A fact recently brought out by Cleghorn deserves mention here. Cleghorn found that glycerin extracts of sympathetic ganglia, when injected into the femoral or jugular vein, caused a fall in blood pressure which was not observed if glycerin extracts of spinal ganglia, or of spinal cord or brain matter, or of nerve or abdominal tissue, were injected instead. Cleghorn seems recently to connect this vascular effect of the glycerin extracts of sympathetic ganglia with the presence in the latter of large polygonal cells, discovered by Stilling, which cells stain deeply in chromic acid and are found also in the suprarenal capsules.

In concluding this subject, attention is called once more to the remote effects of lesions of the sympathetic, these being often late in their appearance and showing a tendency to progression, as was the case with the digestive and respiratory disturbances following removal of the stellate ganglion or of part of the thoracic sympathetic nerve.

Representation of the Sympathetic in the Spinal Cord and Brain.—The rôle of the rami communicantes and their homologues, in establishing a connection between the cerebro-spinal and sympathetic systems, has been amply discussed in previous paragraphs (see pages 580 and 581). It remains now to describe in detail the manner of representation of the sympathetic in the spinal cord and brain.

From a study of the degenerations following resection of portions of the gangliated cords, Dr. Collins and the writer (see the monograph above mentioned) came to the following conclusions regarding the course and spinal representation of the afferent and efferent fibres of the sympathetic nerve proper in the cat:

1. Most, or at least many, of the afferent (sensory) fibres of the sympathetic nerves do not originate from cells of the spinal ganglia, as Kölliker claims, but must have their cells of origin within the ganglia or plexuses of the sympathetic system.

2. The chief terminal station for the afferent fibres of the sympathetic is *Clarke's column*, the said fibres probably ending there around the cells of this column. Other terminal stations of such fibres are probably the lateral horn and the zone between the anterior and posterior horns which we called the intermediate zone. The fibres probably terminate around the large cells of these regions.

3. Clarke's column, besides being a terminal station for afferent fibres from the vegetative organs, may be instrumental also in conducting sensory stimuli from the muscles, tendons, joints, and bones to the cerebellum, being thus largely concerned in maintaining equilibrium.

4. The afferent fibres of the sympathetic nerve after entering the spinal cord probably send reflex collaterals to the nuclei of the efferent fibres of the sympathetic (see Fig. 4581).

5. The afferent fibres of the *lumbar* sympathetic nerve, entering the spinal cord by way of the posterior roots, after having arrived at Clarke's column, evidently describe a longitudinal course upward (cephalad) to terminate around cells of a considerably higher level (see Fig. 4579).

6. The afferent fibres coming from the ganglia of the lower half of the thoracic sympathetic take on the whole a rather horizontal course in the spinal cord, to become connected with spinal cells of the same level, but part of these fibres probably descend either in the spinal cord or in the sympathetic nerve through the distance of one or more segments before reaching the cells around which they terminate. This is illustrated diagrammatically in Fig. 4580.

7. Many of the afferent fibres derived from the stellate ganglion probably make a long descent in the spinal cord or possibly in the sympathetic nerve, becoming connected partly with the same cells with which the fibres from the lower portion of the thoracic sympathetic nerve form connections (see also Fig. 4580).

8. The efferent (motor, secretory, inhibitory, etc.) fibres of the sympathetic probably take their origin from the cells of the following cell groups of the spinal cord: (*a*) The paracentral group; (*b*) the small cells of the lateral horn; and (*c*) probably the small cells of the zone situated between the bases of the anterior and posterior horns (intermediate zone). The situation of these groups is diagrammatically illustrated in Fig. 4581. That most (or all) of these fibres do not pass uninterruptedly to the peripheral organ innervated by them, but terminate in some ganglion of the sympathetic, and that from there a new set of neurones originates, giving rise to "sympathetic" fibres which pass to the periphery, has been discussed in other paragraphs, chiefly pages 580 and 581.

9. The paracentral group has possibly a vascular function.

10. The pathway of the efferent fibres of the sympathetic is probably similar to that of the afferent fibres as outlined under 5, 6, and 7; with this difference, that most of them pass through the anterior and not the posterior roots, and that they conduct the impulse in the opposite direction to that of the afferent fibres.

11. The spinal representation of both the afferent and the efferent fibres of the sympathetic nerve is probably bilateral.

12. The homologon of Clarke's column for the oblongata is probably a large-celled nucleus accompanying the so-called solitary or respiratory bundle at its ventro-lateral border.

13. The homologon of the paracentral group for the oblongata is evidently the so-called dorsal vagus nucleus, called also vagoglossopharyngeal nucleus, situated at the floor of the fourth ventricle.

It probably has the function of supplying the non-striated muscles innervated by the pneumogastric nerve, while those nerve fibres of this nerve which supply striated muscles probably originate from the nucleus ambiguus.

The relations between: (1) the ninth, tenth, eleventh nerves, (2) solitary bundle, (3) vagoglossopharyngeal nucleus, and (4) nucleus ambiguus are illustrated in Fig. 4582.

14. Higher up in the cerebral axis the paracentral group (of the spinal cord) is possibly and even probably represented by the vesicular cells accompanying the so-called cerebral fifth root (known formerly under the name of the descending fifth root) and by the cells of the substantia ferruginea.

To the views arrived at by Gaskell by a most ingenious method we can only allude here, giving his conclusions as to the centres for the efferent fibres (including those of the sympathetic) in the form of a table:

A. (Cells of the anterior horns.) Nucleus of efferent nerves to somatic muscles.	Represented in the medulla oblongata by the hypoglossal nucleus.
B. (Large cells of lateral horns.) Nucleus of efferent nerves to striated splanchnic muscles.	Represented in the medulla oblongata by the nucleus ambiguus (motor vagus nucleus).
C. (Cells of Clarke's columns.) Nucleus of anabolic (inhibitory) nerves to splanchnic glandular system and to muscles of viscera.	Represented in the medulla oblongata by the nuclei at the floor of the fourth ventricle, known as the accessory and the dorsal vagus nuclei.
D. (Solitary cells of posterior horn.) Nucleus of motor nerves to muscles of viscera.	No mention made respecting their possible representation in the medulla oblongata.
E. (Small cells of lateral horn.) Nucleus of katabolic (motor) nerves to splanchnic glandular system and to muscles of vascular system.	

This shows that in many respects Gaskell had come to conclusions similar to those reached by Dr. Collins and the writer.

To enter on the conclusions of other workers in this field, for instance Mott, the space allotted to this subject does not allow.

B. Onuf (*Onufrowicz*).

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SYMPATHETIC NERVOUS SYSTEM, DISEASES OF. (GENERAL.)—A systematic discussion of the diseases of the sympathetic nervous system is, at the present state of our knowledge, not feasible. The anatomy, physiology, and pathology of this part of the nervous system are still quite obscure.

At the bedside symptoms referable to and explicable by disordered function of the vegetative neural mechanism are encountered quite frequently. These symptoms are mainly expressions of exaggerated, diminished, or perverted trophic, circulatory, secretory, viscero-sensory, and viscero-motor functions.

Diseases of the sympathetic nervous system may be divided into two large groups:

1. Primary (idiopathic).
2. Secondary (deuteropathic).

Many general diseases were, and some are still, looked upon as expressions of primary functional or structural derangement of the sympathetic nervous system. Such are, for example, migraine, angioneurotic œdema, Basedow's disease, facial hemiatrophy, erythromelalgia, scleroderma, Raynaud's disease, and Addison's disease. These and other trophic syndromes are frequently interpreted as primary diseases of the sympathetic nervous system.

The familiar syndrome of irritation or paralysis of the cervical sympathetic is a good illustration of a secondary affection. Very little is known of primary or secondary diseases of the thoracic part of the sympathetic chain. Oppenheim records a case which presented during life a unilateral œdema, the explanation of which was found in an abscess near the thoracic vertebral column. The writer published a case of abscess formation implicating the thoracic chain of the sympathetic, which presented rather obscure symptoms during life (*Med. Rec.*, June 16th, 1900).

The rôle that diseases of the thoracic part of the sympathetic chain may play in the pathology of asthma and cardiac neuroses is entirely unknown.

The relation of the abdominal sympathetic to the pathogenesis of Addison's disease, diabetes, colica mucosa, is still under discussion. Symptoms referable to disorder of the functions of the sympathetic nervous system are frequently accompaniments of almost any organic lesion of the cerebro-spinal nervous system.

The gastric and other visceral crises arising in the course of tabes, the trophic manifestations accompanying this disease as well as syringomyelia, are good illustrations in point.

Joseph Fraenkel.

SYMPATHETIC OPHTHALMIA includes two quite distinct diseases—sympathetic inflammation, or sympathetic ophthalmitis; and sympathetic irritation. These conditions are liable to arise in a previously healthy eye, after the fellow-eye has been injured and has become the seat of chronic inflammation due to such injury. In addition to these two definite forms of disease, to be described below, there are cases in which a blind degenerated eye seems to exert an unfavorable influence upon the fellow-eye.

Many surgeons believe that any blind and degenerated eyeball should be removed, if the fellow-eye becomes impaired by chronic progressive disease. Thus, if one eye presents an absolute glaucoma and the other a beginning glaucoma, the prospect of arresting the process in the latter may sometimes be improved by removing the blind eye. Or if one eye be blind with general detachment of the retina and softening of the globe, it is thought that choroidal degeneration or cataract affecting the other may be favorably influenced by removal of the blind eye. Such a belief in the dangers of sightless, degenerated eyeballs is not universal and should not be acted upon unless there is some evidence of disease in the second eye or danger of sympathetic inflammation. But if other disease of the better eye arises, the possibility of influencing it favorably by removal of a sightless degenerated stump should be carefully considered.

It is possible that in some cases inflammation of the uveal tract not caused by injury may extend from one eye to the other. But this is not certainly established. It is convenient to speak of the injured and primarily inflamed eye as the *exciting eye*, and of the other as the *sympathizing eye*.

Sympathetic Ophthalmitis.—(Synonyms: Sympathetic uveitis, Cyclitis, or Irido-cyclitis, Malignant Uveitis, or Migratory Ophthalmia.) This is an inflammation involving chiefly the uveal tract of the eye, but liable to extend to all the tissues of the eyeball, running a chronic course marked by repeated relapses, and ending in the majority of cases in complete blindness.

Causes.—Sympathetic inflammation is caused by non-purulent traumatic inflammation in the uveal tract of the fellow-eye. Injury of one eye unattended with uveal inflammation of the injured eye does not cause sympathetic uveitis. Uveal inflammation not due to traumatism does not excite this disease. Injuries to the eyeball, which are followed by severe purulent uveitis or panophthalmitis, are not likely to excite sympathetic inflammation. Disease in other organs may cause metastatic inflammation of the eye, but not the form of inflammation now under consideration. The character of the wound of the exciting eye greatly influences the liability to sympathetic disease.

The lodgment of a foreign body within the eyeball constitutes the most dangerous form of injury, unless the eye be promptly destroyed by suppuration. Yet the character of the foreign body and the structure in which it is lodged modify its evil influence. Minute aseptic foreign bodies sometimes remain in the eyeball indefinitely, without causing serious impairment of the injured eye or danger of sympathetic ophthalmia in its fellow. They are least dangerous when lodged in the crystalline

lens or in the corneo-scleral coat. They are most dangerous when suspended in the vitreous or embedded in the ciliary processes or choroid. Comparatively small or punctured wounds are more to be feared than extensive cuts or lacerations.

Wounds that pass through the anterior chamber are less dangerous than those which enter through the sclera directly into the vitreous. When the body which inflicts the injury reaches the eyeball only after passing through the lid, the danger is lessened. Wounds attended with free hemorrhage or an abundant flow of aqueous humor are less dangerous. Wounds in the ciliary region have long been recognized as especially dangerous. They are made by bodies which have not passed through the lids. They enter directly into the vitreous chamber. The track of the wound is likely to be obstructed by a prolapse of the ciliary body into it or by swelling of the injured margin of the lens. These wounds, furthermore, involve the most complex and functionally active part of the uveal tract. However, any perforating wound of the sclero-corneal coat, even the incision of a smooth, clean operation, may be followed by sympathetic ophthalmitis. Perforations of this coat by disease, as by corneal ulcer, may also cause it. Cases are reported in which injury without perforating wound, such as bruise of the eyeball with dislocation of the crystalline lens, has given rise to sympathetic ophthalmia. But it is not certain that such injuries cause sympathetic uveitis.

The form of inflammation after injury, which makes an eyeball dangerous to its fellow, is a plastic uveitis. In its typical form it is characterized by marked involvement of the ciliary body, prolonged pericorneal redness, tenderness of the ciliary region, punctate deposits on the back of the cornea, diminished tension of the eyeball, and shrinking of the globe. There are also apt to be marked involvement of the iris, posterior synechia, alteration of color or appearance from degenerative changes, and with these more characteristic symptoms may occur changes in any part of the globe. Sight is usually entirely lost.

But it must be remembered that in rare instances the changes in the exciting eye may be comparatively slight yet equally dangerous. In numerous cases sympathetic ophthalmitis has been excited by an eye which retained useful or even good vision. The redness and ciliary tenderness may be transient, the diminution in the tension of the eyeball but slight, and the changes in the iris and deposits on the cornea barely perceptible, and still sympathetic uveitis may arise and destroy the other eye. On the other hand, free suppuration is not sure to prevent an eye from exciting sympathetic ophthalmitis. Neither is mechanical traumatism essential. An eye that has had the cornea perforated by an ulcer, especially if this has been followed by prolapse of the iris, or an eye that contains a tumor, especially sarcoma or glioma, may also be the seat of a uveal inflammation that will cause sympathetic ophthalmitis. All eyes, the coats of which have been perforated either by mechanical violence or by ulceration, must be objects of suspicion.

Premonitory Symptoms.—Previous to an outbreak of sympathetic ophthalmia, the exciting eye is noticeably red and tender. Either it has failed to become entirely quiet or symptoms of irritation and inflammation have reappeared. In the sympathizing eye there may be undue sensitiveness to light, decided loss of the power of accommodation, and quick tiring of the eyes with ordinary use. Symptoms of this kind should be watched for, when there is reason to fear sympathetic ophthalmia, and their presence should excite alarm. But they cannot be wholly relied upon to foretell an outbreak of sympathetic inflammation They may be so slight as to occur unnoticed, or all of them may be absent.

Symptoms and Course of the Disease.—The actual beginning of sympathetic inflammation is insidious. Slight photophobia, increased lachrymation, or a little redness of the eye is noticed. But it is difficult to fix the exact time at which it began. As happens with other forms of uveitis, the hyperæmia may at first seem general, involving the conjunctiva as much as the pericorneal or deep scleral vessels. There may be slight discoloration of the iris and sluggishness in its reaction to light, or even one or two minute posterior synechiæ, by the time it is realized that the eye is inflamed. A little impairment of vision and faint haziness of the dioptric media may be present at the first examination.

From day to day the symptoms of uveitis become more severe and unmistakable. The lachrymation becomes excessive, the pericorneal and deep redness predominate. The photophobia becomes severe, the iris assumes the greenish or brownish tint of iritis, loses its lustre, and becomes distinctly thickened. It yields slowly and imperfectly to a mydriatic. The posterior synechiæ become more numerous and broader. The vitreous grows noticeably cloudy and the vision is greatly impaired. Points of tenderness appear in the ciliary region, and the tension of the eyeball is perceptibly diminished. There may be the severe pain of an acute iritis, but generally the pain is not great as compared with the tenderness, photophobia, and impairment of vision.

After a period of from two to six weeks the symptoms cease to grow worse, and some days later improvement is noticed. The pain and photophobia become less and vision may slightly improve. But some hyperæmia and ciliary tenderness remain, and after a remission, which may vary from a few days to several months, all the symptoms grow worse again, vision sinks lower than it has been before, the media become more obscured, and the tension of the eyeball still further diminished. Other remissions and relapses may occur, and it may be two or three years before the eye reaches its final condition of complete blindness, with greatly lowered tension, degenerated iris, and shrinking of the globe. The retina, vitreous, and lens directly suffer from the disease of the nutritive coat of the eye. The lens and vitreous become opaque. The latter shrinks and the retina becomes detached and completely altered. The cornea, besides the deposits of opacity on its posterior surface, becomes more or less opaque and shrunken. This condition may be reached in a few weeks, before the first remission has occurred, the subsequent relapses being marked merely by increased pain, soreness, and hyperæmia in the sightless and shrunken eyeball.

Atypical Cases.—The course of the disease as altered by treatment will be discussed under that head. In a few cases impairment of vision is at first the only symptom noticed. Ophthalmoscopic examination reveals hyperæmia, haziness, and slight swelling of the head of the optic nerve, with haziness and change of color, and perhaps swelling of patches of the choroid and retina. Cases of this kind usually develop later the more characteristic symptoms of sympathetic uveitis. They have been described as cases of sympathetic neuro-retinitis. But they are probably essentially uveal, the choroid being at first chiefly affected.

As with other forms of uveitis, structures outside the uveal coat, and not directly dependent on it, are liable to be involved. Quite apart from the usual deposits on the posterior surface and the opacity due to degeneration, the cornea may be early affected with interstitial or ulcerative inflammation; or severe conjunctivitis may be present. So prominent are these complications in certain cases that they have been reported as cases of sympathetic keratitis or conjunctivitis. But in all cases in which the sympathetic character of the disease can be regarded as demonstrated there has also been distinct evidence of uveitis.

Diagnosis.—The symptoms presented by the sympathizing eye are not in themselves a sufficient basis for the positive diagnosis of sympathetic ophthalmia. They are merely those of a severe uveitis which tends to become chronic, to relapse, to go on to complete blindness and degeneration of the eyeball. This uveitis is especially insidious in its manner of beginning and progress, and it exhibits a special tendency to cause great diminution in the vision and in intraocular tension. But these features are not alone sufficient to distinguish it from

other forms of uveitis. The history of traumatism and uveal inflammation of the other eye are essential to the diagnosis; not traumatism alone, for this does not cause sympathetic disease, unless it has been followed by uveal inflammation.

The time which has elapsed since the injury must also be considered. Sympathetic inflammation rarely if ever arises within three weeks from an injury. Usually the interval is six weeks or more. On the other hand, it rarely begins more than two or three months after the injured eye has become entirely quiet and free from inflammation. Cases beginning more than six months after the exciting injury are unusual, and more than a year afterward are quite rare. Still a few cases are recorded in which the exciting eye has remained quiet for many, sometimes thirty or forty, years before causing an outbreak of sympathetic ophthalmitis. Eyes that have been so long injured are more apt to cause sympathetic irritation. The diagnosis between these two conditions will be considered in connection with the latter.

An attack of iritis or other form of uveitis occurring years after an injury to the other eye should not be regarded as necessarily one of sympathetic inflammation, although that should be suspected. If the injured eye remains free from tenderness and irritation, even though it be entirely blind, and especially if its tension is normal or nearly so, and the attack does not show the special characteristics of sympathetic uveitis, the case is probably not one of sympathetic disease.

Pathology.—The anatomic changes which mark sympathetic ophthalmia are those of plastic uveitis. There are exudates which become organized into connective tissue, which may in time ossify, while the characteristic elements of the ocular tissues degenerate and atrophy. But although the transmission of disease from one eye to the other has been observed with especial interest for two hundred years, the method of transmission is still unknown. Mackenzie, in 1844, suggested transmission along the optic nerve. Müller, in 1858, suggested transmission by the ciliary nerves. A morbid functional activity was assumed, a reasonable hypothesis to explain sympathetic irritation, which was then confused with sympathetic inflammation. In 1881 Snellen argued that this was a specific inflammation, caused by organisms transmitted through the lymph spaces of the optic nerves. Berlin suggested that micro-organisms causing it might be carried to the exciting eye by the blood current, and find conditions suitable for their development only in the uveal tract of the sympathizing eye. Deutschmann claimed to have produced sympathetic ophthalmia in the rabbit, and to have demonstrated its extension along the optic nerve. Gifford and others failed to confirm his experiments, and showed that the probable explanation of his results was a general infection rather than a sympathetic ophthalmia. Randolph, using dogs, which are less liable to general infection, found that the fellow-eye was unaffected. Finally, it has been suggested that reflex influences through the ciliary nerves may prepare the uveal tract of the sympathizing eye for the invasion of organisms reaching it through the circulation.

That nerve influences starting from the exciting eye may powerfully affect the sympathizing eye is demonstrated by sympathetic irritation and its immediate relief by the removal of the exciting eye. It is equally certain that such nerve influences do not alone cause sympathetic ophthalmitis. In this disease some other factor must be added. Infection with a distinct period of incubation is the factor that best accords with our present ideas of such inflammations. The period of incubation may cover the two or three weeks that always elapse between injury of the exciting eye and sympathetic ophthalmia, or during which sympathetic inflammation is liable to occur after the removal of the exciting eye.

Prevention.—With a disease which tends to cause complete blindness, and which often goes on to this termination in spite of all treatment, prophylaxis is of the highest importance. In timely enucleation of the injured eye we have a certain preventive. The only questions that arise in connection with it are: In what cases should it be applied, and have we any other equally or sufficiently reliable prophylactic?

It cannot be said in any case of injury that enucleation is demanded to prevent sympathetic disease until a plastic uveitis has been set up. But with regard to an eye known to contain a foreign body of more than the most minute size that cannot be extracted, it may be assumed with almost absolute certainty that such dangerous uveitis will occur; and if an eye be so injured that future vision with it is impossible, immediate enucleation will so shorten the period of disability that it will often be justified. When the eye has become the seat of uveitis due to injury, and the inflammation does not begin to subside within three or four weeks, especially if the tension of the globe is much below normal, if blind it should be promptly enucleated. If not blind, but with vision no longer useful and diminishing, the eye should be enucleated if the patient cannot remain under observation. If the eye be blind with greatly diminished tension from an injury that has occurred within two years, even though it be free from hyperæmia or tenderness, it should be enucleated if the patient cannot remain within reach of competent professional assistance. The location of a wound in the ciliary region is an added reason for enucleation. Any blind eye that is known to contain a foreign body should be enucleated.

None of the substitutes for enucleation are so certain to prevent sympathetic inflammation. They have a field of usefulness, but not for this purpose. Nevertheless, if a patient absolutely refuses enucleation, but will allow evisceration, it may be proper to do the latter operation rather than decline further care of the case. In doing evisceration the sclera should be freed from all remains of its contents and mopped with a strong germicide, as carbolic acid or a solution of formaldehyde.

Enucleation of an injured eye does not instantly confer immunity from sympathetic uveitis. In a good many cases the inflammation has appeared within two or three weeks after the removal of the exciting eye, and in a few cases as late as four weeks. But almost invariably sympathetic inflammation arising in this way has yielded to treatment and has ended in recovery.

Treatment.—The first step is removal of the exciting eye. The only exception is when the exciting eye retains useful vision. There are a number of cases on record in which the sympathizing eye became entirely blind, while the exciting eye retained useful vision for many years or throughout life. It has sometimes been thought that removal of the exciting eye was followed by increased violence of the sympathetic inflammation. But probably this was nothing more than the increase observed when the exciting eye was not disturbed. On the other hand, the cases in which the exciting eye has been removed especially if removed early, show a much larger proportion of recoveries than do those in which the exciting eye was allowed to remain. The strong tendency to recovery in cases of sympathetic inflammation arising after enucleation also attests the value of this measure.

The sympathizing eye should be placed under the influence of atropine as soon as possible. A strong solution, two per cent., may be instilled, one drop every ten minutes for an hour; or after the use of cocaine a small crystal of atropine sulphate may be placed in the conjunctival sac, near the external canthus. Until this crystal is dissolved, or while instilling the strong solution, we should guard against atropine poisoning by everting the lachrymal puncta through traction on the skin at the side of the nose, and holding in contact with them a little absorbent cotton, thus keeping the atropine solution from getting into the tear passages. After this first mydriatic attack the strong solution of atropine should be instilled two or three times a day.

Before each instillation of atropine, or at more frequent intervals if the eye is painful, it should be bathed with very hot water, not longer than five minutes at a

time. Dry heat, best from the electric coil, may be applied for periods of one-half hour to two hours. If the hyperæmia is severe, and especially if there is pain, one or two fluidounces of blood may be taken by leeches or by the artificial leech applied to the temple. Subconjunctival injections of physiologic salt solution may be made at intervals of two or more days. These should be placed rather deeply within the capsule of Tenon. On going out in daylight the eye should be protected by very dark glasses or an opaque shade; but it should not be kept continuously covered by thick dressings.

As in other forms of uveitis, general treatment is of great importance. Mercury should be given freely up to the point of causing tenderness of the gums or other evidences of its poisonous action. At first, calomel may be given until it acts as a purgative, with large inunctions of mercurial ointment twice daily. Later, any form of mercurial that will sustain the mercuric influence without symptoms of poisoning may be employed. Nothing will be gained by carrying the action of mercury beyond the point indicated. But its administration should be continued until all symptoms of inflammation have disappeared.

Next to mercury, the most important drug is sodium salicylate. This should be given in the largest doses that will be tolerated; from twenty to forty grains, three or four times a day, in an adult. But such doses need not be continued more than a few days. Later, it may be given in smaller doses or discontinued. Should this drug cause serious disturbance of the stomach, other salicylates or aspirin may be substituted. For a chronic case or at a later stage, the iodides may be beneficial.

It is very important that the patient should be kept in as good a state of general health as possible. On this account the administration of mercury and the salicylates must be closely watched. The patient must not be confined to a dark room. The use of alcohol in any form should be forbidden. Diet and rest must be carefully attended to, and general tonics, especially iron, may be indicated. After apparent cure the patient should be kept under observation, and should avoid excessive use of the eyes or indulgence in alcoholic drinks for many months.

When the attack of inflammation has been finally subdued, it may leave the pupil closed by exudate, with or without an opaque lens. This condition might require for its relief an operation, but the eye should be allowed to remain quiet for years before any such operation is attempted. If done within a year or two after the inflammation has subsided, it will almost certainly cause a recurrence of the disease. Nothing will be gained by the attempt, and much will be lost.

Prognosis.—Sympathetic inflammation, untreated, ends in blindness; and, if those cases which arise after the enucleation of the exciting eye be excluded, probably less than half can be cured by treatment. The nature of the injury or the violence of the inflammation in the exciting eye has little influence over the prognosis when once sympathetic disease has actually occurred. Attacks beginning with lesions in the fundus show a rather better proportion of cures than those commencing with marked iritis. The first remission must not be mistaken for a cure. But when recurring exacerbations grow less severe and the sight is left no worse by them, an ultimate cure with partial or complete restoration of vision may be hoped for. Not until the eye has been free from inflammation for a year should the case be considered cured.

Sympathetic Irritation, or Sympathetic Neurosis.—This is a condition set up by disease in the fellow-eye and characterized by extreme irritability, photophobia, excessive lachrymation, moderate general hyperæmia, and inability to use the eye, sometimes equivalent to complete temporary blindness.

Cause.—This is disease in the fellow-eye, generally of long standing and marked by very extensive degenerative changes. The exciting eye may show no symptoms of present inflammation, no hyperæmia or tenderness. Its condition may be the result of injury, or it may be the result of disease quite independent of traumatism. Intra-ocular tumor, without perforation of the ocular coats, may cause it. Ossification of the choroid is a quite common cause.

Symptoms.—It begins with inability to use the eyes for ordinary work, photophobia, and increased lachrymation. The symptoms vary in severity from time to time, but tend on the whole to grow worse, until any use of the eyes becomes impossible. On close examination the cornea will be found free from deposits, the pupil small but reacting well, the iris normal; and if photophobia does not prevent an ophthalmoscopic examination, the media are found clear and the fundus is normal. The hyperæmia is quite general, may disappear when the eye is kept quiet in a dark room, but increases rapidly on attempting to use the eyes or even with examining them.

Diagnosis.—Sympathetic irritation has to be distinguished from eye strain and from sympathetic inflammation. Hyperopia, astigmatism, or presbyopia may cause apparent lack of accommodation, excessive irritability of the eye, lachrymation, photophobia, and difficulty with near work. Therefore the refraction and accommodative power should be carefully measured before adopting the diagnosis of sympathetic irritation, even though the fellow-eye presents all the conditions that would make it the cause of this neurosis. By placing the suspected eye under the influence of a mydriatic, eye strain can be excluded.

To differentiate between sympathetic inflammation and sympathetic irritation is often of great practical importance. Their chief points of difference may be contrasted as follows:

Inflammation.	*Irritation.*
1. Follows perforation of the sclero-corneal coat, usually by traumatism, especially if the wound is in the ciliary region or if a foreign body is lodged in the eye.	1. Follows extensive degeneration of the fellow-eye, with or without traumatism, especially if the eye contains a foreign body or new-formed bone.
2. Occurs at from three weeks to six months after injury; is rare after one year.	2. Occurs usually many years after injury, or disease, of the exciting eye.
3. The exciting eye is almost always red or sore before the outbreak.	3. The exciting eye may be entirely quiet and free from irritation.
4. There are deposits on the posterior surface of the cornea, the iris is discolored, its reaction impaired. There are posterior synechiæ; the vitreous is hazy; vision is permanently diminished.	4. The cornea is free from deposits, the iris normal; the vitreous clear, and the eye may for a short time be capable of normal vision.
5. There are always marked organic changes.	5. There are no organic changes.
6. There are relapses at intervals of many days, weeks or months.	6. The severity of the symptoms may vary from hour to hour.
7. It terminates in slow recovery under treatment; or, more frequently, in complete blindness.	7. It tends to continue indefinitely; but ends in immediate restoration to normal after removal of the exciting eye.

The differential diagnosis is only difficult at the outset before the organic changes of sympathetic inflammation have had time to occur.

Pathology.—No anatomical changes occur, and the morbid influence is undoubtedly transmitted from the exciting to the sympathizing eye through the ciliary nerves.

Prevention is of little importance. The only effectual preventive measure is removal of the eye that is liable to cause it, and this is equally efficient after the condition of sympathetic irritation has actually developed. And since a degenerated eyeball may be retained many years without causing sympathetic irritation, the possibility of that event at some future time does not justify enucleation.

Treatment.—This is enucleation of the exciting eye or one of the substitutes for enucleation. Where sympathetic irritation alone is to be feared, probably evisceration or even optico-ciliary neurotomy will be as efficient as the removal of the eyeball. If preferred by the patient or operator, resort to one of them, preferably evisceration, in place of enucleation, is entirely proper. If

the patient strenuously objects to operation and the case is clearly not one of sympathetic inflammation, palliative measures may be tried. Avoidance of close work and keeping the eye under a mydriatic, as atropine sulphate 1, distilled water 100, instilled twice a day, will often give partial or temporary relief. The internal administration of bromides is also useful. In a few cases freedom from irritation is thus secured, and it may last for months or years. But in nearly all cases the return of the symptoms, and the continual annoyance and disability which they cause, will in time overcome the strongest objections to operative treatment.

Prognosis.—Without treatment the irritation continues or recurs indefinitely. With removal of the exciting eye the prognosis is entirely favorable. Donders reported a case in which the patient had believed himself entirely blind for two years, but was proved to possess normal vision two hours after the removal of the exciting eye.

Edward Jackson.

SYMPHOROL-SODIUM, SYMPHOROL-LITHIUM, and **SYMPHOROL-STRONTIUM** are the respective caffeine sulfonates of these metals. They are white, odorless, and bitter, are readily soluble in water, except the sodium salt, and are insoluble in ether, benzol, or chloroform. These salts are strongly diuretic in dosage of 1–4 gm. (gr. xv.–℥ i.) a day, and are claimed to retain the diuretic effect of the caffeine without its stimulating action on the heart and nervous system. The sodium salt is in common use under the simple term "symphorol."

W. A. Bastedo.

SYMPHYSEOTOMY. See *Obstetric Operations* and *Pelves, Deformed.*

SYNCOPE. See *Brain Diseases: Anæmia.*

SYNCYTIOMA.—(Synonyms: Deciduoma malignum, Sarcoma uteri deciduocellulare, Chorionic epithelioma, Choriocarcinoma, Syncytioma malignum, and Carcinoma syncytiale.)

Under syncytioma are to be considered those new growths which consist largely or in part of syncytium such as is found covering the chorionic villi. In order to understand clearly the relation and origin of this class of tumors, it is necessary to have a knowledge of the placenta and its development. Of especial importance in this relation is the development of the chorionic villi. The villi are at first clothed with fœtal ectoderm, which, we have reason to believe, becomes two-layered in early embryonic life; the inner layer, the "cell layer" of Langerhans, being composed of separate individual cells with large vesicular nuclei and reticular protoplasm; the outer, or "syncytial" layer, being composed of a continuous mass of dense, deeply stained protoplasm containing smaller nuclei than the preceding layer. The syncytium resembles not a little a huge giant cell. In tumors of this group both the syncytium and the cell layer of Langerhans are usually represented in varying proportions. The origin of the syncytium has long been a matter of dispute, some observers maintaining that it is derived from the epithelium of the uterine mucosa, while others believe that it arises by delamination from the ectoderm covering the ovum. A study of preparations of developing placentas furnishes convincing evidence that the uterine mucosa takes no part in the formation of the syncytial layer. The close relation of the syncytium to the cell layer of Langerhans and the presence of transitions between the two make it clear that the syncytium arises from the fœtal ectoderm.

From the above observations it would appear that a syncytioma is to be regarded as a malignant form of new growth originating from fœtal ectoderm. In the discussion of these tumors the term "syncytium" applies only to a special tissue, identical with or closely resembling the so-called syncytial layer of the chorionic villi. Syncytia other than this special form have no relation to the tumors in question.

Syncytiomata may be divided into two groups:

1. Certain rare cases of teratomata in which a syncytium develops.

2. Those tumors arising in the uterus after pregnancy to which the terms "deciduoma malignum," "syncytioma malignum," etc., have been applied.

Several teratomata of the testicle have been recently described in which a portion of the growth resembles in every respect a true syncytioma of the uterus. Such growths are described by Carey, Wlassow, and Schlagenhaufer. The syncytium in these teratomata represents one of the many lines of tissue differentiation found in these neoplasms, and, from morphological characteristics as well as on theoretical grounds, it may be considered as homologous to the true chorionic syncytium. Teratomata in which the syncytium forms a large portion of the tumor appear to possess a considerable degree of malignancy; for example, metastases have been described as well as a tendency of the growth to infiltrate surrounding tissues. On account of the rarity of syncytial growths associated with teratomata, and since they comprise only a portion of these neoplasms, they are here given but brief mention. Concerning the further characteristics and the theories of origin of teratomata, one should consult monographs on that subject.

Deciduoma Malignum.—*Historical.*—The term deciduoma malignum was first applied by Sänger in 1888 to a peculiar malignant growth of the uterus with metastases following an abortion. Although this term is considered to be a misnomer, it is generally deemed advisable, on account of the multiplicity of terms, to retain, for clinical usage at least, Sänger's original terminology. Sänger believed that the tumor in his case consisted of decidual cells, and he placed it among the sarcomata. Following this and quite independently of Sänger, Pfeiffer described a case and also applied the term deciduoma malignum. In 1893 Sänger published a monograph which comprised all that was then known of this subject. In 1895 Williams and Marchand published monographs. Williams, after reviewing the literature, came to the conclusion that in the various cases reported there is some variation in the nature of these tumors, that is, that some consist for the most part of decidual cells and are thus of maternal origin, others consist of tissue analogous to the syncytium and Langerhans' cell layer and are of fœtal origin, while it is not improbable that others are of both maternal and fœtal origin.

Marchand believed that both the syncytial and the cellular portions of these tumors are of fœtal origin. A great many cases have been reported since the publications of Williams and Marchand. Ladinski, in 1902, was able to collect one hundred and thirty-two cases. Such is the comparative frequency of their occurrence that they are worthy of consideration to the clinician in cases of uterine hemorrhage.

Occurrence.—Deciduoma malignum may occur at any time during the child-bearing period. That it may not be discovered until after the menopause is shown by a few cases. It is always associated with pregnancy, appearing after a full-term labor, an abortion, or the expulsion of a hydatidiform mole. It is evident that in certain cases the tumor has begun to develop before the expulsion of the mole. The frequent association of this tumor with vesicular moles is to be noted. Dorland, in a series of fifty cases of deciduoma malignum, found that twenty-two cases (43.3 per cent.) gave a history of the expulsion of a mole at some time prior to the appearance of the growth.

Gross Appearances.—Metastases of the growth are often found in the vaginal wall. They appear as soft, vascular nodules, friable, often necrotic and ulcerated, and sometimes protruding from the vulval orifice. The cervix is usually soft and enlarged from the preceding pregnancy. On opening the uterus the primary growth will usually be found bulging from some part of the uterine wall. It varies in size and form, is reddish in color, sometimes mottled with gray. The primary growth is often necrotic and is always soft and friable. The uterine