in the higher coloring of the nuclei, the suggestion that the nucleus has an active attacking corrosive breaking-down character — an amebic digestive function.

In this villo-decidual tongue where there is concentrated nuclear activity, notice the loss of decidual cell demarcation and of tissue. Observe, away from the tongue is seen the perfectly outlined decidual formation already fading because of the presence among them of wandering nucleoli rapidly growing into mature nuclei. The decidual fibrinous exudate is also slowly fading before the nuclear action and villus absorption. Here is shown very plainly the great contrast in histologic picture between the large passive decidual cell and

the active invading free nucleus of the syncytium. The maternal structure is characteristically cell-like in outline, with an apparent limiting plasm and membrane, possibly to increase the effectiveness of the cell as a protective agent against the encroaching nucleus of the syncytium. The embryonal structure is a freelance unit, a free nucleus only and not limited by cell plasm walls; most probably to increase its power of amebic action (1906). The nucleus-cell in some cases seems surrounded by slight plasm; but that is environmental not inherent or cytoplasmic.

Here is shown the manner in which an intrauterine villus attaches itself to the decidua serotina of the mother.

In attaching itself to the decidua the villus clings to the decidua by agglutination of its tip; then the cellules of the contact syncytium pour over into the decidua, neutralizing the large decidual cells, breaking down their passive defense, and amoeboid-digest the decidua as necessity of nutrition directs.

The normal amoeboid-digestive conduct of the syncytial cellules, in amoeboiding, here, the inflammatory exudate at the margin of the decidua; and also part of the decidua itself, to connect up with the maternal circulation, etc., is conduct analogous to the amoeboid conduct of the cancer cellules; only here in the villus attaching, it is controlled by the normal growth control hormone or principle permitting such amoeboid activity, for nourishing pabula to the ovum. But at the same time the normal growth control exercises a limiting check to this amoeboid-digestive activity.

In the cancer, normal growth control being absent, great uncontrolled hyperproliferation of the cancer cellules follows, with intense amoeboid-digestion of contact host tissues; and great destruction of such tissues.

Hence the cancer amoebic activity, once started as a rule, ceases only when it causes death of host and self from cancer tissue transformation and exhaustion.

Compare these normal aggressive syncytial cellules with the cancer cellules, and note in both, similarity in appearance and form and in both only, multinucleolated primal nucleus-cellules without surrounding characteristic cytoplasm.

RÉSUMÉ ILLUSTRATIONS OF TYPES OF NORMAL MATRIX EMBRYONAL CELLULE DIVISIONS.

Fig. 4. The primal multinucleolated nucleus-cellules of the syncytium, 2-8th week, human ovum; the matrix embryonal cellules from which the cancer and sarcoma cellules are descended.

The large; the medium or transitional; and the small round cellules, are here to be seen. The same types occur in the cancer and sarcoma.

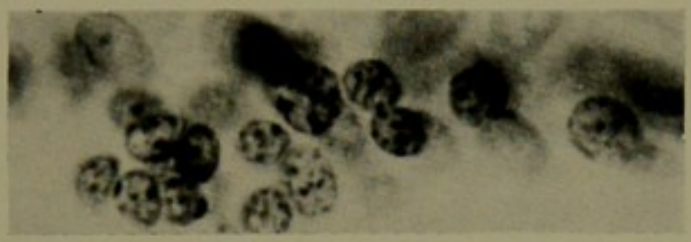

FIGURE 4

The primal matrix embryonal cellules, of the primal and subsequent syncytium. Prototypes of cancer cellules.

Note throughout these normal cellule types, they show a more refined, smoother, higher type structural appearance than their coarser cancer and sarcoma descendants; otherwise both the normal and the abnormal (cancer and sarcoma) cellules have the same physical appearances.

The descendants of the primal multinucleolated nucleus-cellules, identical in form, structure and potential powers, with their mater cellules, continue in the syncytium to maturity, placental delivery; see illustrations of full term villus with nucleus-cellules in the full term syncytium, there referred to as nuclear-nodules, formerly spoken of as cell nodules; see Shaefer in Minot, Kaibel and Mall and other histologies. Here Fig. 27.

It is physiologic for the syncytial cellules, the primal cellules, to wander as explained above, Fig. 12, from the syncytium into the tissues and vessels of the villi and chorion, and thus into the tissues of the body in general, in pre-natal life; see Figs. 1-4-5-9-11-12-13. Here they continue to multiply and to function, characteristically, as other cellules and cells do.

At birth, and in post-natal life, they are already present and flourishing as wandering cellules; they are found wandering among the tissues and cells of the post-natal body in general, where they continue to flourish as before.

That they are present in post-natal tissues as well as in pre-natal tissues, cancer and its cellules, identical in form and structure to their primal cellule ancestors, amply prove.

Fig. 5 from a 2-3rd week extra uterine villus; showing inwandered syncytial cellules into primal blood vessels of this early villus;

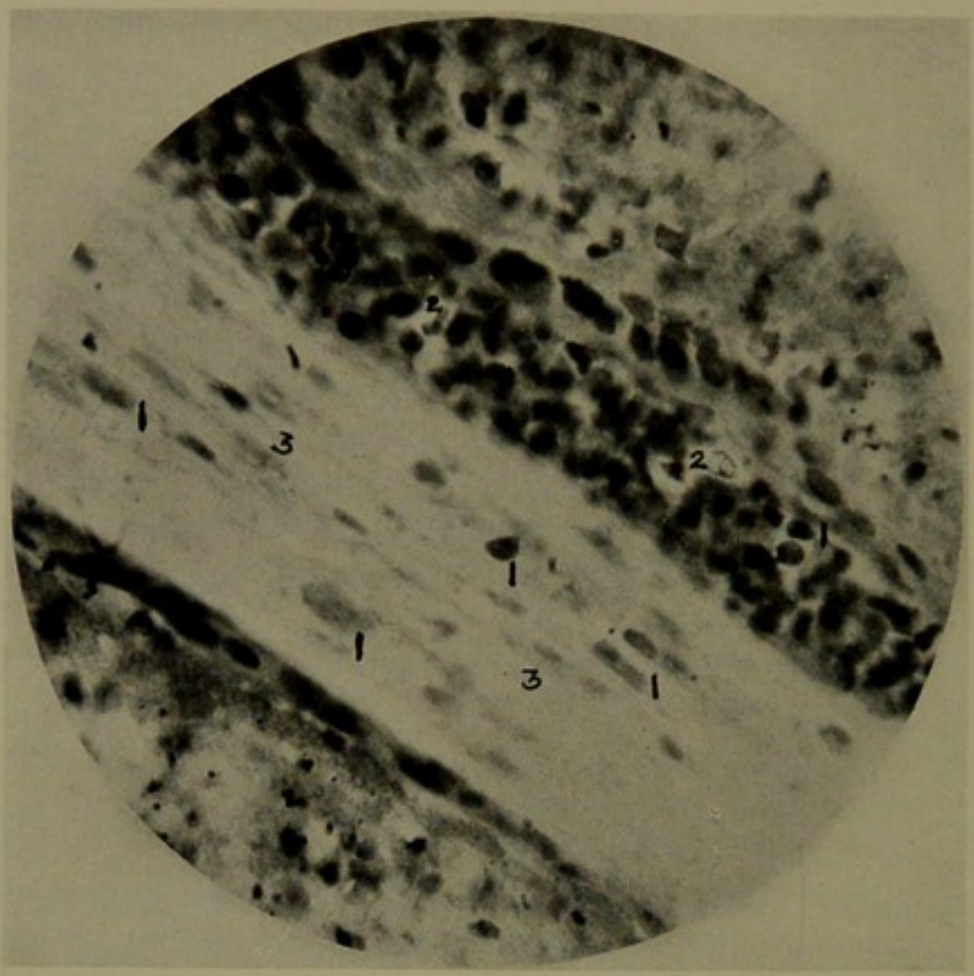

FIGURE 5

FIG. 5. Amplification of ampulla of this long villus, Fig 2, rich suggestions here of first primal blood spaces (2) and blood-vessels with first blood corpuscles (1); centripetal in conduct of flow. The large round cellule (1) and the spindle cellule (1) seen as blood corpuscles in the blood spaces of a 2-3rd week extra-uterine villus. Types seen in cancer (and s).

(1) The large round cell; also at (1) the elongated or spindle shaped cell; see Figs. 4 and 6. These types are of common occurrence in cancer.

Fig. 6. Normal cell division at 3rd to 4th week, showing (1) and (5) large round cellule; (2) (3) elongated, spindle shaped type cellule;

(7) small round cellule cluster form, (3) still clinging together; from area just above free margin of chorion; and from contiguous area just below this free margin in the area vasculosa in chorionic cavity: other cell divisions not numbered; see cluster in Fig. 9 (1). These types are all common in cancer. See Figs. 14 to 25.

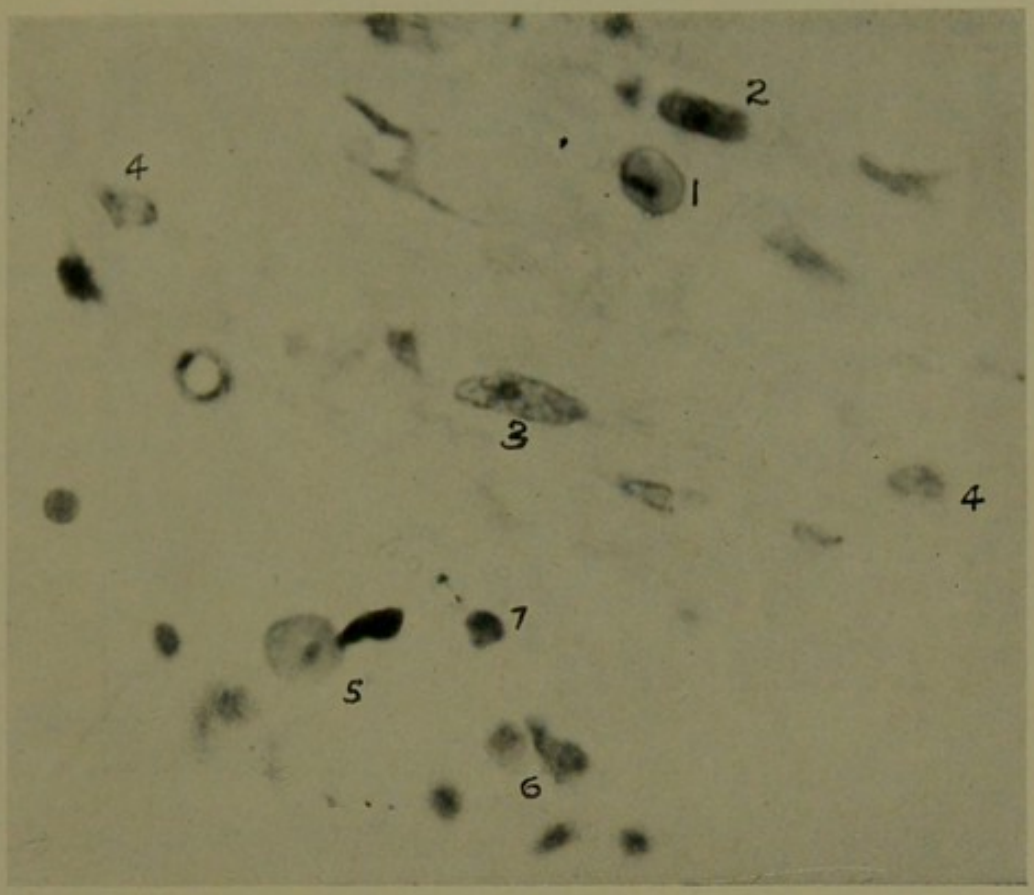

FIGURE 6

FIG. 6. To show division in normal cellules in the contiguous margins of the stroma of the chorion and area vasculosa in chorionic cavity at 3-4th week, human ovum. Types seen in cancer (and s).

Fig. 7. Normal divisions, 3rd-4th week continued; the normal cellulette, the forerunner of the infant small round cellule, neocellule of the normal division; the prototype of the small round cell of carcinoma and sarcoma.

Other types of differentiation, division and multiplication, as seen in a blood island just below the free margin of the chorion in the chorionic cavity.

1. Large round, multinucleolated form, shadow and light suggesting cleavage or separation of the several infant nucleoli.

2. Other smaller, multinucleolated nuclei suggesting plural infant nuclei; medium, round cellule.

3. Kidney-shaped cluster; adherent nucleoli suggesting multiple division and escape of a neonat infant nucleus, two remaining behind closely approximated.

4. Infant nuclei round cellules.

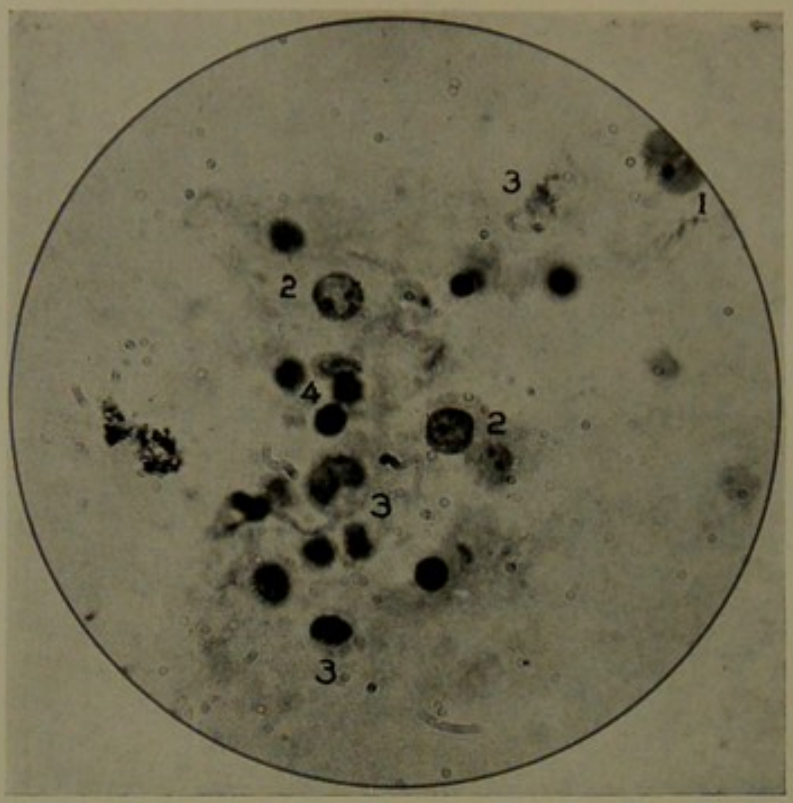

FIGURE 7

FIG. 7. Normal division, 3-4th week continued; human ovum. Observe particularly the cellulettes, the pre-small round cell form. All these types seen in cancer (and s).

5. The cellulette, not numbered but plainly seen; others more faintly seen here; the sub or pre-small round cellule type of division of the large round spinal cellule type of multinucleolated nucleus-cellules; vacuolated condition of cellules, pre-division, seen very plainly in cellules in Fig. 10 and in others.

All types of above cellules are common in cancer and sarcoma.

INTERESTING VERY EARLY PRIMAL DIVISION IN CYTOLOGY OF NORMAL AND CANCER EXPRESSION. THE MATRIX AND BLOOD CELLULETTES. ANALOGUES IN CANCER.

Throughout this field, Fig. 7, blood island of 3-4th week human ovum growth, there is seen many diminutive very small circular cellulettes, in most cases outlined as only a ringlet with clear transparent contents; this in the very small cellulettes. In some cellulettes there is a pin point darker spot within the ring suggesting a nucleolus already at this diminutive period. These must be the sub-smaller form of the small cellule of a normal small cellule division. Use good hand glass again to obtain good detail.

In a few cases, though quite faintly seen, these cellulettes are arranged in link-chain cluster form, the earlier form of the later

well seen link-chain cluster as seen in Fig. 8 (3 above). These clusters are faintly seen here, about (3) in the center of Fig. 7 and S. E. of (3) below; also in other areas of the field; for clusters in cancer see Figs. 14-19-20-21-25.

In fresh specimen these cellulettes are easily to be seen; in older specimen not so easily, their substance seems frail and to disintegrate and disappear with time. Many other normal cellulettes may be found in the cellular divisions seen in the stroma of villi and chorion, 2-8th week, some easily seen, others less readily seen. Figs 3-8-11.

The importance of these findings lie, among others, in the fact that in cancer and sarcoma these cellulette expressions are commonly and more easily to be seen. See Figs. 14-19-20-21-25. In many illustrations, both normal and cancer, these cellulettes are

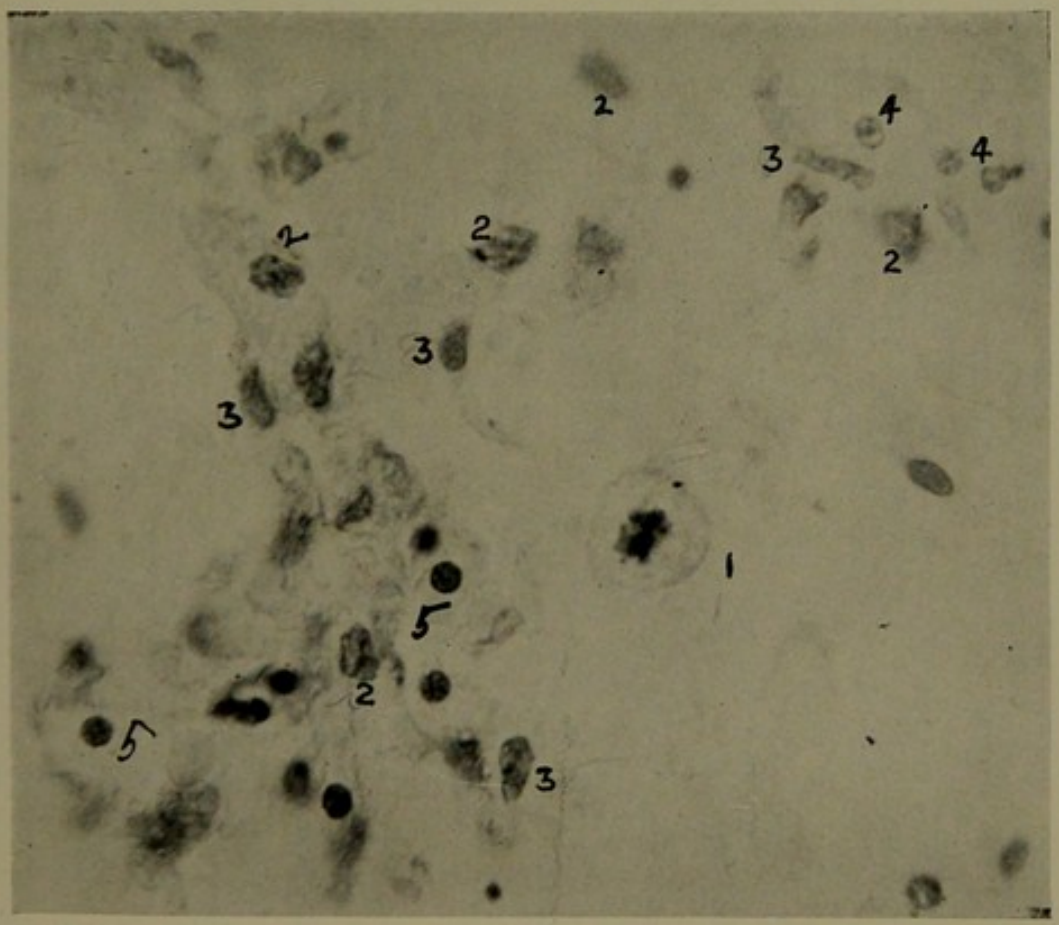

FIGURE 8

FIG. 8. Shows division of normal embryonal cellules, at 5-6th week, extra uterine human ovum. Mitosis and amitosis division. Outstanding is (3) the elongated narrow link-chain cluster. All these types of cellules seen in cancer (and s). (1) Mitosis, Karyokinesis or Indirect Cell Division. (3) below; Amitosis or Direct Cell Division.

CHART 1

A CHART SHOWING PARALLEL DIVISIONS IN THE NORMAL MATRIX EMBRYONAL CELLULE; AND DIVISIONS IN ITS ABNORMAL OFFSPRING, THE CANCER CELL.

The normal primal multinucleolated cellule The normal embryonal cellule of the syncytium 2-8th week 1st stage of evolution	Proliferation Division Multiplication Into	1, small round cellulettes — Fig. 7-8-10-11 2, small round cellule — Fig. 3-7-8-10-11-12 3, medium round cellule — Fig. 1-3-7-8-10-11-12 4, large round cellule — Fig. 1-3-7-10-11-12-13 5, spindle or elongated cellule — Fig. 5-6-8-10-12 6, cluster 2-3 small round cellules cling together, kidney shaped — Fig. 6-7-8-9 7. cluster 4-5 small round cellules cling together in long narrow band link chain form — Fig. 7-8-11
Embryonal nucleus-cellule of syncytium 2nd stage of evolution	Proliferation then Differentiation Proliferation but in many cells, further differentiation ceases and there is only proliferation; the cancer cells	Where 100% growth control hormone conducts; the normal. Majority of the six divisions of the normal embryonal cellule. They continue on to differentiate into many forms of further metamorphotic ultimates. One example, primal uncolored blood corpuscles differentiation through the erythroblast to the erythrocyte. Where growth control hormone conduct is aberrant or absent; the abnormal cellule and tissue. Great proliferation of these cancer cellules but no further differentiation; now forming the blighted cancer cells and tissue.

CHART 2

THE CANCER CELLULE.

Third stage of Evolution of Cancer Cellule

The Cancer Cellule
Great Virulent
Proliferation
Division

Multiplication

Division same as in normal first stage of primal embryonal cellule into

1, cancer cellulettes Fig. 14-15-16-19-20-21
2, small round cellule Fig. 14-15-16-19-20-21
3, medium or transitional round cellule Fig. 14-15-16-19-20-21
4, large round cellule spinal Fig. 14-15-16-19-20-21
5, the spindle or elongated cellule Fig. 17-18-19-20
6, cluster 2-3 small round cellules cling together, kidney shaped Fig. 19
7, cluster 4-5 small round cellules cling together in long narrow band link-chain form Fig. 19

All these cancer forms same as those in the normal divisions.

The complete Cancer cell types seen in the various forms of carcinoma (and sarcoma)

1—The small round cellule cancer type
2—Medium or transitional cancer cellule type
3—The large or spinal cancer cell type
4—The spindle shaped cancer cell type
5—The cluster infant neo-cellule cancer cell type, 2-3 cling together, kidney shaped
6—Cluster, 4-5, neo-cellules cling together link-chain like, cancer cell type
7—The cancer cellulettes.

Another most interesting feature here, and one not a little baffling, is the marked relationship, as suggested above, between the various forms of cancer cell and those of the sarcoma. They are like twins, the same in form and type; both descent types from the normal embryonal cellule. The sarcoma though seems to grow more rapidly and is far more virulent in character; destroys in less time.

The primal matrix ancestral normal embryonal cellule and the abnormal cancer cellule are identical primarily, and have the same origin in the syncytium. Their first stage of evolution.

Both cellules pass through their second stage of evolution, proliferation, as one in normal histologic manifestations. Their second stage of evolution.

The one cell, the normal, continues on through the second stage of evolution, proliferation, to and through its third stage of evolution, differentiation, to normal tissual ultimates. Its third stage of evolution.

The second cell, the now abnormal cancer cell, ceases normal evolution in proliferation; thereafter it exercises its intense energies in great viscious proliferation only; amoeboiding its environment; replacing it with cancer tissue; through exhaustion destroying both host and itself. Its third stage of evolution.

not very clear; they are too primal and faint in color always to appear in the field of microscopical vision, their substance seemingly so primal they do not take the stain so readily and retain it.

Fig. 8. Normal cell division, in the chorion, extrauterine at 5-6th week; (1) Karyokinetic figure, mitosis, indirect division; (2) large round cell; (3) below elongated spindle cell; apparently here amitosis or direct division; both forms seen repeatedly in the normal. Both forms of division common in cancer and sarcoma expression; (3 above) the long narrow band link-chain cluster of clinging infant cellules; see also Fig. 7 for the normal. In the cancers see Fig. 14 in a syncytioma, cluster in a cellule; Fig. 19 in a glioma; Fig. 20 in a neuroblastoma; see also Figs. 21 and 25.

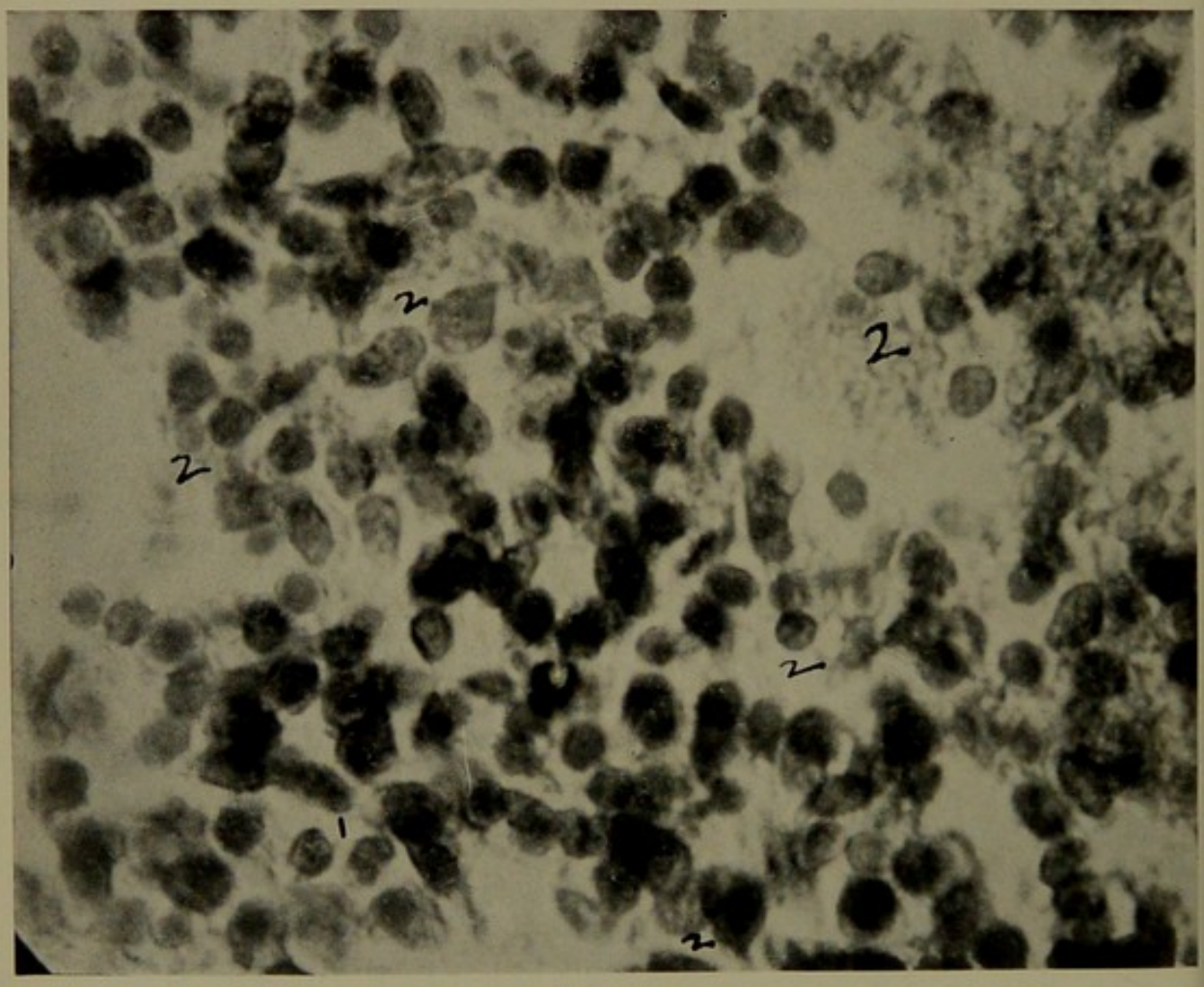

FIGURE 9

FIG. 9. To show large round cellule multinucleolated uncolored blood corpuscle, the syncytial type of cellule still persistant in the embryonal white blood circulation at 5-6th week human ovum. At (1) small round cellule cluster of neo-cellules, as seen in normal division before. (2) syncytial form and type of blood corpuscles same in type as seen at 2-3rd week. These types found in cancer (and s).

Fig. 9. The multinucleolated nucleus-cellule blood corpuscle, (2) the syncytial differentiating blood corpuscle type, at 5-6th week; Primal cellule type of primal uncolored blood corpuscles in blood sinus at head of cord, 5-6th week, human ovum; same at 2-3rd week, see Fig. 5. These types are common in cancer.

(1) the small round cell cluster, 3 small round cellules still cling together, see Fig. 7 (7), also Fig. 11. Both forms are seen in cancer and sarcoma.

Fig. 10. All types of normal cell division seen in small area of intervillus bridging tissue at 8th week. All types of cellules seen here; the large (1), the medium, and the small round cellule type; also the spindle (2) cellule. Note the vacuoles in cellules, suggestion of extruded nucleoli; cellulettes. All these types seen in cancer and sarcoma.

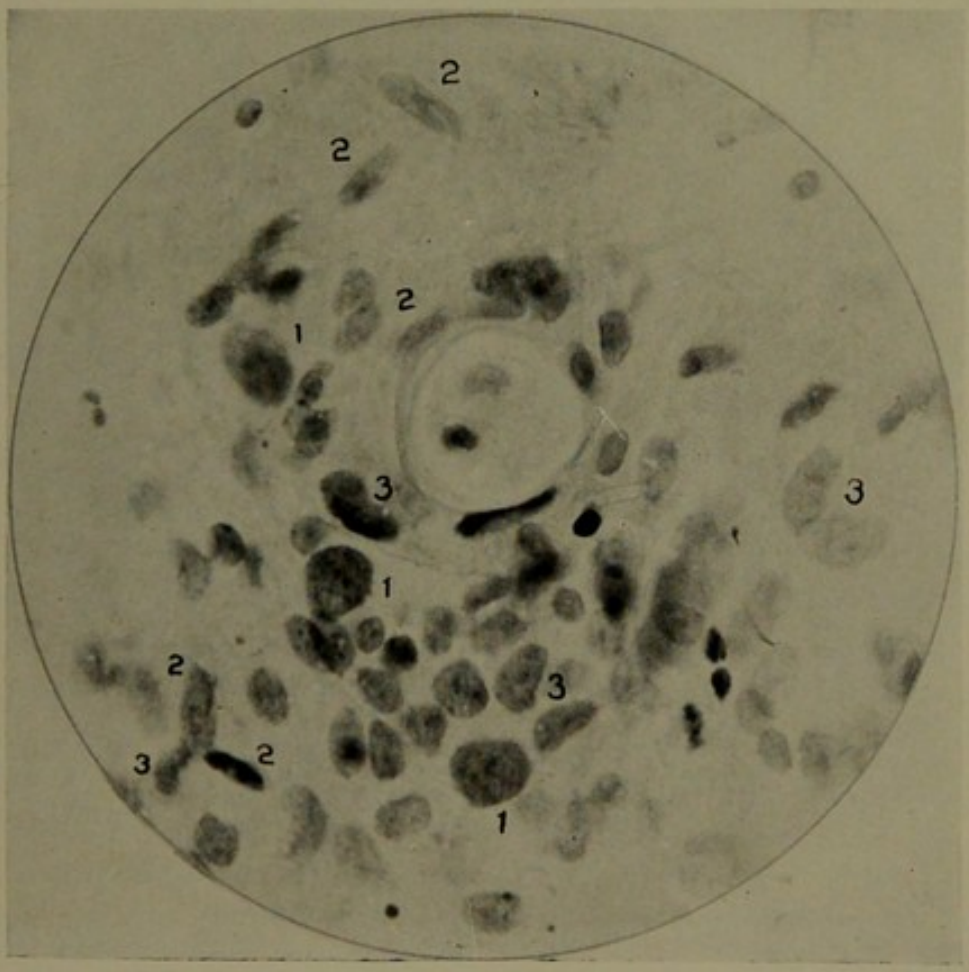

FIGURE 10

FIG. 10. Normal division in a primal inter-villus tissue growth; 8th week; all types of primal normal cellular growth cellules, from 2-8th week are to be seen here; similarity to the large spinal, transitional, small and spindle shaped cancer types easily recognized.

Fig. 11. Cross section of umbilical vein, 5-6th week; showing further division and differentiation in the normal blood cellules at 5-6th week; where it ceases in the abnormal cancer cellule. Introduced here to accentuate the differences between the immature multinucleolated blood corpuscle nucleus-cellule; cellules without characteristic cytoplasm and a true cell like the erythroblast; the latter a single nucleus surrounded by a characteristic cytoplasm. These are the cellule pictures that must be kept in mind when one considers the theory of transmutation of cells; from the mature cell form back to the immature embryonal cellule form. Whereas cancer is progressive cellular evolution not regressive.

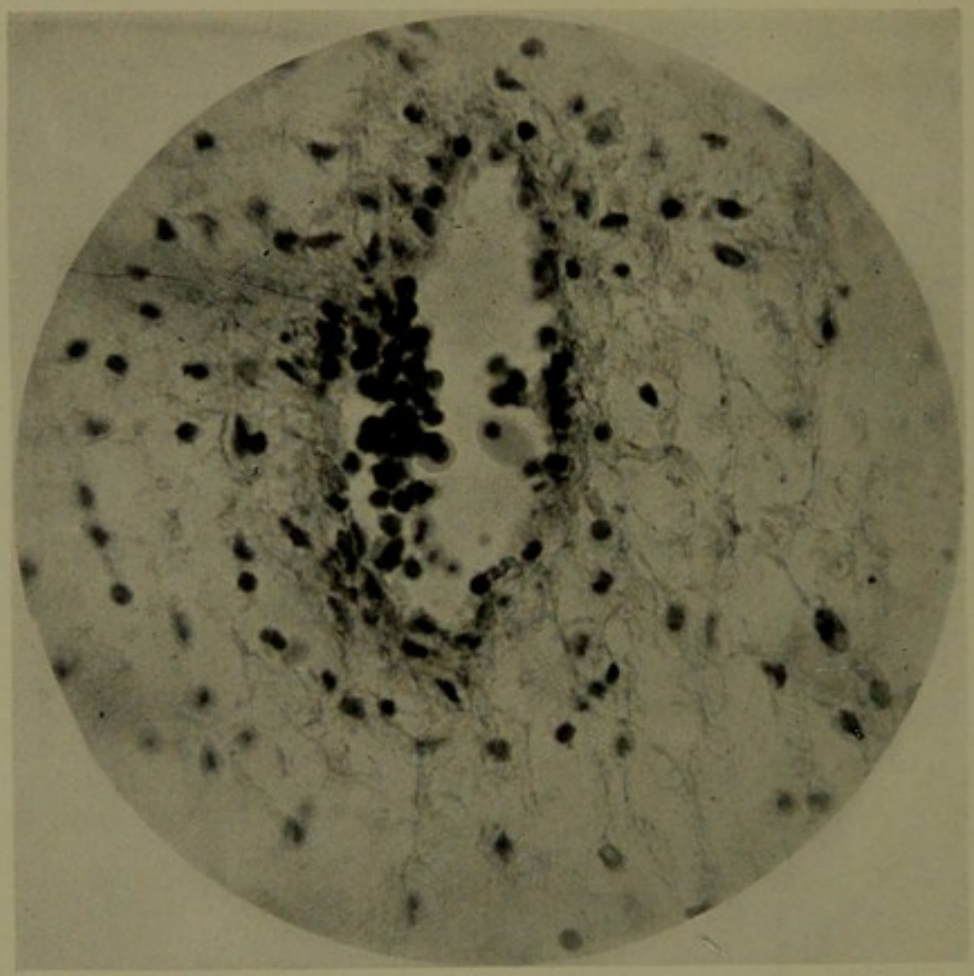

FIGURE 11

FIG. 11. Cross section of the umbilical vein in umbilical cord at 5-6th week, human ovum. To stress the great differences which exist between the mono-nucleated red erythroblast cell of 7.5 to 17.5m in diameter and the much smaller type 4m, of multinucleolated nucleus-cellule type of blood corpuscle at this period; same in type as the primal matrix embryonal cellule of the syncytium. Many syncytial cells in environment of stroma of cord. Note the erythroblast has a characteristic surrounding cytoplasm; the smaller nucleus-cellules have none. Cancer cellules likewise are without surrounding cytoplasm.

The cellules here in the stroma of the cord are all primal wandering cellules, intent on entering the blood stream. In the S. E. border of the rim of the vessel, two neo-cellules, small round cellules are to be seen, the small round cellule cluster, again showing the normal division at this period. And here again the prototype of the small round cells of carcinoma and sarcoma, but with a different future differentiation. With hand glass note the form of the various cellules in the stroma of the cord; many large round cells and many elongated or spindle shaped forms are to be seen. All of these forms seen in cancer, except the erythroblast.

Again note the clearly defined blood cell picture of the red mononucleated erythroblasts; a nucleus surrounded by a cell plasm with limited outline, 7.5 to 17.5m. in diameter. On the contrary, observe as contrast the uncolored blood corpuscle, 3.5 to 4m. in diameter; it is still an uncolored multinucleolated vesicular nucleus without cell surrounding plasm. In type still retaining its primal syncytial character. But from now on it disappears to make room for its offspring and successor, the red erythroblast.

Note: Laboratory treatment and staining of the tissues of this specimen were naturally the same, one. The cytoplasm of the erythroblasts is very plain. Were there cytoplasms, around the syncytial forms of cellules, those, either within or without the vein, such cytoplasm would have come out equally as plainly as in the erythroblasts.

Fig. 12. From a 3-4th week villus from an intrauterine human ovum, to show syncytial detail especially.

Here is shown (3) the origin of the wandering features of the primal embryological free nucleus-cellule, the syncytial cellule, the ancestor of, and which gives origin to the cancer cell. Naturally therefore, the well marked wandering, actively invading characteristics of the cancer cellule comes to it through inheritancy; and naturally, it bestows such wandering characteristics on its cancer offspring.

The epithelial cell is always a fixed stationary cell, never roaming or wandering from its fixed locus; again, handing down its fixed characteristic to its epithelial offspring.

Here is seen another, but very important differential, between the wandering invading cancer cellule and the fixed stationary epithelial cell.

Fig. 12 inserted here to stress larger amplification of empty nucleus-space; nucleus wandering into blood-space vessel; from 2nd or inner row of the syncytium of an elongated villus at 3-4th week. Note the consolidated plasm of outer row. The circumferential villus plasm plus the absorbed nutrient material amoeboided from the villus environment.

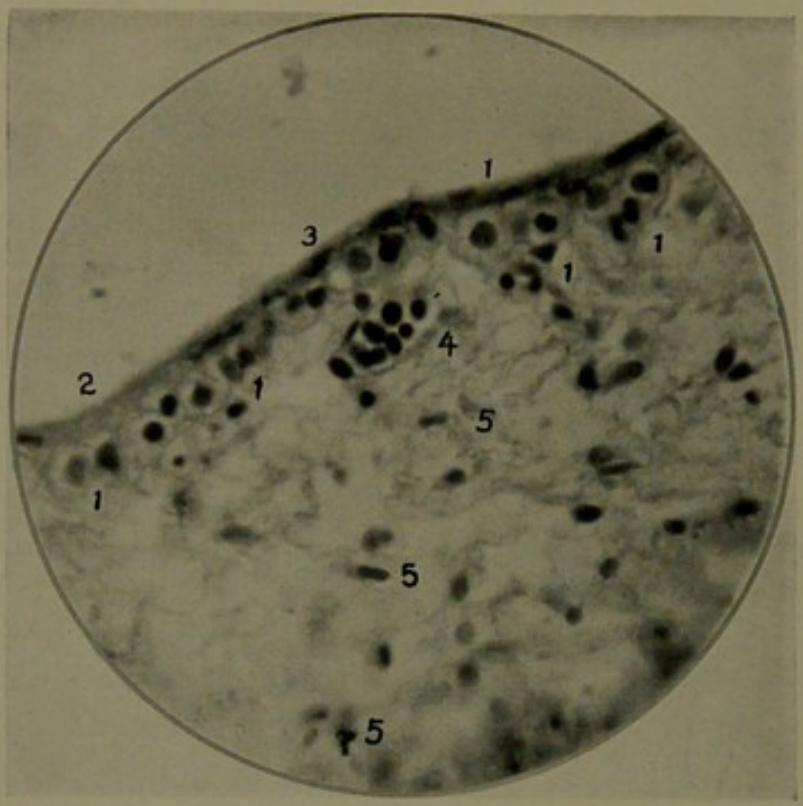

FIGURE 12

FIG. 12. To show detail of syncytium; syncytial cellule (3) wandering from second row into blood space vessel; all divisions of primal cellules seen in stroma of villus, at 3-4th week, human ovum. Prototype of inherited cancer cellule wandering.

1. Attitude of nuclei as though in the act of inwandering from circular seat.

2. Above in outer row very faint empty effects, as though seat of departed nuclei.

"Each nucleus, or primal cellule, having a circular digestive influence, clears a circular space around itself, leaving and bounded thereby a slight intermediate margin of plasm persisting for future assimilation; thus there is given to the inner row its separate cell appearance." (Syncytium 1902).

In the course of the wandering cellules in the stroma of the cord, to enter the vein, crowding its rim is seen another explanation for

the theory formerly held, that proliferation of the endothelium of the vessels gives origin to the blood corpuscles. It will be noted that here, there is no endothelium.

3. Empty space from which nucleus cell is wandering into blood space-vessel. Proof positive that the empty space lodged a smaller nucleus-cellule only, not a larger regular technical cell as of an epithelial nature; viz.: a nucleus surrounded by a self limiting cytoplasm. The outline would have been entirely different.

4. Note no endothelial lining, margin in blood-space and vessel darkened as though the effect from hardening of laboratory media; such marginal effects seen frequently even in the large vessels of the early cord.

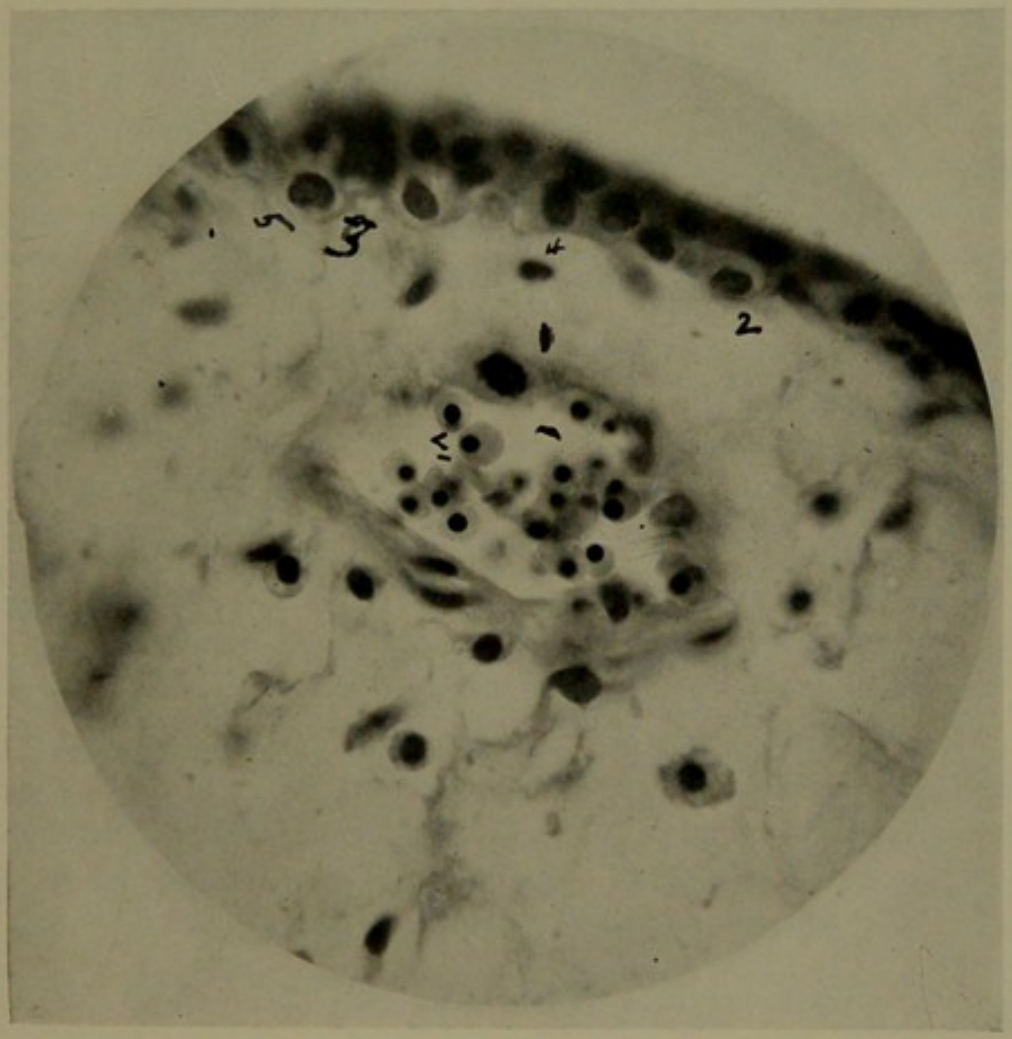

FIGURE 13

FIG. 13. Again to show detail of syncytium in cross section of villus at 7th week, human ovum. Consolidated plasm of external margin of villus, made up of peripheral margin plasm of the villus, plus the absorbed nutrient material from syncytial amoebic activity.

Many nuclei wandering in stroma of villus; in stroma here like in blood spaces, nuclei undergo division (5) and multiplication, well shown as smaller nuclei (5), adolescing.

Fig. 13. To show again high type blood corpuscles differentiation from syncytial form to erythroblast. Conduct of multinucleolated-cellules in the syncytium is to leave the syncytium and to wander and enter blood vessels. Stain too rich here but allowance can be made.

Cross section of blood vessel from villus, 7-8th week; to show, especially, syncytial detail. Vessel margin, no endothelium, content, mono-nucleated erythroblasts; erythroblasts in stroma of villus; observe nuclei wandering from second row, several empty space effects there; several coarse multinucleolated nuclei in rim of blood vessel; unusually clear, distinct minutiae of syncytial border with double row cellules; likewise of stroma of villus, blood-space, etc.

1. Large second row nucleus wandering into blood-space vessel; with large hand-glass, this nucleus appears to have several nucleoli as though ready to break up into several offspring, future erythroblasts; others around edge; no endothelium. This picture probably one explanation of former theory, that proliferation of endothelium gave origin to red blood corpuscles.

2. Note second row nuclei seem directed inward toward stroma of villus, especially at (2); showing principle of direction and activity.

3. Empty spaces suggesting departed nuclei.

4. Empty space with nucleus just below; nucleus appears dividing into 2 offspring.

5. Large nucleus-cellule about to divide.

Above, in previous pages, the assertion is made that cancer is extra-embryonal in origin, not embryonal per se; that cancer is descended from primal extra-embryonal syncytial cellules shown here; and that the cellules are not epithelial; therefore that cancer is an atypical proliferation of epithelial cells, is error.

RÉSUMÉ ILLUSTRATIONS OF TYPES OF ABNORMAL CANCER CELL DIVISION.

Fig. 14. From a pulmonary metastasis of a syncytioma. Original presentation and lantern slide illustration of the specific cancer

cell read before the Chicago Gynecological Society and the Chicago Medical Society, June 1902; published, Amer. Jour. of Obstetrics, N. Y., December 1902.

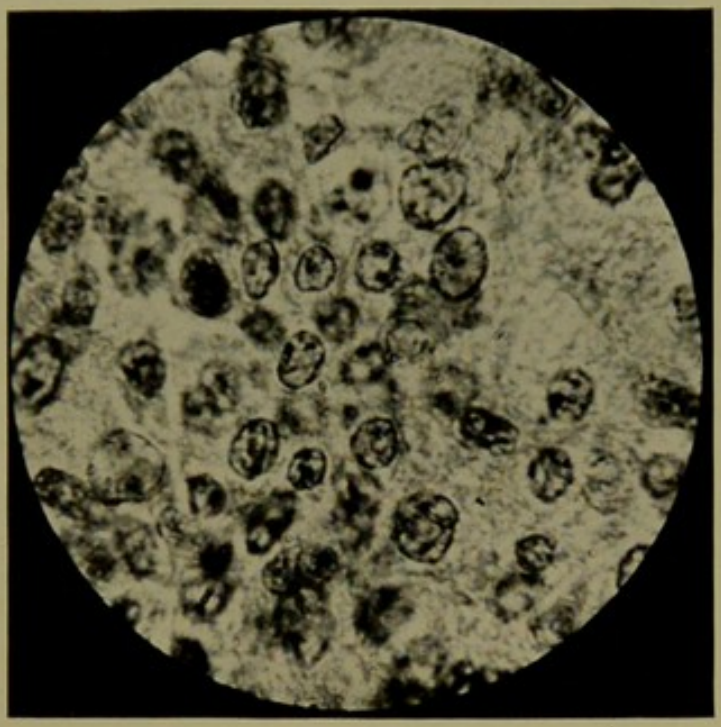

FIGURE 14

FIG. 14. Pulmonary metastasis of uterine syncytioma. Note all forms of the cancer cellules. The spindle type missing. Note in cellule to the left below, beaded cluster of nucleoli soon to be cancer cellulettes.

All forms of cancer cells are seen here; the large, medium and small round cell types. Compare with figure 21, Annals of Surgery, January, 1931; also Fig. 25, metastasis lymph nodule.

THE CANCER CELLULETTES; FIG. 14.

Note the diminutive cancer cellulettes, the successor forms of the nucleoli of the cellules; both in the cellules proper and extruding from their circumferences. Not many but a few distinctly, though faintly to be seen in the center of the cancer field; some also in other fields. Small vacuoles in large cancer cellules seem origin; follow the circumferences for suggestion of other cellulettes. One medium sized cell, lower left margin of field shows two cellulettes through its diameter, like a small chain cluster. For normal blood cellulettes cluster see Fig. 9 (1). Cellulettes here in Fig. 14 seem extruding from circumferences of cellules; note in Figs. 14, 15, 21, 25 like features.

Fig. 15. Large spinal type round cell cancer.

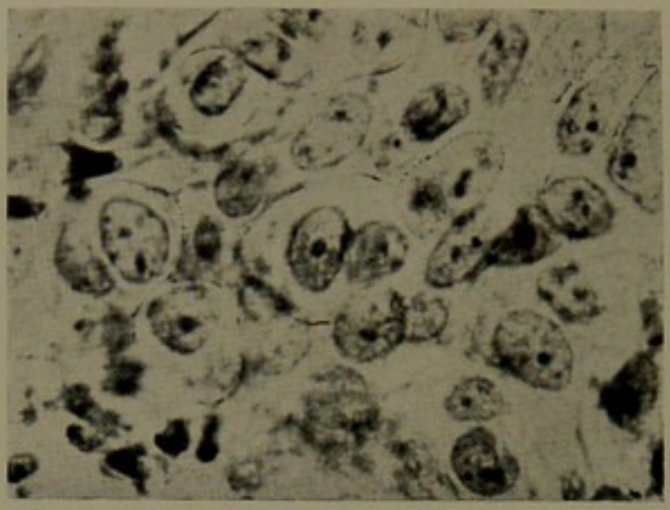

FIGURE 15

FIG. 15. Spinal types of cancer from epidermoid forms of uterine cancer. Multinucleolated nucleus-cellules features very marked; no cytoplasm. Types same as normal.

Fig. 16. All types of cancer cellules; the large, the spinal;

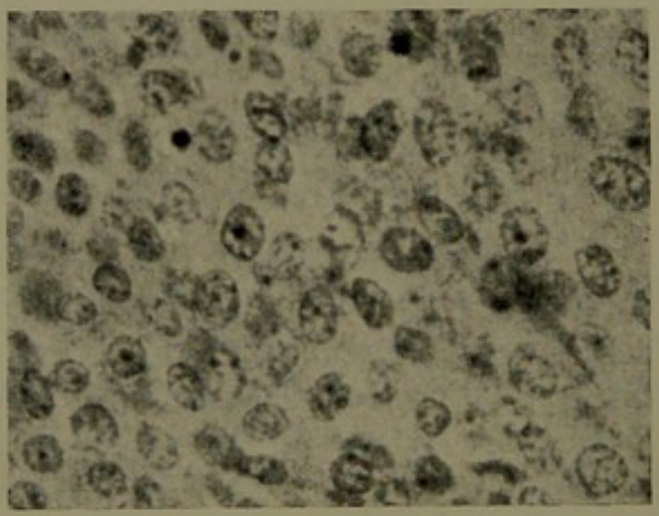

FIGURE 16

FIG. 16. Epidermoid uterine cancer. All types of cancer cellules to be seen; excepting the spindle type; no clusters. Types same as normal.

the medium, the transitional; the small round cell type of cancer cell.

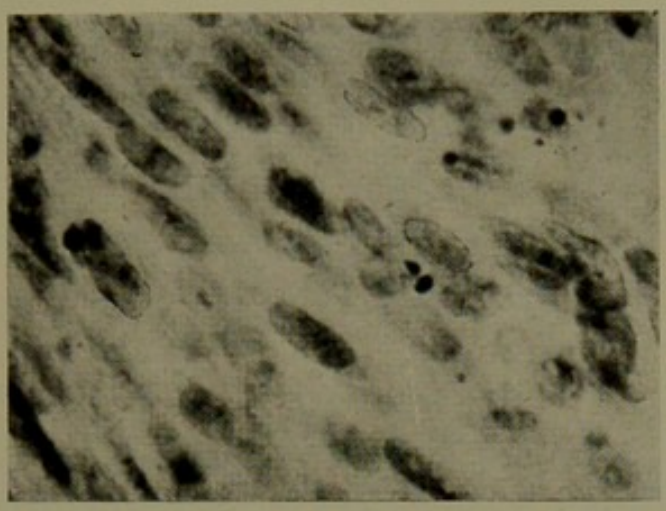

FIGURE 17

FIG. 17. Epidermoid uterine cancer. Spindle type of cancer cellules very prominent. Types same as normal.

Fig. 17. The spindle cell type of cancer cellule. For normal prototype see Fig. 5 (1), 2-3rd week; Fig. 6 (2) (3), 3-4th week; Fig. 10 (2); Fig. 12 (5). Human ovum.

Figs. 15-16-17 from Dr. Karl H. Martzloff, Portland, Ore., Epidermoid uterine cancer, Amer. Jour. Obst., and Gyn., Oct., 1928.

Fig. 18. Elongated spindle cellule, enlarged 2500 times; infant cellule below. From Dr. Michael Levine, New York, Cytology of Cancer, Am. Jour. of Cancer, pages 144-145, Jan., 1931. For normal prototype see Fig. 5 (1): see Fig. 6 (2) (3); Fig. 10 (2); Fig. 12 (5).

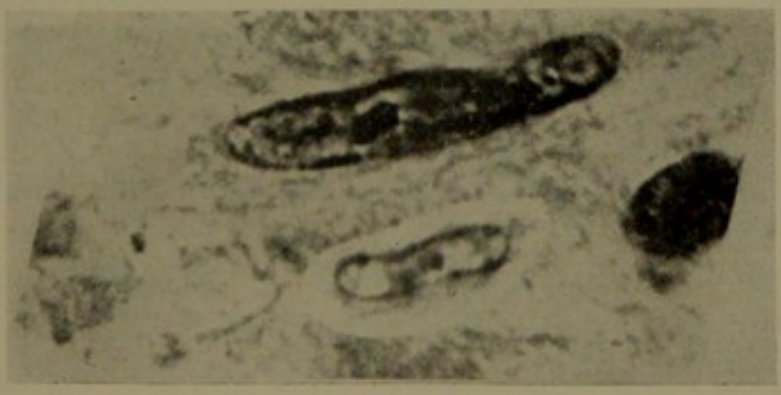

FIGURE 18

FIG. 18. Elongated spindle cellule, enlarged 2500 times; infant cellule below; from lip cancer. Type same as normal.

Fig. 19. The long narrow band link-chain cluster of small infant round cellules. From a specimen of glioma. As will be easily recognized, all these types of cancer cellules are seen in the normal prototype divisions shown above, from which the cancer cellules are descended.

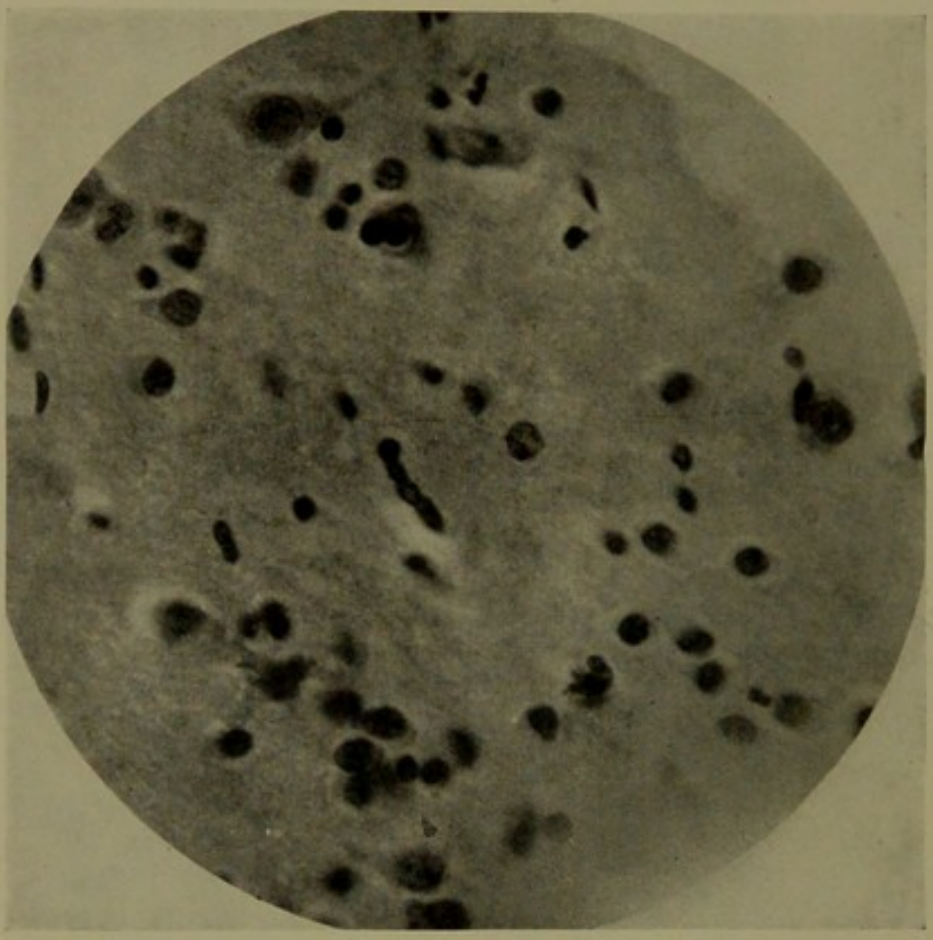

FIGURE 19

FIG. 19. The long link-chain cluster and other forms of cancer cellules, from a glioma; all these types of cancer cellules are to be seen in the normal divisions.

Fig. 20. Shows again this rarer form of the long narrow band link-chain infant cancer cellules cluster, here running through the center of this neuroblastoma. All types of cancer cellules are to be seen in surrounding field; no characteristic surrounding cytoplasm.

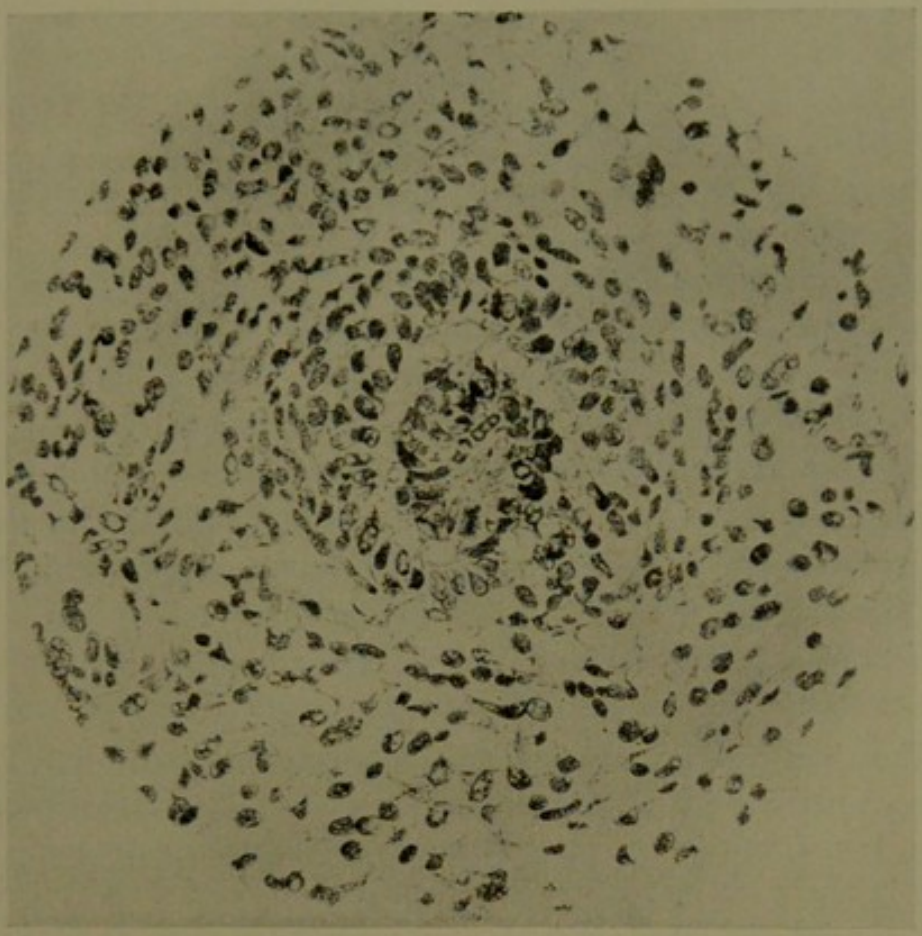

FIGURE 20

FIG. 20. All types of cancer cellules to be seen in this field from a neuro-blastoma; the long link-chain cluster to be seen extending through the center of the growth. Similar types in normal divisions.

This plate was made from illustration 512, page 965, Dr. W. G. MacCallum, Pathology, John Hopkins, 1928.

AGAIN THE NORMAL PRIMAL EMBRYONAL MATRIX CELLULES. FIG. 1.

The normal primal matrix embryonal cellules descend and exist throughout life, pre-natal and post-natal; the normal matrix cellule is the Original Wandering Cellule of the body; wandering throughout the tissues of the body general, same in type, form and characteristics as in primal syncytial days.

Injure these primal matrix embryonal cellules in pre-natal or post-natal life, through irritation, burns or other traumatic causes or through disease; beyond the limits of their normal physiological resistance, then change in their intrinsics and chemics follows; abberration in normal growth control occurs; hence the cancer cellule.

Therefore the now, bruised, just formerly normal embryonic cellule, becomes aberrant in further division and changed in function, now taking on the ens malignitatis of the cancer cellule, and transmitting the same to their cancer offspring cellules.

Again here the great question in cancer. What is the inherent cause of this aberration, this change in the intrinsics and chemics in the cancer cellule, this ens malignitatis function? Chemico-physiologic would be the answer here. Chemical first, physiologic following; a common result in all nature where primal original chemical arrangement of vital complex units is disturbed.

And too, another thought here.

Oft times expression reads as though, effort has missed or lagged in its endeavor to fix exact changes and causes in the intrinsics of the aberrant cancer cellule.

Yet, who has ever discovered the intimate intrinsics that cause life to start in the primal cell!

The ripe ovule awaits the eager spermatozoa; each electrified to a new fertilization. Contact alone ensparkles that double decomposition, transformation of the old two into the new single one! What was in the spark that initiated, activated the new life? Was it chemical, physiologic, spirit, each alone or all three in one?

That question has never been and probably never will be answered unless our means of appreciation will change quite materially from those of today.

Prenatal and postnatal cancer (and s) cytology are one, identical in type, appearance, form and malignant characteristics.

FOR CORROBORATION.

This cancer cytology may be and must be found in all cancer (and s) histology, in prenatal or postnatal life; also that it can be found in all such illustrations in appropriate technical journals and books, of today as well as of yesterday. Of course there where clear distinct illustration rules, barring only the inartistic work and illustrations. And these conditions must and will apply to International as well as to National effort.

SEARCH.

With this crucial test in mind, one of the first technical books taken in hand was The Annals of Surgery, January 1931, containing

the "International Contributions to the Study of Cancer in Honor of the James Ewing Cancer Symposium, 1930."

Casually scanning the pages, sought to apply to such appropriate illustrations that test. It worked; results very good. But soon came upon one illustration, described here under Fig. 21 below; the test ruled on the spur of the moment, pure cancer. The illustration presented a splendid picture of pure cancer cytology only; six of the seven forms of cancer cellules being present, all clearly and distinctly outlined; only the spindle shaped cancer cellule, so common in sarcoma, is missing. Not having looked at the caption given in the article, was surprised when looking up, to read: Undifferentiated Round Cell Sarcoma.

Why was this tumor termed sarcoma; only pure cancer cytology appearing in its histology. In the picture, connective tissue remnants are absent or negligible. Is the connective tissue remnant determinative for diagnosis or is it the process?

The amount of intercellular connective tissue remnant in such a malignant tumor growth will depend up two factors.

First, the invasional activity, amoeboid digestion, and irritation caused by the invading cancer (or s) cellule, which at all times causes more or less defensive inflammation, congestion, hypertrophy and swelling in the local host tissues, even in cartilage and bone. This is seen in the irritation inflammation hypertrophy of the papillae in the skin cancer and also in the inflammation hypertrophy caused and seen in the primary stages of the experimental skin cancers in mice. But soon this hypertrophy disappears in the maw of cancer (and s) amoeboid digestion of local tissues unless hindered by death of the process.

Second, the other factor permitting remnants of local tissues to appear in malignant tumors, is the resistance offered by the physiological resistance of the local host cellules and tissues to the vicious efforts of the invading malignant cancer cellules.

Finally in general the pictures of destruction and clinical morbidity in the cancer and sarcoma are much the same, the detail of destruction merely differing. Like in the dynamite, the destruction of the explosion is general in principle; the detail only differing as the physiologic resistance of the construction material determines. Cancer and Sarcoma are one.

A SIMILAR THOUGHT.

Dr. Ferdinand Blumenthal, Berlin, in his contribution to the London Cancer Conference, Page 18, 1928, writes: "Under the term Cancer we should understand all malignant blastomas whether of epithelial, endothelial or of connective tissue origin."

Some cases in point, as types, where revision, restudy of reported Fibrous-lympho-sarcoma, Lympho-sarcoma and Sarcoma yielded cancer. Such cases (27) were the Schneeberg Miners' Disease, originally thought sarcoma.

These Schneeberg cases are of much interest. Aside from their purely cancer features, they show the irritation effect of the "dust with sharp-angled particles with an arsenic content reaching 0.45 per cent;" and "air in the mines, radio-active up to 50 Mache units."

Reflection asks here.

Contrary to the greater amount absorbed in the match and watch-dial industries, does the small arsenic and radio-content of the air and dust in the mines, assist in building up a resistance to the active processes of cancer, for in four of the cases, even after changing occupation, their cancer continence extended over a period of 10-18 years; then first, recurrence and cancer death.

A short excerpt of these reports is as follows:

"The mining industry in Schneeberg at the present time comprises bismuth,' cobalt and arsenic. The air in the mines is radio-active. According to the calculations of Ludewig and Lorenser, it possesses an emanation content up to 50 Mache units. In drilling the hard rock, great quantities of dust are produced, and this dust, which is somewhat radio-active, is composed of minute sharp-angled particles with an arsenic content reaching 0.45 per cent. In certain places the mines are moist and show abundant growths of fungi — Polyporus, Paxillus, Lentinus, Penicillium, and Aspergillus.—Clinical and Radiological Study of Schneeberg Lung Cancer by O. Rostoski (Dresden)."

"The first pathological observations on the lung disease so commonly found in the miners of Schneeberg were made by Hesse and Harting in the seventies of last century. By the co-operation of Weigert they established from autopsies the fact that the pulmonary affections were due to tumor formations. As a result of histological studies by Wagner and Weigert in Leipzig these tumors were regarded as lymphosarcomata. In Munich, Anke, after examining

microscopically the lung of a Schneeberg miner, expressed the opinion that it was fibromatous lymphosarcoma. For this reason the tumor which is endemic amongst the Schneeberg miners is referred to in the literature as lymphosarcoma, but doubt arose regarding the real nature when Arnstein in 1911 re-directed attention to the disease. He and Margate Uhlig, who had each the opportunity of examining two cases histologically, found that they were pure epithelial tumors, and the doubt increased, when Risel of Zwickau, on examination of an old specimen in the Leipzig Pathological Institute dating from Weigert's time, showed conclusively that it was a small-cell carcinoma; and, in addition, he was able to demonstrate pure carcinomata in two other Schneeberg miners on whom he had performed autopsies. It first became possible to remove the last doubts about the nature of the tumors under consideration through the investigations on the Schneeberg lung disease instigated by the Saxon Cancer Campaign Committee. The Committee made arrangement for autopsies on a large number of miners who had died of the disease, and the study of the material was entrusted to the author, who was able to examine 22 cases in all. It was found that in 21 cases the lungs were the seat of pure epithelial tumors, that is, carcinomata, and in one case the lesion was a chronic pulmonary tuberculosis.

The above considerations provide the incontestible proof that the tumors developing in the lungs of the Schneeberg miners are pure epithelial neoplasms, that is, carcinomata. It seems not improbable that the tumors found last century and identified as lymphosarcomata were likewise carcinomata. The earlier investigators had not at their disposal the technical aids which nowadays are employed for establishing the diagnosis. The case of Schneeberg lung cancer preserved in the Leipzig Pathological Institute, which Risel examined, provides evidence in favor of such a conclusion. It may be mentioned that vegetable or animal parasites which some have endeavored to associate with the development of cancer were never found in the cases that I investigated.

In conclusion, I would refer to a remarkable fact. Among the cases investigated by me there were four individuals who had been employed for a long time as workmen in the Schneeberg mines, but

had given up this work on account of lung trouble, and it was not until ten to eighteen years later that they developed lung cancer, from which they died. As we can scarcely assume that lung cancer had developed during the time of their employment in the mines, and since an accidental incidence of lung cancer such as occurs in other people is not specially likely, we must conclude that during their work as miners they not only developed anthracochalicosis, as the examination showed, but they also acquired the cancer-exciting factor which, however, remained inoperative in them for a very long time. Experimental attempts to produce cancer by inhalation in white mice with the fine dust set up by drilling operation, or by rubbing this dust into the ears of rabbits, have, up 'till now, proved negative.—Pathological Study of Schneeberg Lung Cancer by G. Schmorl (Dresden), Cancer Conference, London, 1928."

Note: The cancer exciting factor. Were these the cancer cellulettes, whose ens malignitatis was held in abeyance by a strong physiological resistance?

Many other types of reported sarcoma are open to a like question in diagnosis, as the many forms of carcino-sarcoma, sarco-carcinoma; osteo-condro-sarcoma following prostate carcinoma, etc.; their names alone suggestive of doubt as to the purity of their histologic and cellular origin.

Fig. 21 illustrates another such case, as type, where pure cancer cytology only is found in a reported case of Undifferentiated Round Cell Sarcoma. This case shows in its cytology six of the seven cancer cellule forms, the missing form being the spindle cellule so common in sarcoma.

HISTORY OF CASE IN FIG. 21.

"Male, aged twenty-nine, was seen because of a large swelling of the dorsum of the right scapula which had developed during the previous seven months. At first the swelling was symptomless, but recently it had produced some pain and considerable limitation of motion in the shoulder. No loss in weight or strength. Examination showed a large, firm, soft, oval swelling involving the entire posterior scapular region and limiting elevation of the arm. General condition of the patient excellent. No enlargement of axillary or cervical glands. Rontgenogram of right shoulder negative for bone change. Rontgenogram of chest was negative for lung metastases.

Operation — Intrathoracoscapular amputation. Pathological examination revealed a grayish, soft, oval tumor which occupied the entire posterior scapular region. It came into contact with the bone above and was separated from it below by the infraspinatus muscle. The tumor protruded forward between the glenoid and coracoid processes making an egg-sized mass extending into the axilla. It produced little bony erosion where it was in contact with the upper portion of the bone. The muscles of the region were extensively invaded by the tumor and at the level of the spine of the scapula it infiltrated the deeper portion of the skin.

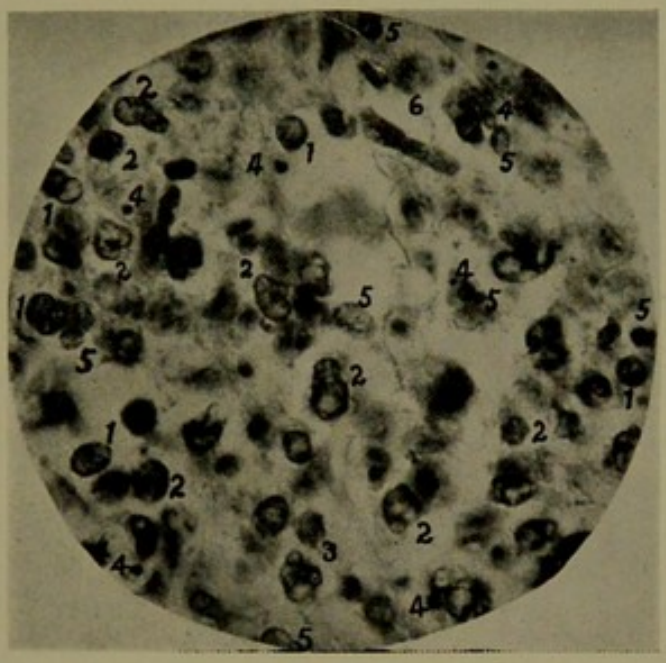

FIGURE 21

FIG. 21. Pure Cancer Cytology in a case reported as Undifferentiated Round Cell Sarcoma.

Microscopic examination showed the entire tumor to be extremely cellular. It was composed largely of medium sized round cells with more or less irregularly shaped nuclei. Hyperchromatic nuclei were common but mitotic figures were scarce. There was practically no intercellular substance present aside from blood vessel. Sections from a deeper portion of the tumor showed much necrosis.

Diagnosis — Paraosteal undifferenitated round-cell sarcoma.

Post-Operative Course — There was an uneventful recovery from operation and the patient has remained well and free from signs of recurrence up to now, ten years nine months after operation. —Dr. Dallas E. Phemister, Undifferentiated Round-Cell Sarcoma, Annals of Surgery, January, 1931."

ANALYSIS OF THE CYTOLOGY IN FIG. 21.

(The large round embryonal cellule both normal and cancer, in the process or stage of its disintegration or breaking up, releases its plural nucleoli, now the cellulettes, singly and in clusters).

1. In Fig. 21 the large round (1) cellule; in its final dividing seems to dissolve in its fluid environment much like a cube of sugar would, in particles; thus these triangular (3) and other clusters (2) of nucleoli, now cellulettes, would remain for a moment or two still clinging together, soon to separate and go on to the small (4) neo-round cellule stage.

2. The cancer cellulettes. In cellule one it is readily seen as disintegration of the main large cellule (2) occurs; that several cellulettes may still cling together in cluster form, at times.

3. In triangular cluster (3) form; for the normal see Fig. 6 (7) 3rd to 4th week, in blood island; Fig. 9 (1) in blood sinus at head of cord, 5-6th week. Cellulette formation is also readily seen in other large cancer cellules (1) (2) (3) here, about to break up. Also trace in and through Fig. 14 (1902); Fig. 15 (1928); Fig. 21 (1931); Fig. 25 (1932).

4. The small round cancer cellule form, successor to the cellulette. For normal see Fig. 7 (3, 4).

5. The medium or transitional cancer cellule; see Figure 19 in a glioma, there all other cancer cellules well and clearly seen; for normal type see Fig. 7 (2).

6. The long narrow link-chain cluster of cancer cellulettes and small round cells; others see in Fig. 19, a glioma; Fig. 20, in a neuroblastoma; for the normal type see Fig. 7, also Fig. 8 (3) above.

7. The spindle cell form, missing here, though so common in cancer (and s); in cancer see Figs. 17-18-19-20; for normal type, see Fig. 5 (1), at 2-3rd week; Fig. 6 (2) (3) at 3-4th week; Fig. 8 (3) below; Fig. 10 (2) 5-6th week; Fig. 11, in stroma of umbilical cord, 5-6th week; Fig. 12 (5) 3-4th week.

8. All pure cancer cytology.

OTHER OUTSTANDING FEATURES OF THIS CASE 21.

(1) The slow chronic growth of the tumor.

(2) Clinical history not very marked of the usual signs and symptoms of the acute virulent sarcoma.

(3) The passivity in expression; no metastases; chest or cervical, showing a remarkably mild degree in intensity of the ens malignitatis of cancer; suggesting that there are various degrees in intensity of malignancy in cancer expression.

(4) Operation of autosite is so successful that apparent cure follows with no recurrences.

(5) Cytology shows pure cancer, with profuse appearance of the distinctive cancer cellulettes, the diminutive causal unit of metastases and recurrences.

(6) Cure, still at last visit, after ten years nine months; no morbidity, comfort; no recurrence, no metastases; ideal result.

(7) Why this dormantcy, this abeyance of virulent cancer activity, during the whole period of pre-operation; and of cure; ten years nine months?

Because of a strong physiological resistance of the host tissues?

Was the normal physiological resistance of the body general so great as to neutralize the disseminated cancer cellulette potentialities preventing metastases? Or were the cellulette potentialities of a milder more passive nature than is usual in the acute rapidly growing painful cancers? Probably both.

(8) Do not cases such as this case suggest there is a form of Benign Cancer? Surprising as it seems, but no more so than when yesterday the similar question arose: Is there a Benign Form of the Malignant Cancer Syncytioma? The answer is, yes. Proof: the innocent placental polyp, slow growing and clinically apparent; usually so easy of eradication and cure, as with simple curage. In the case of the innocent placental polyp, where the Normal Growth Control is unhindered, there is no pathology; but where Growth Control is abeyant or lost, there the Malignant Syncytioma.

(9) If not operated upon at that time, how long would it have taken for this seemingly benign form of cancer to develop into the acute malignant form?

(10) The profuse cancer cellulette dissemination, presumably continuing over the whole time of the pre-operation period and also continuing, though dormant or abeyant, throughout the ten years nine months of cure.

(11) Fortunately here, over this period of cure, the cancer cellulettes were not aroused; analogously to the many "Embryonal Included Rests" in the body general.

(12) Again, there must be an Immunity Hormone against cancer in the body general, though apparently ineffectual in many or most cases. Was it of influence here?

(13) Case 21 is a case of cancer.

TWO MORE INTERESTING CASES OF SUSPENDED DORMANT CANCER; CURE, BUT WITH FATAL RECURRENCES AFTER TEN YEARS

An Editorial, American Journal of Cancer, March, 1932, Page 427, speaks of : "Disappointment in ultimate results. Final Straws, etc.

(1) Cancer of the Breast, cured for ten years; returns with an inoperable cancer of rectum.

(2) Cancer of Cervix, after ten years cure, returns with multiple metastases in the pelvis."

In all of these cases, the four Miners' Cases; these latter two cases and Case 21 of cure and suspended malignancy over so long periods of ten-eighteen years, two queries are instant.

Was it the reinvigorated physiological resistance following successful operation and removal of the autosite cancer that induced the neutralization, dormantcy of the suspended cancer process, especially of the malignant cancer cellulettes?

Was it the lowered ens malignitatis of the cancer cellulettes, following operation that induced the malignant dormantcy?

SURGERY

There can be no doubt that Surgery until today, in the operable cases, has proven to be the best weapon against cancer. Already, there seems a small murmur arising on the wave of international thought that Surgery has failed to keep step with other advances in means to successfully cope with cancer, as radium and Roentgen rays. It would be interesting to learn of the successes of the newer electric knife of today over the Pacquelin cautery of yesterday.

But such murmur is error and will and should decline. Compare surgical results of today with those of yesterday, the great number of 1. 2, 3, 5 and 10 years' cure without morbidity and recurrences; where yesterday these were all blanks! Even though treatment until today continues and grows as "Palliation and prolongation" both are sweet to the cancerite; yesterday offered nothing like unto it.

However great the strides that have been made with surgery, radium and Roentgen rays, effort must be especially directed to the

elimination of the cancer cellulette, that diminutive foe, so subtly disseminated and concealed in the intricacies of the tissues of the body general; the cause of metastases and recurrences!

But how, for here there is demand for a constitutional remedy, a remedy beyond the ready surgery, radium and Roentgen rays. How attain that remedy? Empiricism! and constitutionally; through the laboratory, sera etc., or through medical therapeutics, that will penetrate to the cancer cellulettes, in the uttermost recesses of the tissues of he body.

Apropos of sera; as these cases of cure, actual or suspended, serve as sources for seral experiment. In these cases there must be some influence, hormone or anti-body in their sera, that influenced to the dormantcy or neutralization of the ens malignitatis of the cancer cellulettes. Could that hormone or anti-body be secured? The answer to the thought means immense labors to the laboratory gentlemen! but it is worth it!

The same thought would hold true in the many experimental forms of mice cancer; where, withdraw the chemical or physical irritation and there is often complete recovery to the normal. Why? Some anti-body in the host serum? more especially aroused through the stimulation of the traumatic irritation.

That somatic anti-cancer-hormone or anti-body is influential in some few cases, even so far as effecting a spontaneous cure, is seen reflected in:

"The spontaneous healing of histologically confirmed malignant tumors cannot be denied. Betrachtungen über Geschwülste. Spontanheilung, Krebszellen in strommenden Blute etc. R. Hauser, Zentralblatt fur Gynekologie, Abstract, March 12, 1932."

What is this anti-body? Obtain it and Happily, Effort! a Cure!

ADDENDA

Brief Discussion of the Cell Reversion Theory As Implied In Transmutation, Anaplasia and Dedifferentiation; As Explanation for Origin of Cancer

As test in applying this theory to explain origin of the familiar Lip Cancer, Squamous Epithelioma.

Both application and explanation are in error.

In the work Origin of Cancer, among other assertions, supported by histologic evidence, it is mentioned that:

1.—Cancer cytology is identical with mater cytology.

2.—Transmutation cytology would be wholly different to mater cytology.

3.—Cancer cellule is progressive in evolution; to and through the stage of proliferation.

4.—Cancer cellule is not reversion from a higher typed cell or cellule.

5.—Cancer of the Lip can be and is proven by histologic evidence.

6.—Squamous Epithelioma, as cancer, can be and is proven incorrect by histologic evidence.

The discussion is assisted by two illustrations; another, a third. Fig. 24., photomicrograph has been developed and with the discussion is also shown here. This last photomicrograph becomes of much assistance in solving this problem, because of its greater magnification; its splendid clearness and distinctness in outline of detail, cell and structure; it makes easy contrast between theoretical suggestion and histologic reality; consequently critical decisions are more facile and exact.

Fig. 22, Cancer of the Lip, shows gross growth formation and destruction of tissue; a surviving quadrant of normal tissue, enclosing two so-called "pearls," laterally flanked by columns of pure cancer tissue; replacing in toto the normal squamous skin tissue; necrosis.

Figs. 22-23. There is irritation hypertrophy of the normal skin tissues. They show an invasion of a destructive process that amoeboids away normal tissues and replaces them by its own wholly different tissual formation, cancer. The slight local inflammatory hypertrophy is a mere iota in volume and force, compared to the invading new cellular tissue of the cancer. Now if there were a typical proliferation of the normal epithelium, would not the picture show a reversed process than that here; it would show a proliferation of epithelium and a great multiplicity of an atypical neo-offspring, where the epithelium would present a principle of aggression with inward-outward conduct? But in all pictures of Lip Cancer the rule is, the aggresive process shows, in direction and aggressiveness, a direction from without inward, as in Fig. 23. In fact, Fig. 23, pictures the cancer process as gradually enclosing from without inward, the still normal remnant squamous tissues. Were the process of epithelial nature the picture should show aggressiveness in direction, the other way around.

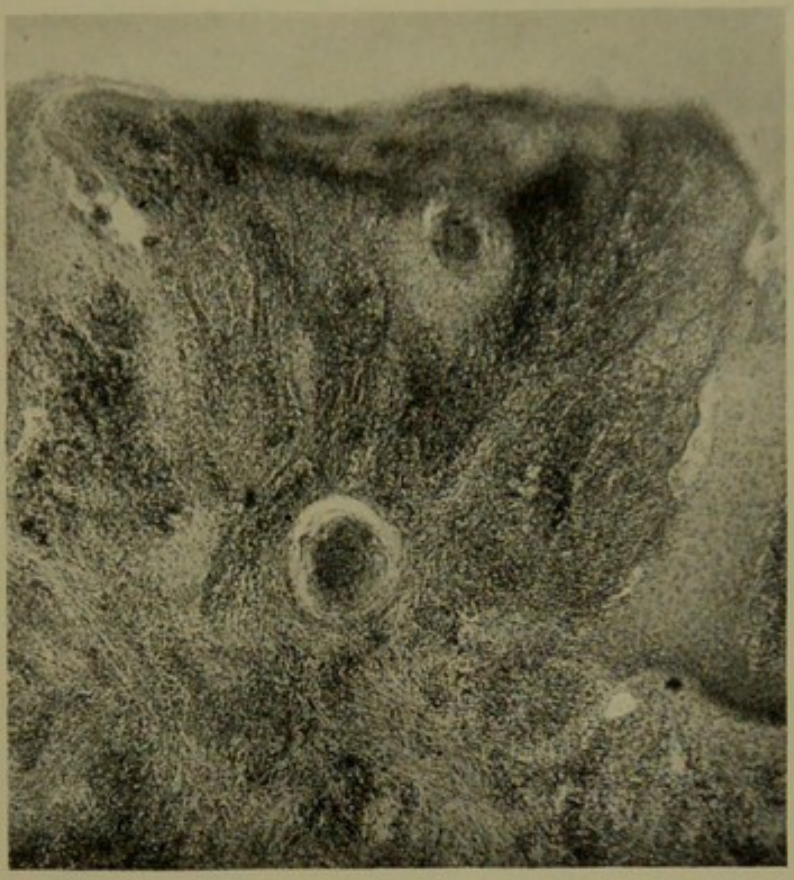

FIGURE 22

FIG. 22. Lip Cancer. General view; laterally cancer columns bounding quadrant of succumbing normal squamous tissues; two pearls; necrosis.

Particularly note here. This increase in the local epithelium and other cellular inflammatory hypertrophy, here or in any other tissues similarly situated, never gives birth, proliferates an offspring cellule that induces metastases or the ens malignitatis of cancer; nor is there epithelial amoeboid digestion of its environmental tissues and hence great proliferation of epithelium cells as is found in cancer and its cellules.

Again here Figs. 22-23, there is picture of a new cellule, offensively invading as aggressor; and the epithelium as slowly, passively, defensively disappearing before and because of some destructive absorptive power of the new invading antagonistic cellule; and in this process the normal tissues in great part wholly disappear to be replaced by the new process tissue, the cancer.

The pure epithelium never conducts, amoeboids and replaces in this manner. But primal matrix embryonal cellule (normal) does, as has been explained; for example see normal amoeboid villus con-

duct in villus attachment to the normal maternal tissues; the decidua in intrauterine pregnancy, Fig. 3; or the mucous membrane of the tube, or the peritoneum in the extra uterine pregnancy.

It is the primal matrix embryonal cellule of the syncytium that normally amoeboids maternal environmental tissues for pabula to the ovum in general and to the embryo in particular. When this primal matrix cellule, or its descendant, normal, in pre-natal or post-natal life, possessing these physiological amoeboid characteristics, is injured then it aberrates in proliferation and function, and becomes the great hyperproliferate destructive cellule of the cancer (and s).

In the normal cell proliferation, differentiation is controlled by normal growth principle. In cancer, as mentioned, growth control is lowered, abeyant or absent, hence great proliferation only. No further progressive differentiation into normal ultimate tissue.

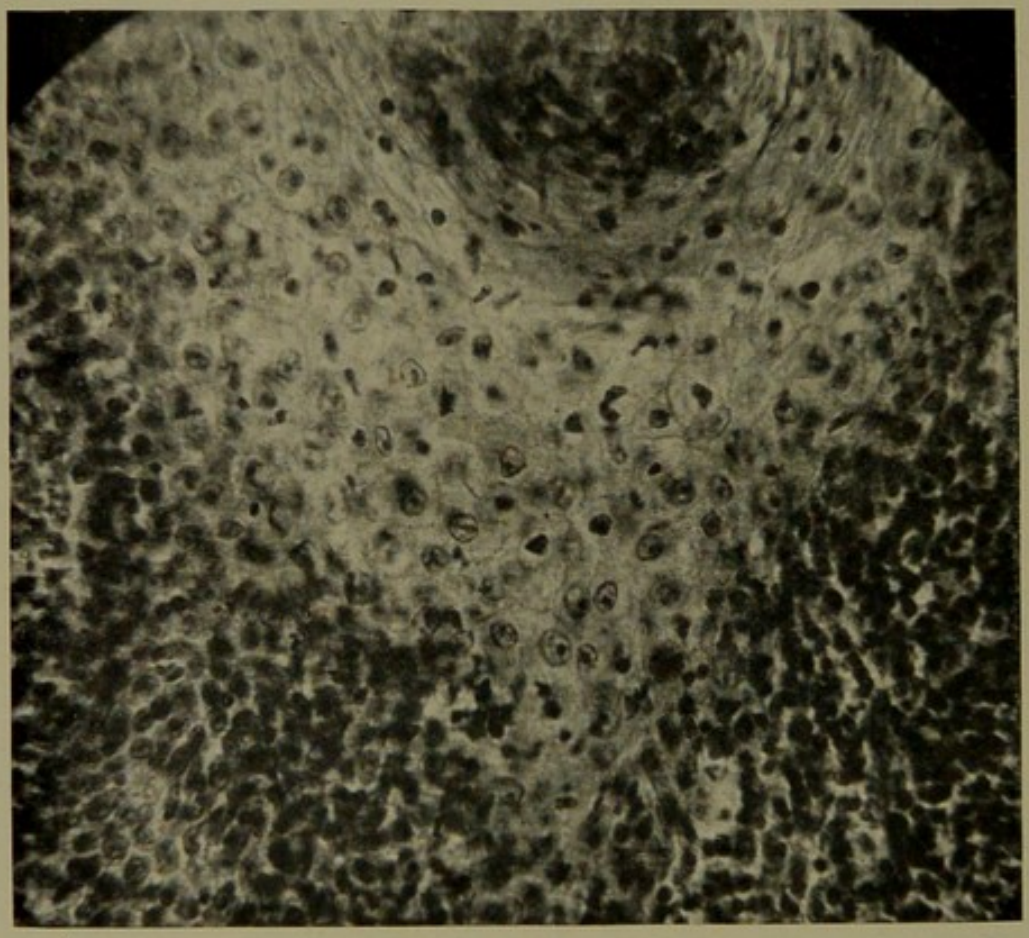

FIGURE 23

FIG. 23. Normal area about upper pearl enlarged, to show contrast between normal squamous cell tissue rapidly fading and replaced by invading cancer tissue. Compare normal squamous cells, rapidly disappearing because of amoeboid cancer process; with cancer cellules best seen in lower left corner of illustration.

Note how different is the epithelial cell, Fig. 24, in structure, form and appearance, from the cancer cellule, Figs. 23, 24, 25 and Figs. 14 to 21. The cancer cellule runs true and identical to its ancestor mater cellule, the primal matrix embryonal cellule, Fig. 1, in form and structure; the epithelial cell is a far maturer picture and differs from the primal immature cellule and the cancer cellule.

As mentioned, the cancer cellule is identical in structure and form with the primal cellule to and through proliferation; see Fig. 23. In proliferation the cellule aberrates, no further differentiation characteristics of the normal cellule.

One phase of this continued differentiation of primal cellule is the progressive differentiation of the primal matrix multinucleolated cellule blood corpuscle, Fig. 1-9, through the erythroblast, Fig. 11, to the mature non-nucleated blood corpuscle, the erythrocyte. Nothing like this in the epithelium.

Epithelium transmutation would be wholly different in cell form and structure and retrogressive. How grossly different the great squamous epithelial cell, Figs. 23-24, from the small cancer cellule, Fig. 23, in left lower corner. The epithelial cell is of wholly different outline, structure and maturity, than the immature primal embryonal cellule of the cancer. Is not the epithelial cell a differentiated cell more especially of the embryo, per se, rather than that of the ovum, sans embryo, extra-embryo? The cancer cellule is identical with its mater, the primal cellule; as mentioned the cancer cellule is somewhat coarser in structure and appearance, not so refined in texture as its ancestor, the primal cellule.

This is the interpretation these illustrations show. In the familiar Fig. 23, this passive, defensive, dying, disappearing attitude of the normal squamous cell tissue is well shown over against the offensive, amoeboid, very lively replacing conduct of the cancer cellules. Would not great proliferation of the epithelium, granted, even bearing a numerous transmuted offspring; would that process not show, a picture of great activity rather than such a passivity, and lethal conduct? Undoubtedly.

Fig. 24 is introduced here as an enlargement and in further proof of great differences between the squamous cells and those of the cancer cellules; the squamous tissues here are made up of large cells, mononucleated with prominent nucleolus, Fig. 24 (1), and with sur-

rounding cell cytoplasm. No evidences whatever of proliferation. How different the active growth proliferation of the cancer cellule.

The squamous cells, Fig. 24, are larger than usual because of defensive inflammatory swelling caused by the cancer irritation, Fig.

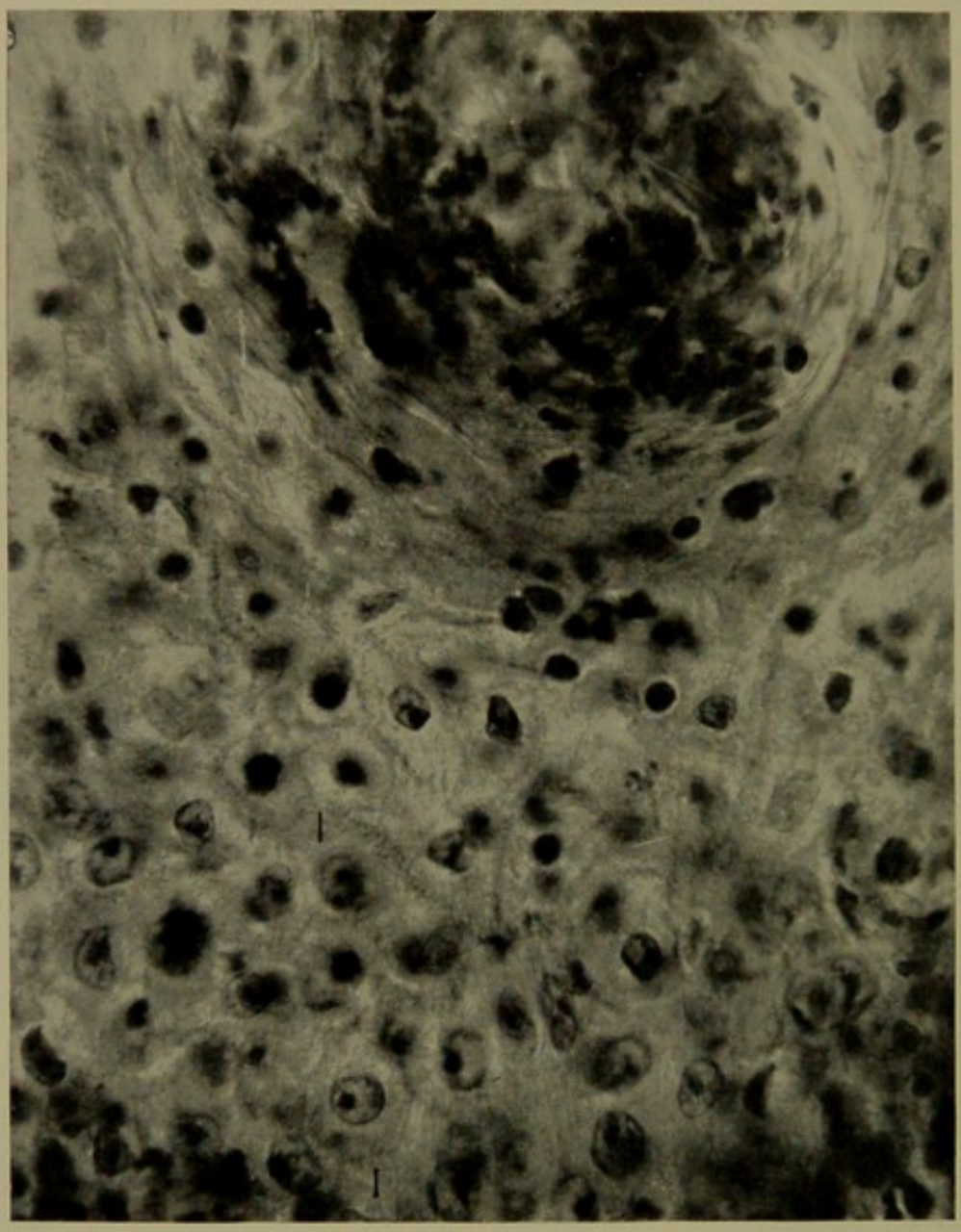

FIGURE 24

FIG. 24. The normal squamous cells enlarged, the better to show contrast with the cancer cellules. Observe perfect cell form of squamous cells, a single nucleolus within the nucleus surrounded by its characteristic cytoplasm; also note their fixed compact form in tissue structure. On the other hand, observe the cancer cellule; it is multinucleolated without surrounding cytoplasm, free as a rover cellule, loose, not in compact structural form as in the epithelial squamous tissues. Epithelium is a mature form of cell evolution; cancer cellule immature. The size of the whole cancer cellule is about the same size as a nucleus alone of the normal squamous epithelial cell.

24 (1). The cell form and construction is yet well maintained and outlined; the area as a whole suggesting gradual contraction, and disappearance like in the other great replaced areas of normal tissues, because of the cancer process.

To stress:

Nowhere are there signs of a mature epithelial cell proliferation or of such a proliferation of epithelial cells, analogous to the great glaring proliferation seen in the process of cancer cellule proliferation and multiplication. The squamous cell perfect, a single nucleus with its single nucleolus, Fig. 24 (1), surrounded by characteristic cytoplasm, without evidence of proliferation. On the contrary, as contrast, the cancer cellule is multinucleolated and without surrounding cytoplasm. And everywhere the normal areas grow lighter and smaller because of the amoeboid process of the invading cancer cellule. On the contrary the cancer areas are on the increase. How different the epithelial cell, it lethals and disappears; the cancer cellule much smaller remains, true to its ancestor in form and structure.

Squamous Epithelioma is error as cancer. The term refers to the locus of the cancer process; but not as to the origin and nature of the ens malignitatis, the cancer cellule.

Fig. 24, squamous cell area enlarged; shows beautifully enlarged squamous cell (1), with mono-nucleus and mono-nucleolus, surrounded by a cell limiting cytoplasm. Squamous cells here look very much like the large technical decidual cells in the decidua vera and serotina of pregnancy, and conduct very much the same, see Fig. 3, being destroyed and absorbed away; in the decidua, however, only in part.

Fig. 25 cancer cellules, from a metastatic lymph nodule. These cellules are common in cancer; the spindle cellule is missing; for normal prototype in the normal see Figs. 4 and 7.

Nothing like these cellules seen in epithelium cytologic division, from infant to the mature epithelial cell.

Transmutation epithelial conduct would have to go back beyond the phase of infant epithelial formation. Through how many links from the mature cell back to the immature commencement? Which link, unknown, and why that link? in the chain of ultimate mature epithelial differentiation?

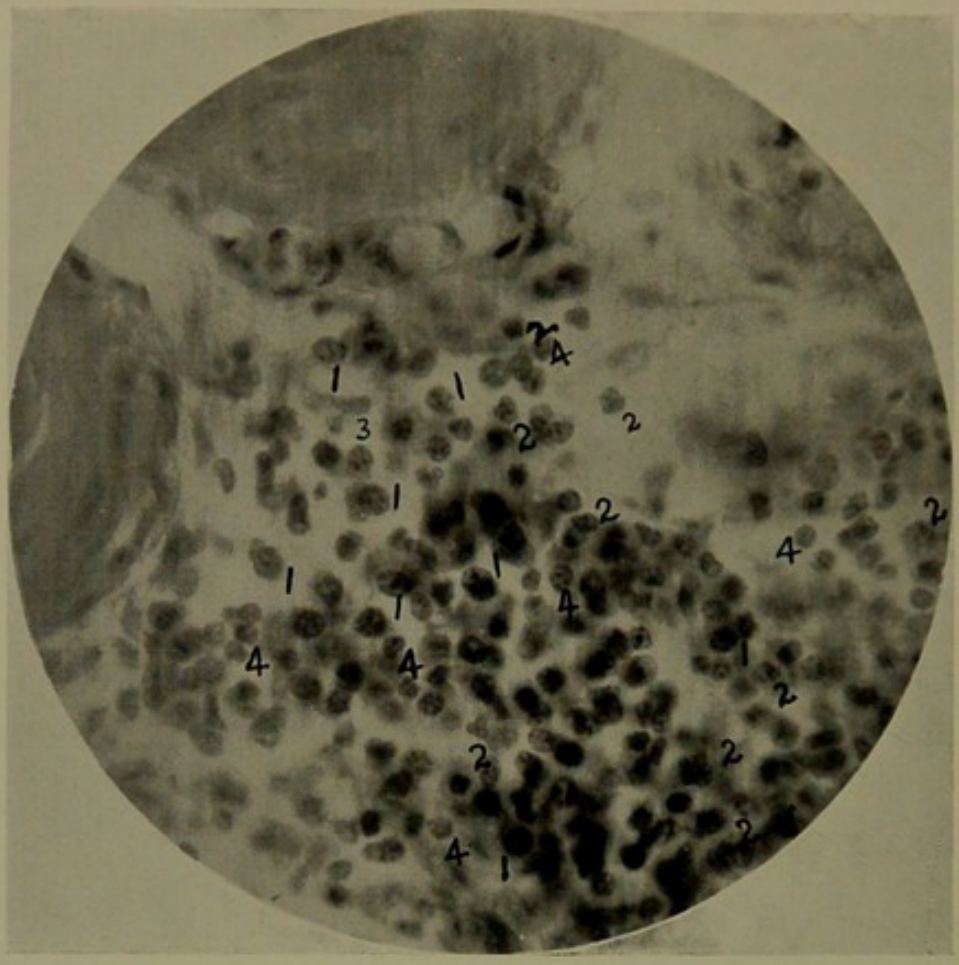

FIGURE 25

FIG. 25. Cancer cellules from a metastatic lymph nodule; no surrounding limiting cytoplasm. Beautiful clear illustration showing Amitosis, Direct Division in the cancer cellules. Note this amitosic feature in other illustrations; common in occurrence; no room for doubt as to the occurrence of this form of cell division.

(1) Large round cellules with multiple nucleoli. (2) Ripe large round cellules extruding nucleoli, singly and in clusters, now cellulettes, soon neo-small round cellules. (3) Faint long-link cluster cellulettes, neo-small round cellules. (4) Medium round or transitional cellules, all multinucleolated without surrounding cytoplasm. Use good hand glass.

Finally even if that suggested earlier link, manifestation of that mature cell, epithelial or other cell, could possibly be fixed, that earlier unit could not proliferate into anything but its normal self or, its differentiate, the higher ultimate mature epithelial or other mature cell. For transmutation to hold it must show a fixed unit in retrogressive cell evolution and then aberration in cell structure and cell function, which transmutation as a theory might do, but practically, histologically, it cannot do.

In the primal matrix cellule origin and descent of the cancer cellule is shown, histologically, direct from such primal cellule. In the

second stage of cellular evolution, proliferation, some insult or injury occurs and causes in a few cellules aberration; the majority of such primal cellules, go on to further normal differentiated ultimates; the few cellules aberrated, because of such injury or insult in their intrinsics or chemics, then become the at first few cancer cellules, as explained above; then prolific hyperproliferation and destruction. Refer here again to discussion under Fig. 4 above.

To further assist in following this discussion an excerpt from Origin of Cancer, Cancer of the Lip, is presented herewith.

Fig. 22. Cancer of the Lip. To show growth formation and destruction of tissue; superficially and laterally ulceration; at the right border a surviving area of squamous epithelial tissue. Two prominent "pearls" seen in midst of cancerous process. Surrounding the lower part of the upper "pearl" is a small area of normal squamous tissue, succumbing to amoeboid-digestive transformation and replacement, into cancer tissue. All types of cancer cellules are to be seen in this specimen though only the three principal types just here; the large, the medium, and the small round cancer cellules.

Fig. 23. An area of Fig. 22 enlarged, shows an imperfect surviving squamous area undergoing cancerous replacement and transformation. It is noticeable here that though the nuclei of the squamous cells with their single nucleoli, seem to stand out well at this stage of cancerous destruction and transformation; the suggestion is that the cellular surrounding cytoplasm of the normal cell is the first structure to undergo lysic solution and disappearance; the limiting membrane of the nucleus offering a stronger resistance to the amoebic action of the cancer cell; the last structure to disappear apparently, seems to be the nucleolar substance of the nucleolus. Faint cell outlines of a squamous character seem yet to be suggested in some few cases. The nature of the cancer cellule, large, medium and small round cellules, may be made out in the left lower corner of this specimen. Use good hand-glass; splendid detail.

It will be most interesting and not a little puzzling to compare these lower left corner cancer cellules in structure and function, to those normal types seen in the normal villus conduct as these latter stream over into and amoeboid the normal decidua basalis, in the process of the villi attaching themselves to the decidua; as fuller shown and discussed in the here Fig. 3.

How same, yet different seem these expressions in function, form and ultimate construction.

Why? Normal growth control hormone or principle being present in the one; but not so well marked in the other.

In endeavoring to solve these difficult histologic-pathologic problems, explanation, histologic-pathologic thought, seems always to look back to the embryo, per se, for origin of the primal embryonal cellule, so-called. But this is error as explained above.

The primal embryonal cellules, Fig. 1, are extra-embryonal, ovum; but not embryonal, per se.

As explained before, even the embryo, per se, depends for life and continuity upon extra embryonal function; that is of the ovum, its chorion, more exactly speaking its syncytium. The cellules of the syncytium seize upon environmental tissues by amoebic action, thus providing nutrient pabula; digestion and assimilability, is furthered by chorionic conduct as a whole; likewise transportation to the embryo is initiated and continued by chorionic function through the primal white blood circulation. The embryo transforms chorionic conduct, cellule and fluid; it does not imitate them, or its own necessities. Therefore, histologic-pathologic philosophy must look to the ante-extra-embryo centers and developments for origin of primal cells and fluids. And there, among other features, is found the primal uncolored blood circulation, made up of differentiating syncytial cellules and plasm won by syncytial conduct as a whole, furnishing life-giving pabula to the embryo; not the other way around, embryo to ovum. Hence origin of embryonic cellule, normal and cancer, needs to be sought extra-embryo.

Further in Figs. 22-23. Cancer of the Lip. The diagnosis is correct. But if it is termed Squamous Epithelioma or Epithelioma of the Lip, that term is incorrect.

The statement is made here especially to encourage opposition in thought, for just here before this clearly defined picture of lip cancer, as in all squamous cancer, the reader can readily see and easily differentiate that the squamous epithelial cell is an entirely differing formation, in structure and function, than that of the cancer cellule. Again note the perfect squamous epithelial cell here, a nucleus surrounded by its cytoplasm; on the contrary, the cancer cellule is like a nucleus-cellule with multiple nucleoli; hence nucleus-cellule but without surrounding cytoplasm.

There is no typical proliferation of epithelial cell here. Here there is death and lysis of the epithelium, entire dissolution; hence impossible, an active epithelial proliferation from dead epithelial tissue. And this death of epithelium is caused by the chemico-physiologic characteristics of the entirely differing and invading cancer cellules; not an atypical epithelial proliferation, transformation. As is well known, at one time it was thought it was a transformation caused by parasitical conduct.

The cancer cellule has the power of amoeboiding its environment; that is seen so well again, here in Figs. 23 and others. This means paresis, destruction and death of the cells of its environment. As remarked the epithelium does not amoeboid its environment. At the same time this destruction serves as food to the cancer cellule. Thus epithelium and all other contiguous tissues the cancer contacts, disappear in the maw of cancer digestion and there follow great cancer cellule proliferation and cancer life, but at the cost of the destruction of normal tissues, epithelial, connective tissues, bone, nerve, etc. There is no epithelial transformation or atypical epithelial proliferation. There is entire destruction and replacement of the old by a new entirely different cellule growth, the cancer.

And what applies here to cancer of the lip, applies to all other tissues. The steps of cancer are invasion, of the small round cell primarily, then adolescence; some defense hypertrophy of local host tissue due to offensive invasional irritation, as seen so well in the dipping down hypertrophy of the papillae in the skin cancer; followed quickly by death of that part and lysis; amoeboid destruction of normal tissues; disappearance of normal tissue by amoebic digestive function of the cancer cellule; great proliferation of the cancer cellule with great disappearance of normal tissue; replacing of the normal tissue by cancer tissue; finally exhaustion and death of both host and cancer.

The cancer is not epithelial, a mature tissual expression. The cancer is an immature cellule expression, extra-embryonal, ovum, in origin; not embryonal, per se."

CELLULE (EXTRA EMBRYONAL): CELL (EMBRYONAL) DIVISIONS.

Of outstanding import to the histologist as well as to the pathologist.

Bearing in mind the multinucleolated picture of the primal mature cellule and its descendants, also the multinucleolated picture of the cancer cellule. But nowhere is there a picture of such cellules with a single nucleus or nucleolus that multiplicity of nucleoli might be attributed to proliferation or division of that single nucleus. Multinucleolation seems to be the normal birth status of these cellules; as inheritance from their corona radiata—zona pellucida cellule forbears.

In many of these illustrations, normal and cancer, shown in this work, opportunities are offered to observe and critically so, cellular and cell divisions; and therefore here advantage wishes to be taken of these opportunities.

One of these divisions, Amitosis, opinion regards as of doubtful actual occurence. The cellular divisions here are clear, distinct and plain, unusually fortunate conditions in minute histologic illustrations.

The large round cellule is shown as a nucleus-cellule with multiple nucleoli, Figs. 1-4. As the large round cellule divides, nucleoli now cellulettes, neo-small round cellules are freed, singly and in clusters. This form of cellule division would suggest it be described under the descriptive term Amitosis or Direct Cell Division.

Such features of cellule division are seen in the normal and Figs 1, 3, 4, 5, 6, 7, 8, 9 (blood) 10, 11, 12, 13; in the cancer cellule seen in Figs. from 14 to 25, especially in Figs. 14, 15, 21, 25.

These features of division and of multiple nucleoli were shown and illustrated in 1906 in "A New Form of Blood Cell. Amer. Jour-Obstet. New York, Vol. 53, No. 4, 1906."

But the 1906 demonstration and discussion seems not to have been recognized or considered under the description of "Cell Division" as given in standard histologies.

The divisions of the early primal immature cellule, Fig. 1, and its descendants, both normal and abnormal, the cancer, must be recognized, as such divisions continue throughout all life, pre-natal and post-natal; such division would be covered by the term Amitosis, Direct Division, Multiple Form; this conduct of division being a fact, not doubtful as spoken of in text book Amitosis; see also Fig. 8.

The nucleus-cellule contains multiple nucleoli, Figs. 1-4; as cellule maturity approaches the cellule gives off or extrudes nucleoli, Fig. 25 (2), as the new cellulettes, neo-small round cell cellules;

when division or breaking up in toto occurs, the mature cellule breaks up or divides into single or multiple cellulettes, at times in cluster form; for each nucleolus one cellulette, one neo-cellule offspring of the mater cellule. Both mater cellule and offspring show no characteristic surrounding cytoplasm; therefore no division of such technical cytoplasm occurs.

Scanning these illustrations, both normal and abnormal, cancer, they easily show this form of division, Amitosis, as of regular occurence and prolifically so; there is no room for doubt as to the occurence of Amitosis.

As perspective of histologic acceptances and thought as given in standard books, a few quotations are given here:

"Cell Division. The usual mode of cell division occurs through a highly complex process known as karyokinesis, mitosis, or indirect division. This is in sharp contrast to the much less frequent direct division or Amitosis. Page 14.

"Amitosis, page 18. There has been much discussion as to whether this process actually occurs under normal conditions." — Maximow-Bloom, Histology, Saunders Co., 1930.

"Amitosis. In typical amitosis the nucleolus first becomes binucleated and then divides. This is followed by nuclear division, each resulting nucleus enclosing one of the nucleoli. Nuclear division is followed by cytoplasmic division. This typical condition is rarely realized. It was first described by Remak (1841) for blood cells." Jordan, Histology, Appleton and Co., 1931, page 19.

So far as detail of steps of division in Amitosis as described by Remak; there is nothing to hand that could be reported here; apparently though confirmation.

But as result of Amitosis, there is conclusive evidence of this Direct Division in the Direct Division, Amitosis of the Uncolored Multinucleolated Blood Corpuscles of the Primal White Blood Circulation, to the 6-7th week. And this is seen in Fig. 9 (1), Fig. 11 (S.E. border) picturing Amitosis in such Blood Corpuscles in the Blood at 5-6th week of human ovum development. There are many such evidences of Amitosis to be found in villi and chorion stroma tissues. In these instances of Amitosis, division consists in extrusion of nucleoli from the large round ripened cellule while still intact; as may be seen in following the circumferences of such large round cellules in various illustrations here, especially in the cancer. See Fig. 10.

But, as mentioned, when the cellule divides in toto or bursts apart, nucleoli are released singly or in clusters, soon to break up. These freed nucleoli giving origin to the Blood Celluettes (The Blood Plattelettes?); the Primal Cellule Cellulettes; in cancer division, to the prolific Cancer Cellulettes or sub-small round cancer cellules. Division, proliferation here in the extra-embryo cellule is not nucleus or part nucleus; it seems entirely nucleoli.

Amitosis in these illustrations seems far more numerous than karyokinesis.

RÉSUMÉ

Cell Division. From foregoing illustrations three forms of cellule and cell divisions are shown, and should be accepted.

1.—Indirect, Mitosis or Karyokinesis, Fig. 8, Cell Division of common occurence and well known in normal and cancer expressions.

DIRECT, AMITOSIS, CELL DIVISION AS SHOWN ABOVE OCCURS IN TWO FORMS:

2.—Direct Division, Amitosis, of the multinucleolated cellules; the normal primal cellules, Figs. 1-4, etc., and the numerous cancer cellules, Figs. 14-25.

As explained above this form of Direct Division is prodigious, clear and without doubt. Here the nucleoli exist pre-cellule division and are extruded from the ripening and the bursting ripened cellules, unchanged, as the now new cellulettes, the pre-sub-small cellules of the normal mater cellule and its cancer offspring.

3.—Direct Division where nucleus, nucleolus and cell plasm divide to form the new cell, Fig. 26. Instances of this nature were shown in Origin of Blood, Fig. 83, where two bi-nucleated red blood corpuscles, unfinished and yet in process of differentiation to the finished mature non-nucleated erythrocyte form, are to be seen midst multitudes of finished mature erythrocytes, in the area vasculosa of a 7-8th week ovum.

Another example of this Direct Division in the blood cells, may be seen in a cross-section of a villus blood vessel, where the so-called Hoffbauer blood-cell is seen; likewise an unfinished bi-nucleated blood cell, in process of further differentiation to the erythrocyte. See Histologies.

As the plate of Fig. 83, Origin of Blood, here Fig. 26, is conveniently at hand, this illustration is added here to further assist in appreciating that this form of Direct Division actually occurs.

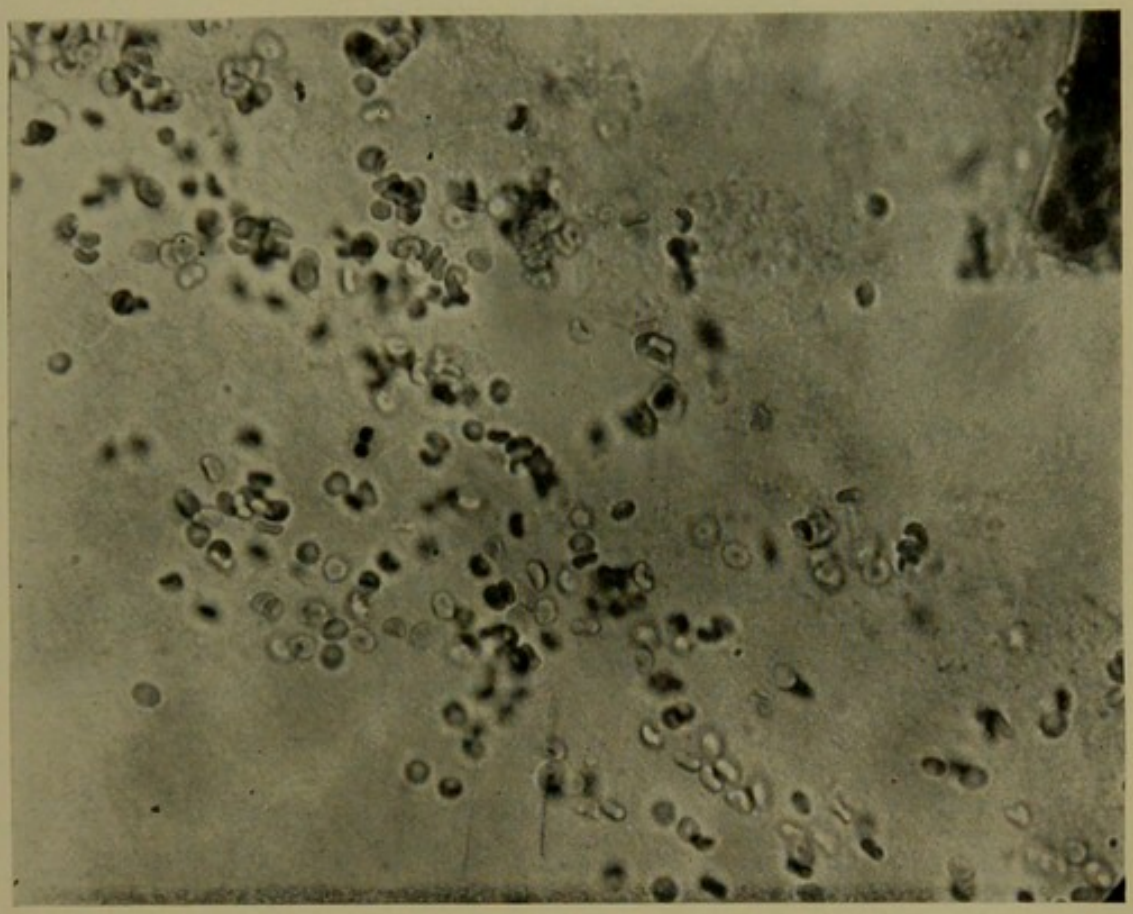

FIGURE 26

FIG. 26. Unfinished bi-nucleated red blood corpuscles; Remak's Direct Division in blood cells.

Two unfinished bi-nucleated red blood corpuscles. When cleavage occurs and extrusion of the nuclei, there results the mature finished non-nucleated red blood corpuscle, the erythrocyte.

FINALLY, THE SYNCYTIUM WITH PRIMAL NUCLEUS-CELLULES AND THEIR NORMAL DESCENDANTS; FROM PRIMAL DAYS, FIGS. 4, 5, 12 AND 13, TO MATURITY, AT TERM, FIG. 27.

Fig. 27. Transverse sections of chorionic villi at the end of pregnancy to show:

Syncytium with nuclei at term, now referred to as remnant; yet syncytium but attenuated and shorn of its early richness in quantity and importance, also some of its physiologic function. See and compare this Fig. 27, senile, with the active Figs. 12 and 13, where syncytium is also at the height of its physical and physiologic functions.

Nuclear groups made up of nucleus-cellules, again seemingly shorn of much of their activities. Here they picture a nucleus-cellule with multiple nucleoli and without characteristic surrounding cytoplasm, true to their ancestor nucleus-cellules, Fig. 1.

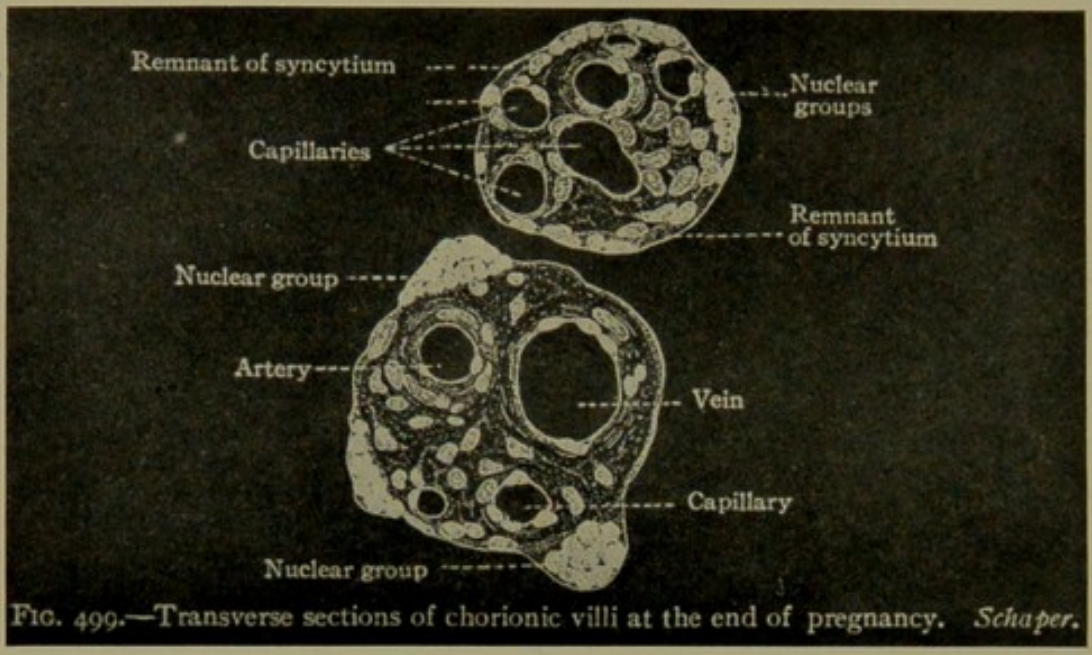

FIG. 499.—Transverse sections of chorionic villi at the end of pregnancy. *Schaper.*

FIGURE 27

FIG. 27. Syncytium and syncytial nuclei, primal cellules, at term.

Note same nucleus-cellule outline, with multinucleoli but without surrounding limiting cellular cytoplasm as in a mature epithelial cell. In form, structure and potential powers true to primal ancestors, the primal multinucleolated nucleus-cellules of the primal syncytium.

As descendants of the primal matrix embryonal multinucleolated nucleus-cellules of primal and subsequent syncytium, these nuclear cellules are true in physical form to their ancestors, as mentioned, the primal cellules, here Figs. 1 and 4. Physiologically they seem less active, more subdued, yet possessing all their latent primal potentialities of functions.

The large round spinal cancer cellule and these nuclear-cellules are similar in form, though not wholly so in function.

This similarity in physical form between the here normal nucleus cellule and the abnormal nucleus cellule of cancer is plain and shows direct ancestor and descendant in qualities and form and to a certain extent only, in conduct. Every cancer shows these multinucleolated nucleus-cellules as aberrated normal cellules in its cytology.

These nucleus-cellules of these nodules wander like their forbear cellules, Figs. 12 and 13, from the syncytium into the tissues and blood-vessels of the villi and chorion, and are to be seen in the blood

vessels of the villi and chorion at this time. Thus they have access to the blood circulations of the fetus-neonatus, and are already in the blood circulations and thus become disseminated throughout the tissues of the new born at birth. Thus they continue as such primal wandering cellules and function as such throughout subsequent post-natal life.

Injure one of these primal wandering cellules as through traumatism, irritation or other insult then there is aberration in the cellule, viz. the cancer cellule.

Fortunately not all these cellules are injured at any one time. Hence only a few are aberrated and become cancerous.

These researches are fortunate in being able to show the primal matrix nucleus-cellules in direct descent and histologically so, in unbroken chain from the pre-ovum corona radiata-zona pellucida nucleus-cellules of the ovule, through the primal syncytial cellules of the ovum, to the ultimate nucleus-cellules of the syncytium at term. It is from these cellules that the cancer cellules are descended.

DESCENDED FROM THESE PRIMAL MATRIX NUCLEUS-CELLULES OF THE OVUM ARE:

1. The syncytial nucleus-cellules of the primal and subsequent syncytium. Figs. 1, 3, 4, 11, 12, 13, 27.
2. Descended from these primal matrix embryonal syncytial cellules are the primal uncolored multiucleolated type of blood corpuscles of the primal white blood circulation. Figs. 1, 4, 5, 9, 11, 12.
3. These primal blood-corpuscles differentiate through the red mono-nucleated erythroblast, Figs. 11-13, to and into the finished ultimate red non-nucleated blood corpuscles of blood corpuscle maturity, the erythrocyte; about 6-7 week, Fig. 26.
4. When these primal or their descendant primal matrix syncytial cellules are injured or traumatised in pre-natal or post-natal life; whether these matrix cellules be wandering, and there injured or traumatised among the ordinary cells and tissues of the body, or among the special cells and tissues, as the pigmented cells and tissues, as of the choroid, naevi or melanomata; when so injured these matrix nucleus-cellules aberrate in their intrinsics and chemics, but retain their same cytologic types and forms and become the aberrant matrix syncytial embryonic nucleus-cellules, the cancer cellules, see Figs. 14-25, seen in all cases of cancer (and s).

With all their varying degrees of conduct and malignancy; such malignancy and conduct is due to direct inheritance from mater cancer cellule to offspring cancer cellule in the stage of proliferation; not a direct transference of the ens malignitatis from one cell to another, or between cellules of various kinds.

5. The erythroblast; that important differentiated connecting link in blood corpuscle differentiation, between the primal uncolored syncytial type of blood corpuscle and the ultimate mature red colored hematinized blood-corpuscle, the erythrocyte. It is through the growth of the erythroblast that hematin is introduced and continued in the blood corpuscles. No hematin before the erythroblast.

Whence comes the erythroblast, is it embryo or extra-embryo? It is extra-embryonal in origin; the embryo will prove this assertion. But its discussion would be too broad and extensive to be entered upon here; if not anticipated, it will be considered in a separate research in the near future.

In cancer cytology; though cancer is extra-embryo in form and type of cellule; there seems a dividing line here in the erythroblast, also extra-embryonal, for until today no features of an erythroblast are seen in cancer (and s) cytology.

CONCLUSIONS

Cancer and sarcoma are one.

Cancer is not epithelial.

Sarcoma is not connective tissue.

Today it is easy to remove the autosite cancer, in many cases.

The problem in cancer cure today, is, how neutralize, annihilate the cancer cellulettes, containing the ens malignitatis! This would reach all cases operable or inoperable.

Case 21, Undifferentiated Round Cell Sarcoma, is a case of Round Cell Cancer, for only pure cancer cytology is found there; all forms of cancer cytology being present excepting the well known spindle form, so common in cancer (and s); no one form of cancer cellule being predominant.

Origin of cancer is now beyond the stage of theory. Origin of cancer has been traced, shown and demonstrated through illustrations of histologic cytology, as being descended from the primal matrix embryonal cellule of the primal and subsequent syncytium.

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