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,

many-headed crown, occasionally reaching several times this size; tortuous, tapering, and bearing several similar horizontal branches, and few rootlets, and for a variable distance below the crown a more or less sharp keel, varying greatly in prominence, and usually taking a spiral direction, often nearly absent; externally yellow-brown to dark-gray-brown, longitudinally wrinkled; fracture rather tough, but short and sharp, the bark thick, yellow or brownish, of a waxy or resinous, faintly fine-radiate appearance, enclosing a yellowish-white wood, from which a larger or smaller radial segment is usually wanting; odor slight, disagreeable; taste sweetish, then acrid.

FIG. 4184.—Senega, Root and Stem. One-half natural size. (Baillon.)

Various adulterants of senega have been reported, consisting chiefly of the roots of other species of *Polygala* of the Southern United States, but any other than the genuine root is now scarcely seen in commerce.

CONSTITUENTS.—The important constituents of senega are the two saponin-like glucosides *senegin* and *polygalic acid*, from one to two per cent. of the former and about five per cent. of the latter. There are also present from six to nine per cent. of fixed oil, some glucose, resin, gum, and very variable amounts of methyl salicylate and methyl valerianate, which latter give to the drug its characteristic odor. Starch is wanting. Senegin resembles in properties and action the saponin of soap-bark, while polygalic acid resembles quillajic acid, but they are weaker, respectively, than these substances.

ACTION AND USE.—Senega possesses in medium degree the characteristic physiological properties of the saponin-containing group of drugs. In the nares it is sternutatory, in the mouth acrid and somewhat sialagogue. In small doses it is stomachic and laxative; in larger ones emetic or purgative. It is a nauseating yet stimulant expectorant, and this constitutes the basis of its principal use. It has been extensively employed in chronic bronchitis and other diseases accompanied by cough. As an ingredient of cough preparations it has probably its most extensive use, but is much less valued than formerly. As an emmenagogue and diuretic it is obsolete.

The dose of senega is about 1 gm. (gr. xv.). The official preparations are the fluid extract and from it the twenty-per-cent. syrup, which also contains one-half per cent. of ammonia water. The compound syrup of squill contains eight per cent. of fluid extract of senega.

Henry H. Rusby.

SENILITY.—(Latin *senilis*, from *senex*, an old man.) Senility is the condition of body and mind resulting from the sum total of degenerative changes characteristic of old age.

The period of old age has its beginning, from the biological point of view, at the time of cessation or decline of the reproductive function. This occurs suddenly in women at the time of the menopause, while in men there is a gradual decline from about the fortieth year. Some animals have no old age, as is the case with those insects that die from exhaustion or shock immediately after completing their reproductive functions. Other animals, notably man, have a considerable period of old age in their lives, unless they are cut short earlier by accident or disease.

During this period, which has been studied chiefly in man, the body undergoes certain degenerative changes which result in the gradual loss of function in the various organs until, if no other cause intervenes, the individual dies, as we say, of old age.

One of the most characteristic features of senility, as was pointed out by Canstatt (1839), is the fact that it does not appear in all the organs of the body at the same time, but it begins sometimes in one organ, sometimes in another, while the remaining organs of the body continue in a normal condition.

Of the various pathological conditions met with in the aged, it is not easy to determine always which should be regarded as strictly senile and which are more characteristic of the period of maturity. But it seems to be the general opinion that the most important and characteristic of the senile changes are those that occur in the walls of the blood-vessels, especially the arteries; and, according to Demange (1886), the impairment of nutrition thus brought about is responsible for all the other strictly senile conditions. In five hundred carefully made autopsies on old people Demange found in every case evidence of endarteritis. The importance of arteriosclerosis as a cause of lesions of the tissues appears to have been recognized first by Gull and Sutton in 1871. In regard to this disease Osler says: "Longevity is a vascular question, and has been well expressed in the axiom that 'a man is only as old as his arteries.' To a majority of men death comes primarily or secondarily through this portal. The onset of what may be called physiological arteriosclerosis depends, in the first place, upon the quality of arterial tissue (vital rubber) which the individual has inherited, and secondarily upon the amount of wear and tear to which he has subjected it. That the former plays the most important rôle is shown in the cases in which arteriosclerosis sets in early in life in individuals in whom none of the recognized etiological factors can be found. Entire families sometimes show this tendency to early arteriosclerosis, a tendency which cannot be explained in

any other way than that in the make-up of the machine bad material was used for the tubing."

As causes of wear and tear of the arteries Osler enumerates: (1) Chronic intoxications, as from alcohol, lead, gout, and syphilis; (2) overeating; (3) overwork of the muscles; and (4) renal disease.

It may be too much to say that senility is always associated with or caused by a diseased condition of the arteries, for, according to Schrötter, the literature contains a number of cases of persons who have lived over a hundred years and whose arteries were found to be normal. Unfortunately, these cases have never been gathered together and discussed collectively. So we know practically nothing of senility from which arteriosclerosis is absent. It is interesting to note that according to the statistics of Eppinger, quoted by Schrötter, the period of maximum frequency of this disease is for men between the ages of sixty and seventy years and for women between seventy and eighty years, thus corresponding fairly well with the period of maximum death rate for aged men and women, respectively. (See *Longevity*.)

Councilman (1891) distinguishes three forms of arteriosclerosis: (1) Nodular arteriosclerosis, (2) senile endarteritis; and (3) diffuse arteriosclerosis. While these forms grade into one another more or less, the third form is regarded by Councilman as a definite disease arising during middle life, and is of little interest in connection with the subject of this article. But the other forms are so characteristic of old age and appear to play so important a part in fixing the natural limit to man's life, that they possess a unique biological as well as medical interest. The histological picture of these conditions is described elsewhere (see article *Blood-Vessels, Pathological Anatomy of*), and we will refer here only very briefly to their causation.

The most generally accepted theory is that of Thoma, set forth in a long series of papers, the last of which appeared in 1898. According to this view, the beginning of the process is a "compensatory endarteritis." Thoma's law, as quoted by Peabody (1891), is that "every slowing of the blood current in the arteries and veins of man which is not completely and at once remedied by a proportionate contraction of the media, leads to a new growth of connective tissue in the intima, which narrows the lumen of the affected vessel and thus restores the normal swiftness of the blood current more or less completely." The first cause, according to Thoma, appears to be the slowing of the blood current, which may be due to a stoppage of the flow beyond or to a decrease of pressure. If the muscular coat is not able to contract sufficiently to restore the normal rapidity of current, the slowing of it in some way stimulates the nerve endings, Pacinian bodies, in the arterial wall, and this sets up a reflex hyperæmia of the vasa vasorum, which in turn results in proliferation of connective tissue and an accompanying proliferation of the vasa vasorum themselves into the media, which normally lacks these vessels. According to Councilman, this explanation is extremely hypothetical and without analogy in pathology.

Henri Martin and Huchard have developed an attractive theory of the etiology of arteriosclerosis, starting with a primary lesion in the vasa vasorum. But, then, the lesion in the vasa vasorum is left to be accounted for, and there is the further difficulty, as pointed out by Schrötter, that arteriosclerosis occurs in arteries that have no vasa vasorum.

Sokoloff (1892) has shown experimentally that it is not a lessening but an increase of blood pressure that causes new formation of connective tissue. And this harmonizes with most of Thoma's results, as, for example, thickening of the intima of the aorta between the ductus arteriosus Botalli and the umbilical arteries of children after birth, and the similar process in the arteries of amputated limbs, in both of which cases there is a sudden rise of blood pressure due to the stoppage of the peripheral circulation. It would simplify Thoma's law if we might make it read somewhat as follows: Whenever the intima is unduly stretched the connective tissue tends to increase, giving rise to a compensatory endarteritis. The stretching may be due to increase of blood pressure or to failure of the media under normal pressure. Such a proliferation of connective tissue under strain has its analogy in the reactions of bony tissue to stresses and strains, and might result from the direct stimulation of the cells without the establishment of a reflex arc.

We are indebted to Seidel (1890) and to Councilman (1891) for convenient reviews of the senile changes in the various organs of the body. It will suffice for the present article to give little more than a list of these phases of senility, and the reader is referred for details to the articles on the special topics indicated.

The nodular form of arteriosclerosis, which is found frequently in autopsies on old persons, is confined to the aorta and large arteries. "We find along the course of the vessel, which is otherwise smooth and of normal calibre, elevated plaques, sometimes translucent and cartilaginous in appearance, sometimes calcified or softened. The growth is entirely within the intima, and the media at the point affected is thin and degenerated" (Councilman). This condition frequently involves the orifices into lateral branches and may descend to the valves of the heart, thus giving rise to serious functional disturbances.

In typical cases of senile endarteritis the aorta and all the larger arteries are converted into almost rigid calcareous tubes with walls thinner than normal. Similar conditions may be found less commonly in the veins and even in the capillaries.

The heart is one of the organs most frequently impaired in old people. In cases of senile endarteritis it is frequently small, the condition of brown atrophy being common. On the other hand, in seven out of fourteen autopsies on such cases Councilman found a small degree of hypertrophy. The coronary arteries may become sclerotic, and this is a potent cause of disease of the heart, the walls of which, according to Seidel, may undergo changes analogous to the atheroma of the larger arteries.

All of the organs concerned in respiration are subject to senile changes. The segments of the sternum become ankylosed, the costal cartilages become ossified, the ribs change somewhat in shape, with the result that there is a loss of mobility and capacity of the chest. The chest muscles also atrophy, adhesions form in the pleuræ, and, owing to atrophy of the alveolar walls, the respiratory surface is decreased, producing senile emphysema.

The digestive tract is also subject to important alterations, especially characterized by the atrophy and degeneration of the glands. The muscular fibres of the stomach and intestine lose their tone and allow of dilatation. In connection with disturbances of the circulation, the liver frequently suffers the lesions resulting from engorgement, atheromatous changes, sclerosis of the connective tissue, and reduction in size of the gland cells.

The kidneys are subject to atrophy and sclerosis, and the renal arteries become atheromatous. The result is a diminution in the secretion, and the consequent retardation in the removal of waste products from the body adds another factor to the unfavorable environment of the component cells of the organism in old age.

In the urogenital system the cessation of the reproductive function is accompanied by loss of weight and volume of the ovaries and testicles, with atrophy of the germinal cells and increase in the connective tissue. According to Seidel, the arteries are atheromatous, but Metchnikov calls attention to the fact that degeneration of the ovarian ova and their replacement by connective tissue begins early in life and is completed before arteriosclerosis sets in. Hypertrophy of the prostate occurs in a large number of old men (according to Seidel, in sixteen to twenty-two per cent.), and atrophy of that organ is also common. The muscular coat of the bladder becomes atonous, due to fatty degeneration of the wall with passive distention. The ureters may also become distended, and their muscular coat may undergo fibrous degeneration. The connection of all these troubles with arteriosclerosis was first demonstrated, according to

Seidel, by the Guyon School in Paris (Lannois, 1885; Arthaud, 1885).

The first change in the skin is the appearance of gray hair. The subcutaneous tissue atrophies, producing wrinkles, and the atrophy extends to the cutis. These changes are accompanied by atheromatous arteries and varicose veins.

Even the apparently stable skeleton suffers extensive alterations in old age. There is in general a tendency toward calcification of the cartilages and ankylosis of the joints and sutures, while the spaces within the bones become enlarged and the compact bone becomes more spongy. But arthritis deformans does not belong in the category of senile alterations, as it appears commonly at an earlier period.

Accompanying these changes, there is more or less atrophy of the muscles, sometimes with fatty infiltration. In the central nervous system there is some loss of weight and volume, often with local areas of degeneration; while the meninges become thickened and adherent. The brain is one of the organs in which the connection between the senile changes and an atheromatous condition of the vessels is most evident.

With the changes in the brain substance there appears a decline of its functions. The reflexes become slower and less intense. The organs of special sense become impaired, and there is a decadence of the intellectual and moral attributes.

Recently Mühlmann (1901) has described a deposition of fatty pigmented granules in nerve cells. This begins in the third or fourth year of life, the granules being at first scattered and later collected in a definite place in the cell. With age the number of cells showing this phenomenon gradually increases, and the amount of the inert, pigmented, fatty material becomes larger in each cell until in old age there is but little protoplasm left, for the cells do not increase in size. Mühlmann regards this as a normal process of senile degeneration, finally ending in death.

There are three principal theories as to the biological significance of senility: (1) Senility is the result of the inherent properties of protoplasm; (2) it is not due to the inherent properties of protoplasm, but has been acquired as a normal process by the multicellular animals and plants for the good of the species; (3) it is a pathological condition resulting from the imperfect adaptation of the organism to its surroundings.

Maupas in France and Minot in this country are among the chief advocates of the first theory. They imagine the organism to receive a store of vital energy at the time of fertilization, and this energy is supposed to be dissipated gradually until, if no accident occurs, the organism dies of old age. As evidence Minot cites the results of his investigations on the growth of guinea-pigs, in which it was shown that the rate of growth diminishes from the time of birth. (See *Growth.*) But it would seem that the facts could be accounted for equally well by the familiar physiological principle that with growth the surface for the absorption of food increases in proportion to the square of the stature, while the tissues to be fed increase as the cube. Thus, other things being equal, the larger the organism the slower would be the rate of growth.

The second theory is Weismann's. According to his view, the protozoa never die of old age. But the higher organisms have been endowed with senility and natural death through natural selection, those species which are composed of the greatest proportion of young uninjured individuals being the best fitted to survive the struggle for existence with other species. This involves the idea that senility is a normal condition like growth or hunger, which is denied by the advocates of the third theory.

Metchnikov (1899) has appeared recently among the latter with a remarkable hypothesis of phagocytosis as a cause of senile degeneration. He calls attention to the fact that the loss of the power of cell multiplication is not universal in old age, as one would expect it to be on the theory of senility being a normal process. On the contrary, the connective-tissue elements show a remarkable capacity for growth. He supposes a struggle for existence to be going on continually in the body, in which the megaphagocytes are on one side and all the remaining cells on the other side. These megaphagocytes attempt to attack and devour everything they touch, whether living or dead. Healthy living cells can resist them, but when a cell is weakened by any cause, for example, bacterial poison, the phagocytes are successful, and, after destroying the weakened cells, they take their place and change into connective-tissue corpuscles. Unfortunately for this hypothesis, however, there is very little observational evidence to support it.

On the other hand, there is abundant evidence of the close relation between senility and a diseased condition of the walls of the blood-vessels, and Thoma's main thesis that this condition arises primarily as an adaptive modification to meet unfavorable conditions of life harmonizes well with the facts. The pathologists are agreed that with the beginning of arteriosclerosis a vicious circle is soon established, resulting in the progressive increase of the various troubles associated with that disease. If this group of pathological conditions does not form the sole feature of senility, it certainly forms the most conspicuous one. And in the absence of any knowledge of the unknown conditions, if there be any, we should expect, on the theory of chances, that these unknown conditions would be of the same general character as the known conditions. Spencer has defined life as "the continuous adjustment of internal relations to external relations," and we may define senility as the progressive result of imperfect adjustment of internal relations to external relations.

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SENNA, U. S. P., B. P. (*Folia Sennæ*, P. G.).—The dried leaflets of *Cassia acutifolia* Delile (*Alexandria Senna*), or of *Cassia angustifolia* Vahl (*India Senna*); fam. *Leguminosæ*.

In the United States, German, and French pharmacopœias, the general title covers both the principal varieties; in the British, the Alexandrian (*Senna Alexandrina*) and Tinnivelly (*Senna Indica*) sennas are distinguished by name; they are always entirely distinct in the market.

1. *C. acutifolia* Delile is a small shrub about a meter (a yard) high. Its pod is broad, flat, coriaceous, slightly curved, rounded, and oblique at the ends, containing about half a dozen seeds. This species has a wide and unknown range in Central Africa, is abundant in Nubia, Kordofan, Sennaar, etc., and is said to be found in Timbuctoo. The leaflets are gathered twice a year by native tribes and carried to Alexandria, where they are very carefully freed from sticks, stems, stones, and other impurities, and the broken and defective leaves separated, the different portions of leaf, even to the siftings, being marketed separately.

2. *C. angustifolia* Vahl. is also a small shrub, a good deal like the preceding, but it has larger flowers and larger, more numerously paired leaflets. Its pod is narrower and straighter than that of *C. acutifolia*, and contains about eight seeds. It is a native of Arabia, and in the wild state supplies an inferior, carelessly collected variety of senna (Arabian). It is also said to be found

in Somaliland. This is the species cultivated in the south of India (where it is not indigenous) as the source of Tinnivelly senna. The leaves, under cultivation, are increased in size and improved in quality.

Although the Alexandrian variety brings a higher price and is generally held in higher esteem, there seem no good grounds for the idea that it is essentially different in its action from the other.

DESCRIPTION.—*Alexandria senna* consists of leaflets with extremely short, stout petioles, about 2.5 cm. (1 in.) long and 1 cm. (⅜ in.) broad, inequilaterally lanceolate or lance-ovate, acutely cuspidate, entire, subcoriaceous, brittle, pale-green or slightly yellowish- or grayish-green, sparsely and obscurely hairy, more so underneath, the hairs appressed; odor peculiar, tea-like; taste mucilaginous, tea-like, bitterish.

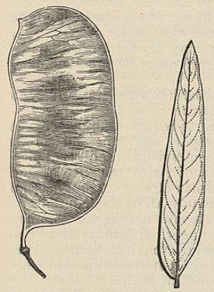

FIG. 4185.—Leaf and Pod of Cassia Angustifolia; about natural size. The leaf is Tinnivelly senna. (Baillon.)

It is frequently contaminated with the one-nerved, thick, wrinkled, glaucous, equilateral Argel leaves.

India senna leaves average nearly twice as long, though but little broader, are more abruptly pointed, usually more yellowish, and the hairiness is even more obscure.

CONSTITUENTS.—As to its active constituents, senna is closely related to rhubarb, cascara sagrada, and some other laxative and cathartic drugs. Like them, its active principles appear to be the anthraquinones emodin, isoemodin, and chrysophanic acid, all of which have been considered elsewhere, with which exist rhamnetin, a large amount of gum, a little tannin, and ordinary leaf constituents. Cathartic acid, formerly regarded as the active constituent, is apparently a mixed body, and fairly represents the properties of the drug. However, the administration of anything else than the entire drug, or a preparation of it, appears inadvisable.

ACTION AND USE.—This is one of the most satisfactory and generally useful of simple cathartics, usually emptying the bowels thoroughly in ten or twelve hours, with but little depression or other untoward effects, excepting a variable amount of griping; it acts principally upon the small intestine, and the amount of effect produced can generally be pretty accurately regulated, by the dose, from the mildest laxative to a brisk cathartic. It is in universal domestic use, and is the foundation of numerous proprietary laxatives. By combination with salines its activity is considerably augmented; in small doses it does not readily lose its efficiency. It appears to be partially excreted in the milk, when taken by nursing women.

ADMINISTRATION.—A few senna leaves chewed every day are a favorite habitual laxative with many people, who find them to act efficiently, without griping and without producing after-sluggishness of the bowels. One or two dozen leaves usually display some effect. In large doses (6 or 8 gm. [ʒ iss.] or more), as required for thorough action, it is apt to produce colic, unless modified by aromatics or salines. A strong alcoholic extract is inert; a watery extract, made from the residue after exhausting by alcohol, is active and much pleasanter than one made without this previous treatment. Infusion with hot water extracts the active principles and makes a good form for administration, but prolonged boiling destroys it, as do also mineral acids and alkalies. Bitters are said to increase its action.

The official preparations are numerous and good. The Fluid Extract (*Extractum Sennæ Fluidum*, U. S. P.), made with weak alcohol, represents the leaves weight for weight. It is not often given alone, but is eligible for mixture with other medicines. The Compound Infusion, Black Draught (*Infusum Sennæ Compositum*, U. S. P.), consists of: Senna, 6 gm.; manna, 12 gm.; sulphate of magnesium, 12 gm.; fennel, bruised, 2 gm.; boiling water, 800 c.c.

Pour the boiling water upon the senna and fennel, macerate until cold, strain with expression, dissolve in the Epsom salt and manna, again strain, and add enough cold water to make the infusion measure 1,000 c.c. The syrup has a strength of twenty-five per cent. of senna, and contains a little oil of coriander to flavor. The confection contains ten per cent. each of senna and tamarind, sixteen per cent. of cassia fistula, seven per cent. of prune, twelve per cent. of fig, and a little oil of coriander to flavor. It is a blackish extract-like mass of a sweetish taste, and is an appropriate remedy for chronic constipation, being especially useful for children, who take it readily. The compound syrup of sarsaparilla contains 1.5 per cent. of the fluid extract of senna. The compound liquorice powder has already been considered under *Liquorice*.

ALLIED PRODUCTS.—The leaves of a number of other species of the section of *Cassia* to which the official species belong, possess similar properties, though much weaker, and have been at one time or another proposed as substitutes, especially those of *C. obovata* Collad., widely diffused through the tropics of both hemispheres. No others, however, appear in commerce at the present day.

Henry H. Rusby.

SENSATION, DISORDERS OF.—The scope of the present article is the discussion in brief of those abnormalities of sense perception not described under separate headings. General sensibility is the name given to the power of perception possessed by the various body tissues other than those of special sense. It is not, however, homogeneous in kind nor equal in degree, but varies in acuteness, in localizing power, and in other ways. Sensitiveness of the skin and mucous membranes is divided into that to tactile, that to painful, and that to temperature impressions. Muscular sense is an indefinite feeling, by which the weight and size of objects are appreciated or approximated; it is probably composite, the feeling of tension in the muscles being supplemented by tactile impressions from the skin and other tissues. Visceral sensibility has a varying but comparatively slight localizing value. Its appeal is largely to the subconscious psychic stratum; when visceral sensations rise to the level of consciousness, they may consist of visceral pain, local or referred, or be felt as hunger, thirst, fright, nervousness, psychic pain, anger, and so forth. These feelings are more or less indefinite, often not referred to an organ or part of the body, although at times they may be somewhat localized,—as, for instance, hunger and depression as epigastric sensations, thirst as dryness in the mouth and throat.

The general sense of well-being on which happiness depends is a form of general sensation known to us mainly by its occasional loss, or rather by a feeling of definite depression, not usually localized, though sometimes felt as a cardiac or epigastric sinking or feeling of heaviness. An allied and in some ways opposite state is nervousness, a much abused term, but one of sufficient importance to deserve careful definition and description.

General sensibility, then, is a function of widely different tissues, much varied in kind, definiteness, and degree. The impact on consciousness due to sensory stimuli may give rise to intellectual or to emotional processes, the first being concepts relating to location, form, size, weight; the latter consisting of either pleasurable or painful feelings.

Disturbances of sensation are classed as anæsthesia,—that is, diminution or loss of sensitiveness, which has been described elsewhere in these volumes; hyperæsthesia,—that is, increased sensitiveness; paræsthesia,—that is, perverted sensitiveness; the different varieties of the latter are usually spoken of as paræsthesiæ. Variations in