reached. After the eggs have been placed in fresh water at the normal temperature, the cleavage will proceed and may be followed easily with a hand lens or a microscope.

The first indication of cleavage is the appearance of a slight furrow at the animal pole—the position of the polar body in the echinoderm's or the centre of the black half

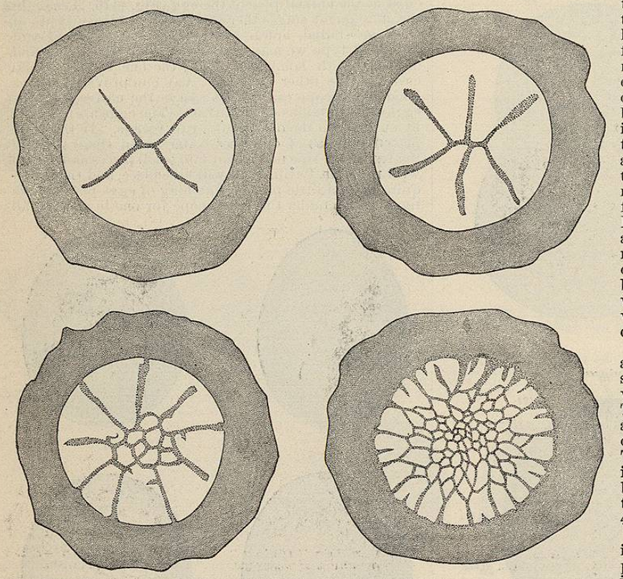

FIG. 4182.—Four Stages of the Segmentation of the Hen's Egg. Only the germinal disc, seen from above, and part of the surrounding yellow yolk are represented. (After Coste.)

in the frog's egg. This occurs in the frog's egg between two and a half and three hours after fertilization. This furrow quickly deepens and extends around the egg to the other side (Fig. 4176, *A* and *B*). Sections of an egg made just before and during this stage show that the nucleus had divided previously by a typical mitosis, and that the plane of cleavage is at right angles to the spindle, so that each blastomere contains one of the daughter nuclei. During the division the blastomeres become more or less rounded. But when it is completed they flatten against one another, so that the line of division becomes indistinct, and the egg rests for a time. At the end of the resting period, about three-quarters of an hour after the appearance of the first furrow in the frog, the blastomeres round up again and the second furrow makes its appearance at right angles to the first at the upper, animal, pole of the egg (Fig. 4176, *C–E*). This furrow extends around the egg like the first, dividing each blastomere into two. The egg is now in the four-cell stage. After another period of rest, the third cleavage furrow appears in a horizontal plane at right angles to both the first and the second. In the echinoderm egg this is very nearly at the equator of the egg, but in the frog it is somewhat above the equator, so that in the eight-cell stage we find four black micromeres and four white macromeres. Compare *F* and *G*, Fig. 4176 with *A*, Fig. 4179. The fourth cleavage in the frog occurs from one-half to three-quarters of an hour later. When this is regular each blastomere is divided into two in a plane at right angles to the preceding one. The planes of division form two great circles bisecting the angles between the first two. But in the frog the eggs are seldom so regular as this and the following cleavages are quite irregular (Fig. 4179).

The spiral form of cleavage is characteristic of the worms and molluscs (Fig. 4180). In these groups the third cleavage plane is not a continuous horizontal circle, but is tilted in each blastomere, to the left usually, looking at the egg from the side. Thus the blastomeres of the two quartets in the eight-cell stage do not lie directly one above the other, but they break joints. The lines of division in the next cleavage are tilted in the opposite direction, and are thus at right angles to the preceding ones. This alternation of spirals may continue for several generations of cells. In these forms the blastomeres are frequently unequal to a marked degree, and the rhythm of cleavage may vary in the blastomeres of different sizes, with the result that there is developed a very complex type of cleavage.

In the bilateral form of cleavage there is but one plane of symmetry, usually coinciding with the first cleavage furrow. The blastomeres are arranged in a bilaterally symmetrical pattern on the two sides of this plane. This form of cleavage is found in both holoblastic and meroblastic eggs, namely, those of tunicates and cephalopods (Fig. 4181).

The cleavage of the hen's egg is not easy to observe, for it takes place before the egg is laid, but it appears to be of an irregular radial type. As in other meroblastic eggs, the earlier cleavage furrows are incomplete, so that the blastomeres are not separated from the undivided yolk. It is only after several radial furrows have formed that concentric ones appear, dividing the blastomeres into a central group of superficially complete ones surrounded

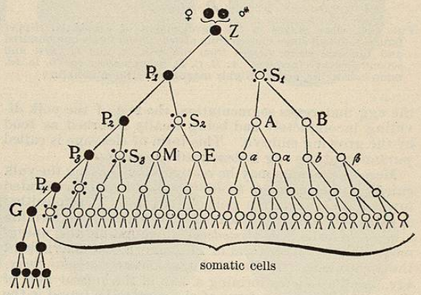

FIG. 4183.—Diagram representing the Determinate Segmentation of the Ovum of Ascaris. *Z*, Fertilized egg; P_1, P_2, etc., protogonocytes; *G*, primordial germ cell (P_3, Fig. 2614, article *Heredity*); S_1, S_2, etc., primary somatic cells; *A* and *B*, primary ectodermal cells; *a*, *b*, daughter cells of the right side; *α*, *β*, of the left side; *E*, primary endodermal cell; *M*, cell which produces mesoderm and part of ectoderm. (Modified from Boveri.)

by a circle of larger blastomeres still connected with the yolk at the surface (Fig. 4182); and it is still later when a horizontal division occurs, separating the central blastomeres from the yolk beneath.

The segmentation of the ovum differs also among the various groups of animals in being either *determinate* or *indeterminate* in character. Typically determinate types of cleavage are found in the eggs of worms (Fig. 4183) and molluscs. In these forms the cleavage is often very complex, and at first glance appears very irregular, but careful study shows that each cell division follows a law that is perfectly definite within the species. Thus the history of each cell may be traced from the first cleavage to the formation of the organs. Conklin, for example, has constructed a remarkable genealogical tree showing the history of each cell in the eggs of Crepidula, the common slipper shell, to the one-hundred-and-nine-cell stage (Fig. 4180), and from the groups of cells present at that stage he was able to observe the development of various important organs.

In the echinoderms and vertebrates, on the other hand, the cleavage soon becomes irregular and no one has succeeded, so far, in tracing the history of the blastomeres. So, for the present, the cleavage of these forms must be regarded as indeterminate. In the frog it has been found that the first cleavage furrow coincides with the principal axis of the body, but this rule is not true for all individuals. So we cannot say that even the first two blastomeres always give rise to certain parts of the body.

Robert Payne Bigelow.

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SEIGLER'S SPRINGS. — Lake County, California. Hotel.

These springs are located at the foot of Seigler Mountain, at an elevation of 2,372 feet above the sea. They are in the neighborhood of Adams and Bonanza Springs, and lie in Seigler Valley, which is about one mile and a half long by half a mile in width. The surrounding country affords many excellent drives, and magnificent views are encountered on every hand. There are twenty or more springs, which yield approximately three thousand gallons per hour. The "Arsenic" Spring has a temperature of 96° F., and is much used for syphilis, scrofula, and cutaneous disease. The "Soda" Spring is alkaline and carbonated, and forms a delicious drinking-water. It has been much in vogue for Bright's disease, bladder troubles, etc. The "Magnesia" Spring is heavily charged with Epsom salts and carbonic acid gas. A glassful before breakfast insures an easy and painless evacuation of the bowels. The Sulphur Spring is mostly used for bathing and for lung, liver, and rheumatic troubles. There are very good accommodations at the springs.

James K. Crook.

SEMILUNAR GANGLIA, PATHOLOGY OF.—A search through the literature of recent years for the results of work on the pathology of the semilunar ganglia is not very satisfactory. The facts that these organs are such near neighbors of the suprarenal capsules and the pancreas, and that they have such intimate nervous relations, especially with the former, have led to many efforts to establish their pathological association; but these attempts seem to have failed of convincing demonstration, the conclusions reached being largely theoretical. The result is that while the journals contain many articles showing extensive research and experimentation and faithful observation of cases and autopsies, the number of established facts bearing on this subject which can be found in the systematic treatises is small and disappointing.

Romberg was among the earliest to ascribe to the semilunar ganglia definite pathological manifestations. Under the title of NEURALGIA CŒLIACA he describes "a sudden and violent epigastric pain or one preceded by a sense of oppression. It generally extends to the back and there are a sense of fainting, cold extremities, and small intermittent pulse. The region of the stomach is either swelled or sunken and the abdominal parietes are tense. Pulsation at the epigastrium is common. Pressure gives relief. Sympathetic sensations occur often in the thorax, under the sternum, or in the pharyngeal branches of the vagus, but seldom in superficial parts. It lasts for from five minutes to half an hour, and is succeeded by extreme exhaustion. If it breaks off suddenly it is followed by eructations of gas or fluid, by vomiting, gentle perspiration, or copious enuresis. The suppression of accustomed hemorrhages gives rise to it, also it often precedes rheumatism and melæna. Gout predisposes to it, and the development of carcinoma ventriculi is often preceded for years by cœliac neuralgia. The peculiar sense of fainting and annihilation which accompanies it is pathognomonic of this disease, and distinguishes it from such neuralgia of the vagus as is included in the term cardialgia."

Byron Robinson also includes neuralgia cœliaca among the functional disturbances connected with the semilunar ganglia. After remarking that "there may be post-mortem findings of lesions of the sympathetic, but these may not have been preceded by records of physical complaints in life, and they may be secondary," he continues:

"Hyperæsthesia or exalted irritability of the sympathetic nerves is liable to manifest pain irregularly, periodically, spasmodically, and yet retain some irritability during the intervals. Anatomically we know little of the characteristic changes in structure in hyperæsthesia. Its etiology also is obscure, although malnutrition is probably a bottom fact. The active hyperæsthesia of the great ganglia of the sympathetic system is characterized by an overpowering sense of prostration, a sense of impending dissolution, as if the centre of life would be destroyed."

These views are quite in line with those of Romberg, and correspond closely with those given by F. A. Hoffmann in discussing the diagnostic significance of subdiaphragmatic pain. He considers radiating pain as characteristic of irritations of the retroperitoneal structures as distinguished from those originating in affections involving the mucous membranes or the parenchyma of organs, the muscular tissue of hollow organs, or the peritoneum. Moreover, he finds that the tendency of pain originating in the aorta, the adrenals, and the solar plexus is to extend downward. He finds that little attention has been paid to a neurosis of this plexus, although it is one comprising very numerous sympathetic filaments, and the neuroses of which must exhibit sensory, motor and vasomotor lesions, suggesting the analogue of migraine.

The characteristic site of the pains which belong to a neurosis of the cœliac plexus is the upper part of the abdomen, and from this point they radiate to the sacral and gluteal regions behind, but not to the genital organs or legs in front. These, with sheeps'-dung fæces and polyuria form a group of symptoms pointing, in the absence of hysteria and tabes, to a diagnosis of a neurosis of the cœliac plexus. He cites three cases in support of his views, two of his own, in which the symptoms mentioned were present, and I would call special attention here to the excessive discharge of urine of low or moderate specific gravity (1.008–1.012) containing neither albumin nor sugar and unaccompanied by great thirst, as one of the pathological features ascribed to the semilunar ganglia.

With reference to the pathological relations said to exist between the semilunar ganglia and the kidneys, as shown in the occurrence of *diabetes insipidus*, the following quotations voice the prevailing opinions: Shapiro says, "that physiologists ascribe an especially weighty influence to the splanchnic nerve in regulating the quantity of urine secreted by the kidneys." He gives the history of a case of diabetes insipidus, the patient having

also pulmonary tuberculous disease and becoming greatly emaciated. "The autopsy showed no interstitial inflammation, nor amyloid degeneration of the kidneys, nor pathological changes in the brain or spinal cord, by which the polyuria could be explained, but the microscope revealed noticeable changes in the peripheral nervous elements of the kidneys. The cœliac plexus seemed to be surrounded by a bundle of small lymph glands. The most interesting changes were found on microscopic examination of the cœliac ganglion. There seemed to be thick bundles of connective tissue crossed with many granules. On section, there were seen many enlarged and developed vessels in the neighborhood of which the ganglion cells were distinctly diminished, shrunken, and separated from the capsule. All the cells showed a marked mass of granular pigment. There was also pigment accumulation in the interstitial connective tissue; also fatty degeneration of the axis cylinder of the splanchnic nerve." In this connection it is well to recall the anatomical relations of these nerves and ganglia, viz., that the upper part of each semilunar ganglion is joined by the greater and lesser splanchnic nerves, filaments from them going to the renal plexus and suprarenal gland; also to the inner side of each semilunar ganglion the branches of the solar plexus are connected. The cœliac plexus is a direct continuation of the solar plexus (Gray's "Anatomy"). Futcher quotes from Ralfe as follows: "The sympathetic plays the most important rôle among the peripheral nerves in the etiology of diabetes insipidus. . . . The nerves forming the renal plexus are derived chiefly from the solar plexus. As the right vagus and greater and lesser splanchnics join the solar plexus, it is probable that branches of these nerves enter the kidney by way of the renal plexus. Dickinson reported a case of diabetes insipidus in a patient with *carcinoma of the liver and involvement of the solar plexus;* carcinomatous metastases were found in the retroperitoneal glands which involved branches of the solar plexus. There was marked hyperæmia of the kidneys. . . . Ralfe also refers to tumors pressing on the thoracic and abdominal nerve ganglia, which probably agree in disturbing the vaso-motor governance of the renal vessels."

Pigmentation of the skin, notably that observed as a feature of Addison's disease, has also been ascribed pathologically to the semilunar ganglia. Byron Robinson includes it in his list of the functional disturbances of these organs. Thompson says: "The fact has been confirmed by many observers (Lancereau, Nothnagel, Fleiner, and others) that the pigmentation occurs most prominently in those cases [of Addison's disease] in which the sympathetic nerves are found diseased." Byrom Bramwell quotes two French observers, Alezais and Arnaud, as concluding that the essential cause of the pigmentation and other characteristic symptoms of Addison's disease is implication of the pericapsular nervous ganglia, which they describe, by a tuberculous process extending from the adrenals. Marchand, on the other hand, reports *in extenso* a case of disease involving the sympathetic nerves, the suprarenal capsules, and the peripheral nerves, without any bronzing of the skin. Fleiner also reports cases of bronzing of the skin in which there was found no disease of the adrenals, although the cases were ranked as Addison's disease. In fact, until very lately among German writers the pigmentation seems to have been generally regarded as a regular feature of the disease, and the name "bronze Haut" used for it. T. H. Green, however, says that "the cause of the pigmentation in Addison's disease is not satisfactorily explained. Irritation of the abdominal sympathetic is believed to cause increased pigmentation, and the pigmentation in Addison's disease is merely an exaggeration of the normal."

The trend of opinion most recently, however, appears to be that the pigmentation does not belong to Addison's disease as an essential feature, though it may often be noted in connection with it, very probably because of irritation of the sympathetic plexuses and ganglia in the vicinity of the adrenals, which are so often involved in the morbid processes associated with that disease. Neusser says that the formation of pigment in man is controlled by the vaso-motor nerves; in other words, by the sympathetic system acting through the medium of chromatophore cells. Every case of "bronzed skin" does not justify the diagnosis of Addison's disease. Pigmentation is due to a disturbance of innervation in the sympathetic tract.

The pathology of *Addison's disease* has been held to be closely related to the semilunar ganglia since the earliest commentaries were written on the group of symptoms to which that name was given. Thomas Addison's original essay on "The Constitutional and Local Effects of Disease of the Suprarenal Capsules" speaks of an abnormal condition of the semilunar ganglia in but one case, in which they were the seat of fatty degeneration. The writer of the introduction to this essay in the New Sydenham Society's edition remarks that Addison merely noted the correlation as cause and effect of the post-mortem findings of diseased capsules with the group of symptoms he had observed during life, for which he had been able to find no satisfactory explanation. He adds that "true Addison's disease has essential peculiarities of its own, and those not belonging to tuberculous or cancerous capsules." Rolleston quotes Habershon as the first to show that as a result of inflammation spreading from the suprarenal bodies the semilunar ganglia and their branches may become surrounded by dense fibrous tissue. Subsequently to this, and based to a greater or less extent upon the fact noted by Habershon, arose many theories of the pathological relations supposed to exist between the adrenals and the semilunar ganglia, and to account for the various symptoms grouped under the name of Addison's disease. It will be necessary to refer to the more prominent of these, and to some of the cases cited and the arguments adduced for and against them. One of the chief theories was called the "nervous," and Eulenburg and Guttmann say that it "regards Addison's disease as *depending on an affection of the nervous system, especially of the great abdominal plexuses of the sympathetic.* . . . The ganglion semilunare sends a considerable number of twigs to the suprarenal bodies and these form a close network, which is, as Virchow discovered, richly supplied with ganglia. . . . These observations tend to strengthen the theory that Addison's disease is intimately connected with structural changes in the sympathetic. This is not supported, however, by any very good *physiological* reason. . . . Still, *pathological* anatomy has furnished some support for this theory. . . . The results of the examination of the sympathetic still remain, however, antagonistic to each other, at one time negative, at another positive. But even should the positive evidences accumulate in the future, or if it be shown that the changes in the plexuses of the sympathetic are primary and those of the suprarenal capsules secondary phenomena, the question would still be *how* the symptoms of Addison's disease are caused by such changes, a question toward the solution of which we have not advanced one step." This question had to wait some twenty years for an answer.

I quote from Fleiner some account of the pathological findings in two cases of Addison's disease to show in a measure on what this discussion was based. He says: "The facts have been observed that well-marked cases of 'bronzed skin' have post mortem presented no pathological conditions in the adrenals, and, on the other hand, in spite of pathological findings in the adrenals after death the patients presented no symptom of the disease during life."

Fleiner reports two cases of Addison's disease, one slightly and the other much advanced. In the first case on post-mortem examination, tuberculosis of both adrenals was found and noticeable *enlargement* of the semilunar ganglia. In the second case, there was angiosarcoma of the left adrenal, which constituted a metastasis of an extraperitoneal tumor. Here the semilunar ganglia were *diminished* in size. In both cases microscopical examination showed inflammation as well as degeneration of the medullary nerve fibres, not only in the semilunar ganglia

but in the whole upper regions of the sympathetic as well as also in the intervertebral ganglia. He also speaks of the degenerative changes in the splanchnic, the cervical ganglia and portions of the posterior columns of the cord, at the entrance points of the posterior roots and in the peripheral nerves. He then expresses the opinion that the principal groups in the symptom complex known as Addison's disease—viz.: (1) The lesions on the part of the digestive organs; (2) the manifestations on the part of the nervous system; (3) the pigmentation—can be satisfactorily explained by the facts he has brought forward in discussing these cases. As a summary he concludes "that for the reasons he has given he feels warranted in regarding as characteristic of Addison's disease a condition of chronic inflammation, which, advancing from the degenerated adrenals, exhibits its highest degree in the semilunar ganglia of the sympathetic and in the intervertebral ganglia, and is appreciable in slighter degrees in the ganglia of the pectoral sympathetic and in the cervical ganglia, and which is emphasized in connection with the alterations in the connective tissue, especially in an intense atrophy of the ganglion cells and in an extensive degeneration of the medullary nerve filaments in the sympathetic and in the splanchnics."

As opposed to these views we find C. Alexander quoting Kahlden to the effect that the assumption that certain symptoms of Addison's disease are referable to the semilunar ganglia is false, that the cœliac ganglion has nothing to do with Addison's disease. He also calls attention to the fact that in various other diseases the semilunar ganglia are involved in pathological changes, as shown by Hale White. That author made microscopical examination of the cervical sympathetic and semilunar ganglia in thirty-three patients dying from various diseases, such as diabetes, cancer of the bladder, aortic disease, sarcoma of the pelvis and breast, chronic Bright's disease, phthisis, tumor of the brain, diphtheria, anthrax, myxœdema, cerebral hemorrhage, etc. "Of the thirty-three semilunars three came from children, and in all of them the ganglion cells were excellent examples of normal nerve cells, some of them showing processes as distinct as those of the cells of the spinal cord. In six only of the remaining thirty were all the nerve cells normal; all the other ganglia showed more or less degeneration of their cells, which in many sections were reduced to minute masses of non-nucleated granular pigment, free in the middle of the capsule. Often there was a large amount of fibrous tissue. In a few instances the section was crowded with leucocytes, but no cause for this could be made out. . . . We may probably conclude that although the semilunar ganglia in the lower mammals and in young human beings are functionally active, in human adults their nerve cells have degenerated and become functionally inactive, but the nerve fibres always retain their structure and function."

So much of the discussion we are tracing out turned upon the question whether the pathological changes in the semilunar ganglia were primary or secondary to those in the suprarenal capsules that I quote the views of Long Fox as follows. He says: "The sympathetic often gives in its coarser lesions evidence that the influence is not that of the sympathetic on disease, but of disease on the sympathetic and its ganglia, and cites such instances as inflammation of the semilunar ganglia associated with headache, increase of their size in case of a tuberculous suprarenal capsule or of cancer of the stomach. Such secondary lesions may in their turn excite certain reflex phenomena, such as flushing, sweating, faintness, palpitation, diuresis. Also the sympathetic, in its character as a connecting link for function between all organs, and subject to various influences, perhaps emotional, perhaps due to altered conditions of the blood, may modify the functional activity of a ganglion or series of them, leaving them even microscopically unchanged. These effects may be motor, sensory, or vaso-motor manifestations."

Ziegler also supports the statements of Fleiner and others as follows: "Inflammation of the *sympathetic ganglia and fibres* induces changes in these structures similar to those produced in the spinal nerves. Thus tuberculous caseation of the suprarenal capsules extending to the surrounding tissue sometimes leads to inflammation and proliferation in the solar plexus and semilunar ganglia, resulting in degeneration of the fibres and ganglion cells of the sympathetic. So, too, tuberculous disease of the bones of the vertebral column is apt to extend to the sympathetic nerves and ganglia."

Bramwell distinctly favors the view that the changes in the semilunar ganglia are secondary. He says: "The nerves which pass in such abundance between the capsules and the semilunar ganglia themselves are in a considerable proportion of cases implicated in these inflammatory changes. On naked-eye examination they may be seen to be enlarged, thickened, indurated, and sometimes injected and redder than normal. On microscopic examination appearances clearly indicative of inflammatory induration (increase of connective tissue, infiltration with leucocytes, enlargement and engorgement of the blood-vessels) may be present in the fibrous covering of the nerves; in some cases the proper nervous elements (nerve tubes and ganglion cells) are also inflamed, degenerated, or atrophied. These inflammatory changes in the nerve tubes and semilunar ganglia are probably secondary. Sometimes no pathological alterations have been found in the semilunar ganglia or large nerve trunks forming the solar plexus, even when the adrenals have been completely destroyed, and in other cases the solar plexus has appeared quite normal when the adrenals have been completely atrophied, absent, or replaced by fat."

Thompson's study of Addison's disease leads him to the conclusion that the group of symptoms characteristic of the disease, while all pointing to a common origin in a lesion which excites or irritates the sympathetic nervous system, yet are not necessarily referred to the semilunar ganglion alone, but at times to the stomachic, hepatic, or mesenteric plexuses, including irritation of the diseased suprarenal capsules. In a small proportion of cases the adrenals may even remain normal, while the sympathetic nerves and ganglia are alone diseased.

When opinion had reached about the stage represented by authors as quoted above, certain new conceptions and explanations of facts previously observed were brought forward, notably by Rolleston. Attention had been called by Jaboulay to the occurrence of accessory suprarenal glands sometimes to be found upon the semilunar ganglion and in the midst of the solar plexus, and it had been suggested that their presence might explain the absence of the symptoms of Addison's disease in some cases in which the capsules themselves had been found seriously degenerated. Rolleston says "they are often so small as to be found only when carefully looked for, about the size of a grain of corn attached to the main organ by vascular tags and perhaps due to compensatory hypertrophy when the main organ is in a state of caseous degeneration." He quotes Wilks and Greenhow as believing that the *lesion* is primary in the suprarenal capsules and always of the same nature, while the *symptoms* of the disease are due to the secondary effect on the adjacent sympathetic, the solar plexus, and semilunar ganglia. As to the question of the "nervous theory" he concludes that "it is untenable; it does not explain the numerous cases recorded of typical Addison's disease, in which special attention has been paid to the condition of the semilunar ganglia and adjacent sympathetic, and in which they have been found to be normal, since a continued irritation could not last for any time without setting up inflammatory changes *in situ*."

The following quotation from Rolleston indicates the introduction into the discussion of the relations of the semilunar ganglia with Addison's disease, of a new element. It originates in the greater attention paid during recent years to the study of the ductless glands and the results which follow when their secretions are prevented from fulfilling their exact offices in the economy. Rolleston says: "Obstruction to the efferent vessels of the suprarenal capsules is quite a possible cause of Addison's

disease." At this point the following of more recent date from Hektoen will show the growth of new views. He says: "Because Addison's disease occurs without any apparent changes in the adrenals, and because the adrenal changes present often involve the abdominal sympathetic, it was attributed to chronic degenerative and inflammatory changes in the semilunar ganglia and abdominal sympathetic" (Wilkes, Jaccoud, Tizzoni, Semmola). This "nervous theory" quite held the field until recently; but the changes described by some in the nerves are frequent in apparently healthy individuals (Hale White, quoted above), and extensive chronic fibrous inflammation in the vicinity of the adrenals might lead to destruction of the efferent vessels, the sequence of events being comparable to Bonnet's experimental ligature of the veins of the adrenals with fatal effects. Addison's disease may occur without any changes in these nerves."

It has been almost impossible amid all these often conflicting theories to disentangle the pathological relations of the semilunar ganglia to the adrenals from those of these latter bodies to degenerative processes in themselves or to the result of pressure upon their efferent vessels by external agencies. It seems justifiable to express the belief, however, that the semilunar ganglia can no longer be regarded as the sole or chief and efficient causes of Addison's disease. Osler, in fact, in 1896 wrote as follows: "Although the view of disturbed innervation consequent upon the involvement of the abdominal sympathetic meets the case theoretically better than any other and is at present widely held, yet there are signs of a return to the old view of Addison."

Most recently, however, we have in Neusser's article such an elaborate and important review of Addison's disease, with independent and theoretical developments also, as cannot be overlooked, although a few brief extracts must suffice. As far as they concern the special topics treated in this article the following embody some of his conclusions and the reasons for them. He quotes from Brauer the opinion that there is no constant relation between Addison's disease and changes in the sympathetic, but reasons out the relations which he regards as intimate though not quite clear between the adrenals and the cœliac ganglion in this way: "Lesions of the sympathetic system have been observed both in connection with and in the absence of disease of the suprarenal capsules. They may affect, first the sympathetic ganglia in the substance of the suprarenal capsules and the pericapsular ganglia occasionally present, then the nerve fibres running from the suprarenal bodies to the cœliac ganglion, the ganglion itself, and the solar plexus, in addition to the sympathetic tracts extending from this point even as far as the cervical ganglion of the ganglionated cord, and finally the splanchnic nerve. . . . Many of these changes are dependent upon tuberculous disease of the suprarenal bodies and the resulting cicatrization. . . . In every case the symptoms of Addison's disease result from impairment or eventually complete suppression of the functions of these capsules brought about by disease of these capsules themselves or of the nerve tracts controlling their function. This impairment of function causes most symptoms."

Although this exposition throws the light of modern physiological research upon the question that has occupied us, still it cannot be considered so complete or simple as to be wholly satisfactory, although it does clear away many of the mists of the last half-century. Neusser's article is most careful, comprehensive, and readable, and has the fullest possible bibliography.

J. Haven Emerson.

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SEMINAL INCONTINENCE. See *Sexual Organs, Male, etc.*

SEMINAL STAINS, MEDICO-LEGAL EXAMINATION OF.—This examination is often of great importance in connection with cases of alleged rape or sodomy. The substances which are usually submitted to the expert to be examined for seminal stains are articles of bedding or of underclothing of the supposed victim, but it often happens that other substances are also to be examined. Seminal fluid, after it has become dried, adheres very tenaciously to any substance with which it was in contact when fresh, so that it sometimes happens that specimens of hair or scrapings from the skin require to be submitted for expert examination. In some cases it is also necessary to examine scrapings from the mucous membrane of the supposed victim taken in some cases during life, and in other cases, where a homicide also has been committed, after death. Therefore it often happens that the stain examined is not a simple but a compound one. This stain may consist of pure dried seminal fluid, or it may be an admixture of seminal fluid with blood, pus, or cells from various mucous membranes, or of all combined.

As a general rule, a dried seminal stain upon white cloth does not cause much change in the general appearance of the cloth. It may be slightly tinged, or in rare instances it may be slightly bloody. Upon unstarched cotton or linen cloth it will generally be noticed, however, that the cloth has a stiffer feel than the same cloth in the neighborhood of the stain, and, if it be held up to the light, it will be seen that the meshes of the cloth are filled up to a greater extent than those in the cloth surrounding the stain. If seminal fluid happens to fall on a non-absorbent surface, such as starched cloth, and heavy woollen fabrics as in cases of certain articles of outer clothing, or upon the skin or hair, and dries, the stain forms a nearly white deposit upon the fabric. This white stain would be very readily perceptible upon dark clothing.

Up to within a few years no chemical test was known which could be applied to a seminal stain, but in 1897 Dr. Florence, of Lyons, proposed a new test applicable to human seminal stains, which he considered to be a positive test for human seminal fluid. According to the experience of the writer, it always does produce a positive reaction with human seminal fluid, whether dried or fresh, but it also gives the same reaction with certain other substances. It is, therefore, like the guaiacum test for blood stains, an extremely valuable preliminary test for seminal stains, because, if a negative result is obtained, we know immediately that the stain in question does not contain dried seminal fluid.

The reagent recommended by Dr. Florence is a solution of iodine in iodide of potassium, made as follows: Pure potassium iodide, 1.65 gm.; iodine (previously washed), 2.54 gm.; distilled water, 30 gm.

The test is performed in the following manner: If the stain is on a non-absorbent surface, so that it forms a layer of more or less thickness, a minute fragment can be scraped off with a penknife and transferred to a microscope slide, treated with a drop of water, and then a minute drop of the reagent brought in contact with the edge of the drop of the water containing the fragment of the dried stain. If the stain contains dried seminal fluid, there will be formed a brownish precipitate, which, when covered with a cover-glass and examined with the microscope, will be seen to consist of numerous minute brown crystals, often arranged in groups, somewhat resembling the so-called hæmin or blood crystals. If the stain is upon a piece of unstarched cotton or linen cloth, the same result is obtained if a minute fibre of the cloth is cut out, treated with water in the same manner as above described, and brought in contact with a small drop of the Florence reagent.

Dr. Florence, in his original article ("Du Sperme et des Taches de Sperme en Médecine Légale," Lyons, 1897), states that he has been unable to obtain this reaction with anything but human seminal fluid. He has not obtained it with the seminal fluid of any animal, nor with any other human secretion except seminal fluid, but the writer has obtained the same crystals by the action of this reagent upon a little extract of partly decomposed suprarenal capsule, and also with a minute quantity of lecithin treated with water. It does not give the reaction with any of the ordinary human secretions, so that this reagent is of exceptional value as a preliminary chemical test for seminal stains.

A seminal stain can, however, be detected with absolutely certainty only by the recognition of the characteristic formed elements of seminal fluid, called spermatozoa, by microscopic examination, and generally it is necessary to use the higher powers of the microscope. These are usually found associated with various cellular elements coming from the seminal passages and prostatic ducts. Spermatozoa are usually recognized by their peculiar tadpole shape, having a peculiar conical-shaped head, and a long tail several times longer than the head. The spermatozoa of different animals vary somewhat in their size, and in the proportion of the head and tail. Human spermatozoa when fresh are about $\frac{1}{400}$ to $\frac{1}{350}$ inch in length, the length of the head being about $\frac{1}{2000}$ inch. If seen upon its side, the head of a spermatozoon appears pear-shaped, the anterior one-third of the head being less dense than the posterior two-thirds, so that if a stained spermatozoon be examined it will be seen that the anterior one-third of the head is colored less deeply than the posterior two-thirds. Spermatozoa may be detected in dried seminal stains for many years after the stain was made. The writer has been able to detect them in a dried stain more than four years old. Roussin has detected them after eighteen years (*Annales d'Hygiène*, 1867, i., 152). Unfortunately, however, after spermatozoa become dried, they are very brittle, and the tail is very liable to be broken off from the head by ordinary manipulation of the cloth or by the manipulation necessary in preparing the stain for microscopic examination. It is for this reason that so few perfect spermatozoa are found in the examination of a dried seminal stain.

For the certain recognition of a seminal stain it is, in the opinion of the writer, necessary to find absolutely perfect spermatozoa with head and tail complete, since many other substances, such as certain spores, might be mistaken for the heads of spermatozoa, and many other substances, such as delicate fibrils from the cloth fibres, might be mistaken for the detached tails. Care should always be taken, therefore, not unnecessarily to handle or rub the stain suspected to be a dried seminal stain before submitting it to the expert for microscopic examination.

The recognition of the spermatozoa is comparatively easy in cases in which the stain is a scaly one upon a non-absorbent surface. In that case it is necessary only to scrape off a little of the scaly stain and transfer to a microscopic slide, treat it with a drop of water or some fluid which does not dry readily, such as dilute glycerin, or a solution of potassium acetate, and allow it to soak for several hours. It can then be gently broken up and stained, if desired, by some of the ordinary stains used in pathological work, covered with a cover-glass, and examined with the microscope. Dr. Florence recommends a concentrated aqueous solution of crocein, but the writer has had very satisfactory results by staining with methyl green or eosin. Usually, however, the spermatozoa can be recognized very satisfactorily if they have not been stained. A microscopic power of seven hundred or seven hundred and fifty diameters should be employed for the microscopic examination, and in some cases it will be found advisable to use an oil-immersion lens for the examination.

If the stain is upon unstarched cotton or linen cloth, the recognition of the spermatozoa is much more difficult, because they are much more liable to become broken by preliminary handling, and also because the spermatozoa apparently cling very tightly to the fibres of the cloth when fresh, and are very liable to become broken when the fibrils of any individual fibre are separated. In order to prepare such a stain for examination, care should be taken to select a point near the centre of the stain, because the spermatozoa are more apt to be present in the centre of the stain than near the edges. Then a few threads may be cut from this portion of the stain, so that the individual fibres do not exceed in length one-sixteenth or one-eighth of an inch. Each thread should then be treated separately upon a glass slide or cover-glass with a small drop of water for at least two hours, care being taken to prevent the evaporation of the water. It would perhaps be better if they could be soaked for from twelve to twenty-four hours without being allowed to become dry. These individual threads, after having been digested for several hours, should be very carefully separated into their individual elements or fibrils by means of very sharp-pointed needles. The preparation can then be stained or not, according to the desire of the examiner, be covered with a cover-glass, sealed with paraffin so as to prevent evaporation, and examined with the microscope.

Moist material, such as scrapings from the mucous membrane of the vagina, for instance, can be treated with a drop of water and examined immediately with the microscope with or without being stained.

Edward S. Wood.

SENEGA, U. S. P. (*Senegæ Radix* B. P.).—The dried root of *Polygala Senega* L. (fam. *Polygalaceæ*). The senega plant is a smooth, perennial herb, its habit well displayed in the accompanying illustration. Flowers small, pinkish-white, in terminal spikes; calyx irregular, of three small green, and two (lateral) large, petaloid sepals, the latter concave and enclosing the corolla; corolla consisting of three partly united petals, of which the lower is concave and ornamented with a crest of papillæ; stamens eight, diadelphous (4 + 4); ovary transversely two-celled; style single. Senega has a wide range in the United States, from Western New England and the Middle and Western States southward. It is now mostly collected in Minnesota and Manitoba. The variety *latifolia* is a larger form with broader leaves.

The plant takes its common name, "Seneca snakeroot," from the Seneca Indians, by whom it is reported to have been used as a remedy for snake-bites.

The root is mostly 7 to 15 cm. (3 to 6 in.) long and 4 to 8 mm. ($\frac{1}{6}$ to $\frac{1}{3}$ in.) thick, exclusive of the large, knotty,