Since the suggestion was made to me by Dr. Carl C. Warden, who at the time was working with gonococcic vaccines, I have thoroughly washed all bacterial suspensions used in the preparation of vaccines, in the high-speed centrifuge, until the supernatant salt solution (0.45 per cent.) no longer gave a biuret test. The ninhydrin reaction has not been used because of its greater expense. Two or three such washings in the centrifuge will usually suffice to remove most of the extraneous protein. The vaccines so prepared I have called "washed vaccines," since such suspensions are practically free from all unnecessary and undesirable proteins. Washed vaccines possess the cellular elements necessary to immunization, but are free from those proteins which, in many preparations, have been responsible for relatively severe local and general reactions. Washed vaccines are much more suitable for intravenous administration.

Such washing is applicable in the preparation of all bacterial suspensions for vaccine purposes, with the exception of such fragile organisms as the pneumococcus and the gonococcus, the ordinary strains of which autolyze rapidly. Recent work by Sobernheim, by Seligmann and by Warden with more stable mutation forms of organisms offers the hope that less autolysis will take place on thorough washing and that these mutation suspensions may be handled in a manner similar to the suspensions of the more stable organisms.

For purpose of standardization, a suspension of the living, rather than dead, washed organisms is employed. The following method, which is preferable to the blood-cell-slide method of Wright because of greater accuracy, was suggested some years ago by Dr. L. H. Spooner of Boston, and has been used by me as a routine procedure with satisfaction: A small amount of the suspension is drawn into an ordinary erythrocyte mixing pipet, diluting 1:100 with a fairly deep-colored, thoroughly filtered, aqueous carbolthionin stain (the phenol (carbolic acid) content is 0.5 per cent.). The suspension in the counter is well shaken to secure even distribution of cells and a small drop placed on the shallow blood-platelet counting chamber of Helber-Zeiss. The organisms in twenty-five small fields are counted, and the sum divided by 25 to obtain unit value. The number so obtained is multiplied by the dilution 100, by 50 the depth of the chamber, by 400 the number of small squares per cubic millimeter and lastly by 1,000 to convert to cubic centimeters. For example, if forty bacterial cells are counted in twenty-five small squares, the unit value per square would be 1.6, multiplied by 100, by 50, by 400 and by 1,000 or 3,200 millions per cubic centimeter of suspension.

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Observation Must Precede Inference.—Take a group of students who are working at physical diagnosis and it is a constant struggle to keep them making observations and not giving inferences—usually from insufficient observation, if from any at all. No pains are too great to hammer home into the mind of every student the importance of keeping these two processes separate and not taking up the second until the first is as complete as he can make it. It is just as important for the practitioner as for the student, except that in the latter we are trying to form correct habits; the practitioner should have them.—Thomas McCrae, M.D., in the *Canadian Med. Assn. Jour.*

CANCER OF THE PROSTATE *

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The facts that the general subject of cancer presents even to-day one of our most serious and unsolved problems; that cancer of the prostate is a comparatively frequent and exceedingly distressful form of the disease; and the probability that by a more widespread application of the present available knowledge of this pathologic process the prevalent high percentage of disability and death would be considerably diminished, are advanced as sufficient reason for presenting a discussion of the subject with a contribution of data to the fund of experience.

The pathogenesis of cancer of the prostate is still a debatable question; the frequency of its occurrence, the usual sites of its original focus, its mode of extension and metastasis, are phases of the subject which are reasonably supported by facts.

This paper is based, first, on the examination of a series of 716 prostatic specimens removed at operation in the Mayo Clinic from Oct. 28, 1904, to June 1, 1914; secondly, on the pathogenesis of epithelial tumors of the breast, as view by MacCarty; and thirdly, on a review of the literature.

PATHOGENESIS

The opinions of various investigators with regard to the pathogenesis of cancer of the prostate are not uniform. The cause of this lack of uniformity seems to be due, at least in a great measure, to the different points of view from which the pathologic process is viewed.

Some authors maintain that cancer does not begin in a hypertrophic portion of the gland, that its presence here is an invasion from a focus situated elsewhere in the organ, usually in the posterior lobe. Others state that cancer occurs as an almost imperceptible transition from a benign adenoma. Still others, disregarding as unessential the consideration whether the associated process be one of general hypertrophy, adenoma or other pathologic condition, view, apart from its causation, the pathogenesis of prostatic cancer, likewise of cancer in general, as fundamentally a question of the histogenesis of epithelial cells.

A summation of the evidence adduced from the various investigations emphasizes the fact that cancer occurs in the atrophic, the apparently normal and the hypertrophic prostate. In the light of this accepted fact is seen an indication that involved in the development of the cancerous process there is a study far more basic than what is comprehended in a consideration of associated pathologic conditions. Again, the weight of evidence seems to prove that the disease begins most frequently in the posterior lobe of the gland. This seeming selectiveness of the process, however, is no more explicable in the prostate than are similar phenomena in other organs. The incident of original focus is aside from the underlying principle of pathogenesis, as advanced in the theory of the histogenesis of epithelial tissue.

* From the Mayo Clinic.

* Read before the Section on Pathology and Physiology at the Sixty-Fifth Annual Session of the American Medical Association, Atlantic City, N. J., June, 1914.

* Because of lack of space, this article is abbreviated in THE JOURNAL by omission of some of the illustrations. The complete article appears in the Transactions of the Section and in the author's reprints.

CANCER OF THE BREAST

In 1898 Albarran and Hailé,¹ in an oft quoted and adversely criticized treatise under the caption, "Hypertrophy and Epithelial Neoplasms of the Prostate," direct attention to the "remarkable analogy between the hypertrophied prostatic gland and the mammary gland affected with benign tumors, or with those epithelial and interstitial lesions, which are still wrongly classified among the benign neoplasms and the chronic inflammations."

In 1907, Ewing,² discussing "Cancer Problems," stated that "inflammatory hyperplasia passes by insensible gradations into neoplastic growths."

During the past five years, MacCarty³ has contributed several papers to the subject of cancer of the breast. This author was stimulated to undertake an extensive investigation of the epithelial growths of the mammary gland because of inability to harmonize his daily observations with the prevalent views of the pathogenesis of these tumors and the apparently redundant, diverse and unintelligible classification of the pathologic processes.

Fig. 1.—Breast; acinus showing primary epithelial hyperplasia (MacCarty).

At the beginning of this work the hypothesis was formulated, that the cells of cancer are the direct offspring of cells normally present in the acinus of the breast, and that they are derived neither from the differentiated cells through metaplasia, nor from prenatal or postnatal rests, according to the views attributed to Cohnheim and Ribbert. The investigation has been pursued on the foundation constructed by authoritative embryologists; personal speculation has been scrupulously avoided and notes of conditions have been confined strictly to the morphologic evidence presented by epithelial tissue under the microscope.

At the present period of this long investigation, MacCarty states that his earlier observations have been confirmed, and that his view of the origin of cancerous cells in the breast is no longer a hypothesis, but a demonstrable fact.

With the purpose of endeavoring to determine an analogy between the pathogenesis of cancer of the mammary gland, as advanced by MacCarty, and the pathogenesis of cancer of the prostate, I undertook the examination of this series of prostatic specimens.

A brief summary of MacCarty's investigation of epithelial growths of the breast follows:

Fig. 2.—Breast; acinus showing secondary epithelial hyperplasia (absence of inner row of cells and hyperplasia of outer row of cells in lumen [MacCarty]).

The normal acinus of the mammary gland is lined with a single row of cylindrical epithelial cells, which are the differentiated, functional cells of the organ. These differentiated cells arise from the stratum ger-

Fig. 3.—Breast; acini showing secondary hyperplasia and confusion of line of demarcation between acinus and stroma (MacCarty).

minativum, which is usually undiscernible until some stimulus causing hyperplasia is present. Then the germinative layer appears in the acinus as a second row of cells which adjoins immediately the surrounding stroma. For descriptive purposes these cells are termed the outer row and are described as the undifferentiated or partially differentiated precursors of the

1. Albarran and Hailé: Ann. des mal. des org. génito-urin., 1898, p. 797.

2. Ewing: Harvey Lectures, Nov. 16, 1907.

3. MacCarty: Tr. of Surg. and Gynec. Assn., 1910; Surg., Gynec. and Obst., October, 1913, p. 441; Surg., Gynec. and Obst., March, 1914, p. 284.

specific functional cells. The morphologic appearance of these cells of the outer row differs from that of the differentiated cells, which now form and are termed the inner row. In the presence of an excessive or abnormal stimulus the cells of the outer row become

Fig. 5.—Breast; acinus showing secondary epithelial hyperplasia with irregularity of nuclei (MacCarty).

hyperplastic and partially or completely fill the acinus. In this event, the functional cells are present, exfoliated or absent. Furthermore, the undifferentiated cells are observed not only intra-acinic, but also extra-acinic, situated in the stroma. Therefore, some microscopic fields present both rows of cells, differentiated and undifferentiated, distinct in the acinus; other fields

Fig. 6.—Breast; tertiary epithelial hyperplasia, showing irregularities of nuclei—cancer (MacCarty).

show a definite predominance of intra-acinic, hyperplastic undifferentiated cells; and in still others, both intra-acinic and extra-acinic hyperplastic undifferentiated cells are seen. In some fields in which the inner row of functional cells are exfoliated or absent, and

the undifferentiated cells of the outer row hyperplastic, the line of demarcation between the acinus and the stroma is observed obliterated, totally or in part, and the latter cells are noted also in the stroma. The facts that epithelial invasion of stroma is the accepted histologic picture of cancer; that in these observations the epithelial cells in the stroma are morphologically indistinguishable from the hyperplastic undifferentiated cells of the acinic outer row; and finally, that the latter cells represent the normal precursors of the functional cells in the stratum germinativum, seem to lead logically to the deduction that *the cancerous cells of the mammary gland are the direct, immediate descendants of the undifferentiated cells of the germinative layer*, consequently, that the hypothesis of "embryonic rests" is not essential for the explanation of cancer in this organ.

For the sake of simplicity in studying the mammary epithelial tumors, the following classification is based on the observations cited above: The pathologic processes involved in these epithelial growths are designated, (1) primary hyperplasia; (2) secondary hyperplasia; (3) tertiary or migratory hyperplasia. In pri-

Fig. 7.—Normal prostatic epithelium.

mary hyperplasia, both rows of cells, the inner, differentiated, and the outer, undifferentiated, are present and concerned in the reaction; in secondary hyperplasia, the outer row, undifferentiated cells, are excessively proliferated (cancer?); in tertiary hyperplasia, the hyperplastic undifferentiated cells of the outer row are also present in the stroma (cancer).

The probability is advanced that, viewed in the light of these studies, the old criterion for determining cancer histologically, namely, the permeation of the "basement membrane," may be erroneous, consequently, that we may be compelled to view the condition of secondary hyperplasia as possibly malignant before invasion of tissue has occurred.

CANCER OF THE PROSTATE

In examining the prostatic specimens of this series for the purpose of seeking an analogy between the cancerous process in this gland and that noted by MacCarty in the breast, the following observations have been made:

The normal acinus of the prostate is lined with a single or an imperfect double layer of columnar epi-

thelial cells. These are the differentiated or specific functional cells of the gland. In the examination of the pathologic specimens, these functional cells are seen frequently in a state of excessive proliferation, forming projections in and bridges across the acini. There is observed also partial or complete exfoliation of the same cells with the formation of cysts, which are empty or contain accumulations of the cells in varying degrees of degeneration. Some fields are noted in which the acini present not only the proliferated functional cells, but also, outside these and immediately adjoining the stroma, another row of cells, which are morphologically dissimilar to the inner row of differentiated cells. Other fields contain acini with functional cells present, exfoliated or absent, and the lumina partially or completely filled with the hyperplastic undifferentiated cells of the outer row. Still other fields are seen in which these hyperplastic cells of the outer row are both intra-acinic and extra-acinic, consequently presenting epithelial invasion of tissue, the accepted picture of cancer. Furthermore, what is impressive, the three conditions just described have been observed together in the same microscopic field.

Fig. 8.—Normal prostatic epithelium.

An analysis of this comparative study of the prostate and the mammary gland indicates that an analogy exists between the microscopic pictures of epithelial changes in the cancerous process of both organs. The analogy being accepted, it follows that this study of the prostate adds further support to MacCarty's deduction, drawn from extensive observations on epithelial growths of the breast. If adequately prolonged and comprehensive investigations in the future positively confirm the present view of the latter author, it may be stated of the prostate that, whatsoever the associated pathologic condition or wheresoever the original focus, these are factors which are unessential to the process of cancerous development; that this is essentially a process of epithelial hyperplasia, involving the above-described undifferentiated cells, in a state of reaction to an abnormal irritant. Furthermore, for the pathogenesis of cancer it would not be necessary to assume the theory of "embryonic rests."

Frequency.—Statistics indicate that we are indebted to the investigators of the past half century for the existing knowledge of cancer of the prostate. The

first useful statistics were compiled by Thompson⁴ (1854) who cited 18 cases from the English and the French literature. Wyss⁵ (1866) reported 28 cases, including Thompson's; Socin⁶ (1875), 50 cases; Kapuste⁷ (1885), 54 cases; Engelbach⁸ (1888), 144 cases; Wolff⁵ (1899), 110 cases. Since 1902, numer-

Fig. 10.—Normal prostatic epithelium, proliferating and projecting in masses in the lumen of the acinus.

ous reports of malignant prostatic conditions have been contributed. It is the statistics of the more recent writers, however, which, although varying within somewhat wide limits, afford us the nearest approximation to the truth with regard to the frequency with which cancer occurs in the prostate.

In 500 cases of prostatic obstruction, Young⁶ found cancer in 20 per cent. The same author states that about one out of every seven enlarged prostates after 50 years of age is cancerous. Albarran and Hallé noted 14 per cent.; Walker,⁷ 16.5 per cent.; Wilson and McGrath⁸ (1911), 468 reexamined prostatic specimens

Fig. 11.—Normal prostatic epithelium, proliferating and projecting in masses in the lumen of the acinus.

removed at operation, 15.5 per cent.; Freyer⁹ (1914), 1,276 cases of enlarged prostate, 13.3 per cent. In the

4. Cited by von Frisch, *Der Krankheiten der Prostata*, Ed. 2, 1910.
5. Wolff: *Deutsch. Ztschr. f. Chir.*, July, 1899, lii, parts 3 and 4.
6. Young, Hugh, H.: *Ann. Surg.*, 1909, 1, 1232; *The Early Diagnosis and Radical Cure of Carcinoma of the Prostate*, *THE JOURNAL A. M. A.*, March 10, 1906, p. 699.
7. Walker: *Lancet*, London, April 11, 1908, p. 1054.
8. Wilson and McGrath: *Surg., Gynec. and Obst.*, December, 1911, p. 647.
9. Freyer: *Arch. Internat. de chir.*, 1914, vi, No. 4.

present series of 716 specimens (including the 468 cases reported by Wilson and McGrath in 1911), 12.28 per cent.† are cancer.

Age.—One patient was between the ages of 40 and 50. Seventy-eight per cent. were between 60 and 80. Other investigators have found about 68 per cent.

Fig. 12.—Cystic acini, showing two rows of cells, inner and outer row, similar to pictures seen in breast in primary hyperplasia.

between the ages of 50 and 70. Wolff reports six patients under 40; one, 29 years of age.

Mode of Involvement.—Cancer of the prostate is primary in the great majority of cases. The gland is involved secondarily by adjacent cancer or through metastasis by way of the blood-stream. Rectal ulceration is the most frequent source of secondary prostatic cancer from contiguous structures. About one-half the cases of secondary involvement are metastatic from remote organs.

Site of Origin.—The most commonly ascribed site of the original focus in primary cancer of the prostate is the posterior lobe; but its presence in the lateral and middle lobes has been noted by some authors.

Fig. 13.—Cystic acini, showing two rows of cells, inner and outer row, similar to pictures seen in breast in primary hyperplasia.

Growth.—The cancerous process has been observed to involve the entire prostate symmetrically and rapidly, but the consensus of investigators' opinions is that, in by far the large proportion of cases, cancer of this gland is limited, occurring as one or several

little nodules and of slow growth. Consequently, gross serial sections of the specimen and microscopic examination of every doubtful area are essential for the diagnosis in the great majority of cases. Failure to appreciate this point, also neglect to examine the posterior lobe, are the probable cause of statistics with low percentage of cancer, likewise of inadequate treatment and erroneous prognosis.

Extension.—In the extension of prostatic cancer the fascia of Desnonvillers, also the fibrous capsules, both of the prostate itself, and of hypertrophied spheroids, lobules or lobes, are accredited with an essentially important rôle. Young emphasizes this point. Cuneo and Veau¹⁰ have shown that the fascia of Desnonvillers is a prolongation downward of the peritoneum in fetal life, which projecting in a plane between the prostate and the seminal vesicles in front, and the rectum behind, by virtue of its reduplication doubly inhibits the spread of cancerous cells posteriorly. Consequently, ulceration of the rectum is infrequently seen and periprostatic structures are quite effectively guarded. The tendency, then, of the cancerous process is to extend upward to the region of the ejacula-

Fig. 15.—Normal epithelium in acinus, acinus with exfoliation of differentiated cells, acinus with some exfoliated differentiated cells and invasion (?) of stroma by hyperplastic cells of the outer row.

tory ducts, between the fascia of Desnonvillers posteriorly and the trigone anteriorly. Young maintains that the mucosa and submucosa of both urethra and bladder are very resistant to the malignant invasion. On the other hand, von Frisch¹¹ notes extensive involvement of both these structures. The seminal vesicles are frequently involved, and the growth may extend along the ureters, causing complete obstruction, even rupture. Extension upward for a long distance within the vasa deferentia, without apparent involvement of the outer walls, may occur (Young). Cases noted by von Frisch were: Growth into rectum with subsequent extensive involvement of the sacrum. Growth through the obturator foramen and between the adductors of the thigh. Once the capsule is permeated, the cancerous process spreads extensively.

Metastasis.—This occurs in bones, lymphatic glands and internal organs. Important to be emphasized is the fact that, whereas the primary cancer of the pros-

† In the last 248 cases of this series only the specimens already diagnosed cancer were studied. It is probable that reexamination of the remaining specimens will increase the percentage of malignancy.

10. Cuneo and Veau, cited by Bryan: *Lancet-Clinic*, April 20, 1912, p. 433.

11. Von Frisch: *Die Krankheiten der Prostata*, Ed. 2, 1910.

tate is usually small, metastases from it are commonly extensive. The most frequent sites are the skeletal bones and the lymphatic glands. Visceral metastasis is comparatively infrequent, probably occurring in about 25 per cent. Kaufmann¹² remarks the infrequency of glandular metastasis from prostatic cancer compared with that from cancer of other structures. In 100 collected cases this author found involvement of the pelvic glands 27 times; iliac, 24; inguinal, 16. Young, whose figures agree with those of Kaufmann as to the involvement of the pelvic glands, says: "It shows the fact that one should not expect enlarged glands before making a diagnosis of carcinoma of the prostate." Blumer¹³ states that cancer of the prostate gives rise to metastasis in bones in a much larger proportion of cases than any other form of cancer—probably in two-thirds of patients in whom it is allowed to run an unobstructed course. Von Recklinghausen¹⁴ emphasizes the fact that the cancerous focus in the prostate is, in comparison with the advanced foci in the bone, small and insignificant. The author directs attention to the analogy between metastasis in bone from a small nodule of cancer in the female

tate and those of the mammary gland, as presented by MacCarty.

2. In the undoubted histologic picture of cancer, both organs present in the stroma migratory epithelial cells which are morphologically indistinguishable from the hyperplastic undifferentiated cells within the acinus.

Fig. 16.—High power of acinus in Figure 15, showing both exfoliated differentiated cells and hyperplastic cells of the outer row.

breast and that from cancer of the prostate, and gives warning to suspect these sources in case of severe pain in the spinal column without evident cause. Blumer advises similarly in case of spastic paraplegia—involvement of the spine—and apparent osseous tumors in flat and long bones. In the thyroid, likewise, certain forms of malignant tumors occur which present entirely analogous relations, and such growths may be so small as to escape attention (von Eiselsberg¹⁵). The metastasis in bone is more or less general, its transmission is vascular rather than lymphatic and nearly always originates in the medulla—rarely beneath the periosteum (Blumer).

SUMMARY

1. An analogy exists between the microscopic fields of epithelial changes observed in cancer of the pros-

Fig. 17.—Acinus in Figure 15, in which there are exfoliation of differentiated cells, hyperplastic cells of the outer row and immediate peri-acinic invasion of stroma by hyperplastic cells of the outer row.

3. According to the studies of MacCarty, these intra-acinic, hyperplastic, undifferentiated cells represent the precursors of the specific functional cells of the gland; consequently, the positively cancerous cells are the direct, immediate descendants of those which become the differentiated cells under normal conditions.

4. This analogy between the prostate and the mammary gland being accepted, further support is

Fig. 18.—Acinus showing the two rows of cells, acinus showing the hyperplastic cells of outer row, completely filling the lumen.

added to MacCarty's deductions, drawn from extensive studies of the breast. And, if adequately prolonged and comprehensive investigations in the future confirm the present view of the latter author, then it may be stated of prostatic cancer, as well as of cancer in general, that the process is fundamentally one of

12. Kaufmann: *Lehrbuch der speciellen pathologischen Anatomie*, Berlin, 1901.

13. Blumer: *Johns Hopkins Hosp. Bull.*, July, 1909, p. 200.

14. Von Recklinghausen: *Festschr. zu Virchow 71. Geburtstag*, Berlin, 1891.

15. Von Eiselsberg: *Arch. f. klin. Chir. (Langenbeck)*, xlvii, 1893.

epithelial hyperplasia of the above-described undifferentiated cells, in a state of reaction to an abnormal irritant; furthermore, that for the pathogenesis of cancer it would not be necessary to assume the theory of "prenatal or postnatal rests."

5. Cancer of the prostate is a comparatively frequent disease after 50 years of age.

6. The process usually begins in the posterior lobe and occurs more commonly as a small nodule or

Fig. 20.—Same as Figure 19, showing in upper acinus primary and secondary hyperplasia; in lower acinus, secondary hyperplasia.

nodules of slow growth; consequently, *careful investigation of the posterior lobe, gross serial sections of the specimen, with microscopic examination of doubtful areas, are essential for diagnosis in the majority of cases.*

Fig. 21.—Same slide as Figures 18, 19 and 20, showing hyperplastic epithelial cells in the stroma, probably a lymphatic space (tertiary epithelial hyperplasia, or cancer). The cells are morphologically indistinguishable from the intra-acinar hyperplastic cells of the outer row (secondary hyperplasia).

7. The fascial strata associated with the prostate seem to be quite effective barriers to the progress of the disease.

8. The tendency of extension of the cancerous process is upward to the region defined by the fascia of Desnonvillers posteriorly and the trigone anteriorly, and then beyond this area.

9. Metastasis affects bones, lymphatic glands and internal organs. The cancerous focus in the prostate may be, and commonly is, insignificant, while the metastasis in bone is extensive. Similar relations have been observed with small cancerous nodules in the female breast and the thyroid. Manifestations in bone, with cause obscure, should direct attention to these observations.

10. Involvement of lymphatic glands is not a dependable factor in diagnosis, since metastasis may be present in the skeletal structure and these glands apparently unaffected.

A COMMON MECHANISM FOR MOST INJURIES OF THE SHOULDER REGION

SUPPORTED BY THE RESULTS OF TREATMENT, INCLUDING
FORTY-NINE OPERATIONS *

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During the past six or seven years I have been making a study of a series of traumatic conditions affecting the shoulder and arm, and have presented a special study of each condition. The views expressed have been radical because they were based on a new

Fig. 1.—Humerus at normal limit of abduction. Leverage effect begins when capsule tears, which usually occurs at the glenoid or humeral attachment. Each of the diagrams on effects of hyperabduction on skeleton of shoulder region includes, except Figure 10, the clavicle, scapula, upper half of the humerus and capsule of the shoulder-joint.

conception of the etiology. The clinical experience by which they were supported was small because the methods of treatment were new and because the "let alone treatment" has been the rule in most of these cases. Of one of these conditions, recurrent dislocations of the shoulder, I recently reported the results of operation on eighteen shoulders, the largest number previously reported by one writer being four. I have since operated on four more. My particular purpose, now, is to show that they can be traced to an essentially common cause, and that the views offered have a rational anatomic basis and are justified by the results of treatment. It is important to bear in mind that we are dealing here with conditions that have always been the subject of much discussion and conjecture.

The conception of the anatomic basis of these conditions was of gradual development. For more than ten years I had been working on the applications of anatomy to surgery, with the cadaver as the basis of the work. There is no essential difference in the signs

* Read before the Section on Surgery, General and Abdominal, at the Sixty-Fifth Annual Session of the American Medical Association, Atlantic City, N. J., June, 1914.

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