diet, excess of food, additional meals such as midnight suppers, entertainment "refreshments," etc. Hence the greater prevalence of migraine among the wealthy classes. A kindred cause is excessive muscular fatigue, a phenomenon also due to the presence of toxic wastes (principally sarcolactic acid) in quantities which cannot be disposed of by the blood sufficiently fast to prevent the development of morbid phenomena.

In predisposed subjects attacks may, on the other hand, be evoked by conditions which tend more or less to debilitate the general nerve centers implicated.* Emotional factors, shock, fear, grief, worry, anger, excitement, etc., are familiar exciting causes. Equally familiar are the reflex causes of migraine, eye-strain, nasal and aural disorders, uterine diseases, carious teeth, adenoids and other conditions enumerated as capable also of exciting epileptic seizures, and occupations which entail more or less cerebral hyperæmia, especially intellectual overwork, coupled with inadequate out-of-door exercise.

Rachford[74] has clearly demonstrated that an excess of paraxanthin and other xanthins was present in the urine during and for some time after an attack of migraine, and moreover (1895), that an excessive excretion of paraxanthin coincided with it. This is in accord with the views of French clinicians, Guéneau de Mussy, Bazin, Jaccoud, Labadie-Lagrave, Bouchard and others, who, from the time of Trosseau (who held that "migraine and gout are sisters"), have connected migraine with the so-called "gouty diathesis." Bouloumié,[75] in a study of 1348 cases of gout and kindred disorders treated by him at Vittel, found that the prodromes of migraine frequently occurred as the preliminary manifestations of such disorders, that migrainous attacks appeared especially before and during gouty phenomena, and rarely subsequent to them. Fluctuations in the excretion of uric acid have often been observed and recorded by Haig,[76] a copious elimination of this end-product of metabolism coinciding with the disappearance of the migrainous attack. The pathogenetic process submitted requires no evidence in view of that adduced in the articles on Epilepsy, Eclampsia, etc. The influence of heredity and the connection of eclampsia with epilepsy are well shown by the statistics of Féré—a history of migraine in 232 out of 308 cases of epilepsy.

Treatment.—PROPHYLAXIS.—The general indications, in view of the pathogenesis of the disorder, are (1) to adjust the patient's diet to his assimilative powers, thus reducing to a minimum the toxic wastes formed, and (2) to overcome any condition which, either inherited or acquired, tends to reduce the

* *Author's conclusion.*
[74] Rachford: Amer. Jour. Med. Sci., *Loc. cit.*
[75] Bouloumié: Bull. méd. des Vosges, Oct., 1895.
[76] Haig: "Uric Acid as a Factor in the Causation of Disease," 1892.

anterior efficiency of his blood, by inhibiting the functional efficiency of his general nerve centers.*

The first indication is met by reducing markedly the use of highly-nutritious food, late suppers, pastry, red meats and alcoholic beverages being avoided, and meals should be taken at regular intervals. Coffee and tea are harmful, since they raise the vascular tension. Water, however, should be partaken of in large quantities to preserve the blood's fluidity and osmotic properties, and to insure free diuresis. An occasional saline purgative is of service.

The second indication includes the correction of any ophthalmic, nasal, and other condition capable of provoking migraine reflexly.

Liveing,[77] referring to the many distinguished men who suffered from this disorder, states that Marmontel cured himself by eating little, drinking water, taking exercise, and that Haller was equally successful "by drinking every day a large quantity of water, and exchanging a highly nutritious regimen for a much lighter dietary." Linnaeus cured himself by the same means, etc. Patients readily consent to a reduced diet as a rule, but they usually fail to carry out the physician's instructions as to the plentiful use of water. In such cases it is best to order some alkaline water, Ballardvale, Buffalo Lithia, Vichy, etc., and to instruct them to take a glassful at established intervals, one quart being drunk daily. As to reflex causes, according to Lauder Brunton,[78] a careful examination of the eyes, teeth, nose, ears, and throat will reveal some disorder in these organs in practically all cases. This is only mentioned to emphasize the fact that this feature of the treatment should not be neglected.

Preventive remedies should obviously be such as are capable of stimulating the anterior pituitary in order to enhance the proportion of auto-antitoxin in the blood. *Thyroid gland,* 2 grains (0.13 gm.) during each meal, and an occasional saline cathartic are very effective, especially if the patient can lead an out-of-door life.* *Potassium iodide,* 5 grains (0.3 gm.) after meals in a large glassful of water, is equally efficacious, and is indicated when any cardiac disorder is present. *Strychnine,* $^1/_{40}$ grain (0.0016 gm.) three times a day, is of signal service, especially when the peripheral circulation is poor, although it tends to stimulate the vasomotor center besides the adrenal center.* It is especially valuable also when there is a tendency to melancholia, or mental depression, both frequently observed in

* *Author's conclusion.*
[77] Liveing: "Megrim, Sick-headache, etc.," p. 433, 1873.
[78] Lauder Brunton: Practitioner, Feb., 1894.

subjects predisposed to migraine. In children or adolescents strychnine is advantageously combined when constipation does not complicate the case, with *iron,* especially the dialyzed iron or Blaud's pill.* The dietetic measures recommended should, of course, be carried out simultaneously.

The thyroid extract in small doses has given me excellent results, especially in women approaching the menopause, and also where prompt effects were necessary in any case of migraine. Liveing obtained "singular success" from potassium iodide in some cases. J. R. Clemens[79] found it effective in the most aggravated cases. The value of strychnine and iron in such cases is well known. This clearly illustrates the fact that preventive medication in this disorder includes agents which act as "tonics" or, in other words, as stimulants of vital activities through the adrenal system.

Digitalis is indicated when the right heart is dilated, *quinine* when there is history of malarial toxæmia, *sodium salicylate* when gout is a feature of the case or of the history.

Drugs which Reduce Arterial Tension.—When the foregoing measures do not yield the desired result, it is owing to excessive irritability of the sympathetic and vasomotor centers, which causes them to react to the least excitation.* The accesses recur frequently, and the face is usually pale in such cases. *Nitroglycerin* relaxes the entire arterial system, increases diuresis, etc., by reducing the sensibility of the sympathetic and vasomotor centers. Its action should be carefully watched, however, since the recession of blood from the peripheral capillaries tends in itself to inhibit catabolism. For the same reason, the doses should not be too large, $^1/_{60}$ grain (0.001 gm.) twice daily is ample. It may be used with the thyroid extract, potassium iodide and digitalis, but not with strychnine, which tends to counteract its action.* The *bromide of sodium* can be used instead of nitroglycerin, in hysterical women; but as it tends to inhibit oxygenation when given in large doses or when its use is prolonged, it should only be employed temporarily.

Nothnagel and Rossbach[80] state that the lowering of the arterial tension caused by nitroglycerin is not harmful: "the flow of urine is increased and albuminuria disappears." As the latter symptom is due to excessive vasoconstriction, its disappearance proves that the remedy is beneficial by causing general vasodilation.

* *Author's conclusion.*
[79] J. R. Clemens: Therap. Gazette, May 15, 1903.
[80] Nothnagel and Rossbach: "Nouveaux éléments de mat. méd. et de thérap.," Paris, 1889.

TREATMENT OF THE ATTACK.—*Drugs which Reduce Arterial Tension.*—Agents capable of producing this effect promptly do not seem to have been tried in migraine; *amyl nitrite* suggests itself as a valuable agent in this connection, provided, however, that the amount inhaled be small, *i.e.,* not more than 5 or 6 drops. *Chloral hydrate* combined with *sodium bromide,* 10 grains (0.6 gm.) of each repeated in two hours, and taken with a wineglassful of water, sometimes wards off an attack. If the pain appears, the local application of *camphor-chloral,* a mixture of the two drugs, equal parts, which forms a viscid liquid, often checks it.

Drugs which Cause Contraction of the Arterioles.—When the arterioles (those which, owing to their failure to contract, allow the blood to penetrate the affected area) are not the seat of sclerosis, stimulation of the sympathetic center will sometimes enforce their contraction, and thus arrest the pain.* *Acetanilid* and *antipyrin* are very effective agents in this connection, 5 grains (0.3 gm.) given every hour three times, usually sufficing to arrest the paroxysm. *Phenacetin* is equally active, but in large doses, *i.e.,* 10 to 15 grains (0.6 to 1 gm.). *Morphine* is eminently an arteriole constrictor, and a subcutaneous injection of $^1/_6$ grain (0.01 gm.) sometimes acts very promptly. Other agents that have a corresponding physiological action, *caffeine, guarana, ergot, cannabis indica,* have been recommended, but their effects vary so greatly, owing probably to the uncertain strength of the preparations available, that dependence should not be placed upon them.

NEURITIS, INCLUDING NEURALGIA, TIC DOULOUREUX, SCIATICA AND ZONA (SHINGLES, HERPES ZOSTER).

Definition.—Inflammation of a nerve, whether termed "neuralgia" or "neuritis," may occur as a result of so many morbid influences, active and passive, that a specific definition cannot be formulated. It is always attended, however, by engorgement of the nerve's arterioles or vasa nervorum and their capillaries, including those of the nervi nervorum, the hyperæmia of the latter and the pressure of the swollen structures surrounding them being the main source of pain.**

* *Author's conclusion.*
** *Author's definition.*

Symptoms and Pathology.—"Neuralgia," meaning merely pain in the course of a nerve or nerves, is but a symptom of a morbid condition of these structures, and not, therefore, a disease. As it is invariably associated with hyperæmia,* which often progresses to the stage of inflammation, neuralgia is, in reality, but a symptom of neuritis, not only of the incipient stages of this disorder, but also of its advanced stages: *tic douloureux,* for example.*

Dana[81] states that when there is organic disease of the nerve itself, such as neuritis, the disease cannot be, strictly speaking, called neuralgia; and that "it is often impossible to draw the lines absolutely." Some authors limit the term "neuralgia" to cases in which hyperæmia of the nerve is alone present; but there is no legitimate foundation for this interpretation, since, as stated by Dana, "there is hyperæmia with sometimes extravasation of blood" both in interstitial neuritis and perineuritis. Moreover, most acute pain persists even when profound changes of the nerve extending to and involving the ganglion—the Gasserian ganglion in trigeminal neuralgia, for example—are present. Hence the appropriateness of the term "neuralgia" even here—but only when linked with "neuritis."

In the forms of neuritis termed *neuralgia* the pain is sometimes preceded by numbness or stiffness, or conversely by soreness, tingling or throbbing in the affected area, but, as a rule, it appears spontaneously. At first, it is generally intermittent, and may be sharp, stabbing or burning in character, and be localized or extend throughout the entire length of the nerve. The paroxysms, which vary greatly in intensity, occur at irregular intervals of a few minutes in mild cases, and may then only recur after considerable time. When simple hyperæmia has lapsed into inflammation, however, intense pain—usually worse at night—may persist days, weeks, months, etc., *i.e.,* until the cause is eradicated.

While at first the surface of the affected area may present some numbness, or be normally sensitive, the region becomes hyperæsthetic when inflammation of the nerve is impending or is present, especially where the nerve overlies a hard muscle, or passes over the edge of a bony opening, the supraorbital foramen, for instance. The painful area is often infiltrated, swollen and red. In simple hyperæmia, firm pressure often relieves the pain, but the same procedure greatly augments the pain when active inflammation is present.

* *Author's conclusion.*
[81] Dana: "T. B. of Nerv. Dis. and Psych.," sixth edition, p. 156, 1904.

The pain is due to congestion of the minute capillary networks in the peri- and endoneurium, the larger vessels of which are arterioles supplied with sympathetic fibers. Congestion of these arterioles and capillaries gives rise to pain by causing swelling of the nerve and pressure upon the nervi nervorum, which are themselves hyperæmic, and, therefore, hypersensitive.*

The nervi nervorum, the existence of which was shown by Horsley, terminate in minute bulbs similar to the tactile end-bulbs of Krause, and are, therefore, sensory. Weir Mitchell and Marshall concluded that the pain of neuritis and neuralgia was due to pressure upon these delicate sensory organs. That hyperæmia is the source of this pressure, is suggested in many ways. As Gowers[82] says, "we know that in all organs, vascular dilatation attends functional activity." That hyperæmia—a result of vascular dilatation—is the cause of neuralgia, *i.e.,* nerve-pain, is likewise recognized. In neuritis, the case is the same. Tyson,[83] for example, states that "an inflamed nerve is reddish, from hyperæmia of the *vasa nervorum,* though the stage of demonstrable hyperæmia may have passed away when the nerve comes under observation." This coincides with the definition of Dana previously given.

Each of the two disorders included in the term neuritis-neuralgia has been divided into special varieties, the principal of which are *tic douloureux,* trifacial or trigeminal neuralgia, sciatica, the regional neuralgias and multiple neuritis.

Trifacial Neuralgia or Tic Douloureux.—This is the most painful of the entire series, and is the most common. The *ophthalmic division* of the fifth is that most frequently affected, the familiar supraorbital neuralgia. The pain, which may radiate to the inner angle of the orbit, the eye and lids, the eye-ball and the corresponding side of the nose and head, is apt to recur at stated periods morning or night. Lachrymation, conjunctival congestion, local flushing, sweating and swelling, are often observed. The least effort increases the pain, each pulse-beat causing an exacerbation. Such an access may last one or more hours, and cease spontaneously, to return perhaps after a few weeks or months, or it may steadily grow worse and cause many years' excruciating suffering.

Next in order is the *superior maxillary division,* the tender point of which is at the intraorbital foramen, the pain radiating to the malar bone, over the cheek, the corresponding side of the nose, and some of the upper dental nerves. Less common is that

* *Author's conclusion.*
[82] Gowers: *Loc. cit.*
[83] Tyson: *Loc. cit.*, p. 867, 1905.

of the *inferior maxillary division,* with two tender points, one at the mental foramen, the other just in front of the ear, the pain extending over an extensive area in some cases, *i.e.,* the preaural region, the tip of the tongue, the lower jaw, the inferior dental branches, the ear, the temple and the parietal eminence.

The primary changes in the affected nerves are hyperæmia, as stated, but in cases in which the neuralgia persists, inflammation occurs, owing to disease of the neural arteries.

Dana[84] long ago contended that many cases, at least, are due to arteritis. In five cases of trigeminal neuralgia, he found no noteworthy changes in the nerves, while in three there was marked arterial disease. Putnam[85] likewise found, in some instances, the intima of the blood-vessels greatly thickened. Thoma,[86] Rose[87] and Keen and Spiller[88] and others have all found a marked increase in the size of the vessels, the two former in segments of peripheral nerves derived from cases of trifacial neuralgia, the last-named authors in Gasserian ganglia derived from similar cases.

Even the axis-cylinders, which,. as I have pointed out, are channels for the blood's oxidase, are engorged: Stengel,[89] referring to neuritis, says, for instance: "In the acute forms partially degenerated fibers with fatty myelin-sheaths and swollen axis-cylinders, are found very early." Again, Spiller found swollen and irregular axis-cylinders and small vessels in some of the Gasserian ganglia resected by Keen. Of one of these he says, referring to the axis-cylinders: "In most portions of the field, these appear as drops of a red, hyalinelike substance," doubtless methæmoglobin and adrenoxidase.

SCIATICA.—The pain usually starts in the upper part of the sciatic nerve in the gluteal region immediately behind the great trochanter, and follows the course of that nerve, radiating downward into the popliteal space and thence to the internal malleolus and the dorsum of the foot. It is usually very acute, and is increased by the least motion. By raising the limb bodily, thus producing flexion at the pubis, Lasègue's sign is elicited: a violent pain along the entire sciatic. There is marked tenderness over the latter, especially between the ischium and the great trochanter. Sometimes there is fever at the outset. There may be muscular tremor, a forerunner of atrophy of the muscle, or œdema. The skin of the region may then be pale and glossy, its temperature being also reduced. An eruption identical to that of herpes zoster occasionally develops along the

[84] Dana: Med. News, May 16, 1891.
[85] Putnam: Boston Med. and Surg. Jour., Aug. 13, 1891.
[86] Thoma: Deut. Archiv f. klin. Med., Bd. xliii, S. 409, 1888.
[87] Rose: Trans. Med. Soc. of London, vol. xv, p. 157, 1892.
[88] Keen and Spiller: "On Resection of the Gasserian Ganglion," Phila., 1898.
[89] Stengel: "T. B. of Pathol.," third edition, p. 831, 1900.

course of the nerve. The positions assumed by the patient when standing, sitting, etc., are all calculated to favor the diseased limb.

Sciatica is a neuritis from the outset, the primary infiltration, due to the accumulation of colorless blood-plasma in the nerve, being the initial phenomenon of an inflammatory process, which, like all forms of "neuralgia" and neuritis, may culminate in degenerative changes in the nerve.

Sciatica is the only "neuralgia" in which exception might be taken as to its being a neuritis. Thus, while Herter[90] ten years ago referred to sciatica as a neuritis, *i.e.,* as "an *inflammation* of the great sciatic branch of the sacral plexus," Dana, in the last edition of his text-book, says that "a large proportion of the cases is due to a neuritis." In the light of my views, sciatica is a neuritis in all cases, since the exceptions are all attended with hyperæmia, the first stage of inflammation. Again, "hyperæmia" is generally supposed to be denoted by redness of the neural structures involved, but we may have hyperæmia without redness, since, as I have shown, the blood-plasma that circulates in the terminal neural capillaries, including the axis-cylinder, does not contain red corpuscles. Indeed, if we term this fluid "serum" (of which the *extra corpore* serum is but chemically-altered semblance) it is possible to trace the inflammatory process from its incipiency to that of clearly defined trophic lesions. This is facilitated by a review of the literature embodied in a paper by J. Ramsay Hunt.[91] In a case in which Cotugno[92] found *œdema* of the nerve-sheath "15 c.c. (1/2 ounce) of *serum* was removed by puncture." Martinet[93] "described the nerve as *œdematous* and of *reddish hue*, with hæmorrhages into the sheath" in one case and as "infiltrated with *serosanguineous* fluid" in another. This evidently refers to the continuity of the nerve, for Gendrin,[94] in a case of fifteen days' duration, found the nerve-trunk "*reddened*, swollen, and the seat of serous infiltration down to the popliteal space. All this is due, of course, to engorgement of the vessels and exudation of the blood-serum: In a case of fifteen days' duration recorded by Fernet,[95] in which the nerve-trunk was greatly swollen, "the *nerve* was of reddish hue and the *vessels* of the neurilemma were injected." Though the nerve, examined microscopically by Déjerine and Raymond, showed no structural alteration, Fernet held that a neuritis was present. The advanced stage is shown in Ramsay Hunt's own typical case, in which the various parts of the nerve showed concomitantly the various stages of the process: a translucent, jelly-like substance in the epineurium and marked thickening and sclerosis of the larger vessels of the epineurium and perineurium and the smaller vessels of the endoneurium, with "small extravasations of blood" in "the loose areolar tissue of the sheath at all levels examined." The "thigh and leg had undergone a slight but visible atrophy " there were also twitchings and paræsthesia.

This exemplifies the morbid process in trigeminal neuralgia, since similar lesions were found in the Gasserian ganglia removed by Keen, and studied histologically by Spiller. The first-named author states that "the medullary substance of the nerve-fiber within the ganglion"

[90] Herter: Dercum's "T. B. on Nervous Dis.," p. 849, 1895.
[91] J. Ramsay Hunt: Amer. Medicine, Apr. 15, 1905.
[92] Cotugno: "A Treatise on Sciatica," London, 1775.
[93] Martinet: Thèse de Paris, 1818.
[94] Gendrin: "Histoire anat. des inflammations," Paris, 1826.
[95] Fernet: Arch. gén. de méd., 7ième. série, vol. i, p. 383. 1875.

was "immensely swollen, atrophied or entirely gone;" and that the vessels were "distinctly sclerotic," etc.—the sequence of events observed in all cases of "neuralgia" so-called, in which the morbid process has progressed sufficiently far.

NEURITIS OF THE HEAD, NECK AND TRUNK.—Besides the most common of all neuralgias, the trigeminal, already described, there is a variety due essentially to eye-strain, *i.e.*, the *ocular neuralgia,* usually limited to the eyeball. The pain may be quite severe, and radiate to the forehead over the frontal sinus. In *cervico-occipital neuralgia,* the pain starts behind the mastoid process, radiates posteriorly to the upper part of the neck and in front and above to the parietal eminence. The main nerves involved are the posterior branches of the upper four cervical. In *intercostal neuralgia* the pain starts from the spinal cord end of the intercostal nerves from the third to the ninth, and radiates round the chest; it is often very severe and is increased by motion, respiration and coughing. An eruption similar to that of herpes zoster occurs in many cases. *Pleurodynia* is, strictly speaking, neuralgia of the pleural nerves; it resembles the pain of acute pleurisy, and is very severe, especially during expansion of the chest. *Gastrodynia, enteralgia, hepatalgia, nephralgia, mastodynia, coccygodynia,* etc., are defined by their names.

In all these forms, with the exception of the cases of intercostal neuralgia in which the herpetic eruption occurs, the neural hyperæmia rarely exceeds the stage of plasmatic engorgement, the pain, as stated, being due to pressure upon the nervi nervorum.

HERPES ZOSTER (*Shingles, zona*), the form commonly witnessed, is but an intercostal neuralgia in which the inflammatory process, which often includes the ganglion of the affected nerve, has advanced beyond the preliminary plasmatic hyperæmia. The typical eruption—a reddish patch which soon develops into a crop of œdematous vesicles—may occur in any part of the body, wherever "neuralgia" has been observed. Dull pain in the affected region usually precedes by a few days the eruption and the acute pain, which develops suddenly and is extremely sharp, burning, and lancinating, in adults. The site of the eruption becomes markedly hyperæsthetic, the contact of the clothes being unbearable. It is nearly always unilateral, though it frequently reaches beyond the middle line of the body

in the "intercostal" form, and lasts from ten days to two weeks. The lesion may be hæmorrhagic, ulcerative, gangrenous, etc., and may be followed by persistent local neuralgia, anhydrosis or hyperhydrosis, muscular atrophy, etc. In some cases there is considerable fever, showing a tendency to intermittence, and especially marked towards evening.

The kinship of this disorder with the ordinary neuralgia is well shown by the large proportion of cases of trigeminal zona in Greenough's[96] and Cantrell's[97] statistics. Any part of the body in which innervation is impaired and where neuralgia is known to occur, may become the seat of herpes zoster. Hence the so-called "trigeminal," "femoral," "brachial," "ophthalmic," etc., forms which constitute the "regional" zosters. Guermonprez and Platel[98] observed it repeatedly on the fingers. The pathology of zoster also identifies it as a pure neuritis. W. F. Robinson[99] states that "Daniellsen found the intercostal nerve reddened and thickened and the neurilemma markedly infiltrated," and that the more recent investigations have shown that "the ganglion is not alone the part that is first attacked, but that the inflammation may arise at any point in the continuity of the nerve-trunk in its peripheral termination in the spinal cord or within the brain." Robinson's own investigations showed the presence of "a perineuritis of the cutaneous nerves exhibiting a small-celled infiltration of the neurilemma"—a condition witnessed early in sciatica.

NEURITIS OF THE EXTREMITIES.—The most important of the series is sciatica, reviewed above. *Cervico-brachial,* and particularly *brachial neuralgia,* are counterparts of sciatica. Jointly, they include the area supplied by the brachial plexus, *i.e.,* the four lower cervical nerves and the greater part of the first dorsal. The radial and ulnar are the nerves most frequently affected, the main tender points being at the elbow over the ulnar, and at the wrist over the inferior ulnar. The axilla and the shoulder are also the seat of tender points when the upper segments are involved. There is usually a dull, continuous, toothache-like pain along the entire course of the nerve or nerves affected, but with violent exacerbations or paroxysms of a stabbing, lacinating character. Temporary loss of power is often observed, and muscular atrophy may follow severe cases. As in sciatica, the skin may become glossy and thin, and is somewhat œdematous in most instances. As in sciatica also, there is inflammation of neural structures.

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[96] Greenough: N. Y. Med. Jour., Oct. 19, 1889.
[97] Cantrell: Phila. Med. Jour., Mar. 26, 1898.
[98] Guermonprez and Platel: Jour. des mal. cutan. et syph., vol. xi, p. 721, 1899.
[99] W. F. Robinson: Sajous's "Analyt. Cyclo. of Pract. Med.," vol. iii, p. 462, 1899.

Femoral, or *crural, obturator, plantar, metatarsal* and other forms of neuralgia are described, all of which differ as regards the area involved, but the pathology of which (that of a more or less advanced neuritis) is always the same, *i.e.,* that of an inflammatory process.

Musculospiral neuritis, owing to the superficial position of the musculospiral nerve, is not infrequently observed. The pain —though the disorder is not termed a "neuralgia" in text-books —is very severe, and the local tenderness is equally marked. This applies likewise to *median neuritis,* the intense pain radiating to the first two fingers and the thumb. Precisely as a flexure of the thigh in sciatica provokes severe pain throughout the entire nerve, so does a stroke on the end of one of the painful fingers cause a flash of pain up the whole length of the median. *Ulnar neuritis, brachial neuritis, circumflex neuritis,* and the many other varieties of *true* neuritis likewise provoke pain and other symptoms and trophic lesions common to the various forms of neuralgia reviewed.

General Pathology.—In all forms of true neuritis, the congestion may originate from two directions: (1) from the arterioles that enter the nerve from the side by piercing the perineurium, and whose capillaries form networks in the endoneurium to *nourish* the nerve; (2) from the axis-cylinders themselves, whose neuro-fibrils, as I have pointed out, are channels for plasma laden with adrenoxidase, which, by reacting with the phosphorus-laden constituents of the myelin, gives rise to the nerve-energy, *i.e.,* the impulse along the whole nerve, including its terminals.*

Any inflammatory lesion of the nerve-fiber itself, *i.e., parenchymatous neuritis,* must entail, therefore, phenomena ascribable to impairment of the nerve's conductivity: muscular atrophy, paræsthesias, cutaneous lesions, etc. Identical disorders may be produced, however, by pressure of the connective tissue forming partitions around nerve-bundles when these partitions are the seat of congestion, *i.e.,* perineuritis, but only when this is marked, the pressure being caused by the engorged arterioles or their capillaries and the serous exudation derived from the latter. This pressure is also, we have seen, the source of pain, the

* *Author's conclusion.*

nervi nervorum being compressed, while the infiltration accounts for the œdema. When the local lesions assume a morbid trend or the nerve is beyond repair from the start through local injury, the granules of adrenoxidase in the axis-cylinders remain unused and accumulate,* and the myelin sheath breaks up into droplets. Finally, these are absorbed and the field is invaded by lymphocytes, and finally occupied by their product, *i.e.,* newly formed connective tissue—the terminal sclerosis.

This normally leads to the conclusion that *true* neuritis is primarily a vascular disorder of the perineural and interstitial connective-tissue framework of the nerve, and that the nutritional disorders of the muscles, skin, etc., are due to pressure by engorged vessels, serous effusion, etc., upon, or destruction of, the nerve-fiber bundles (axis-cylinders) which these inflamed structures surround.

This does not mean that the cell-bodies in the central ganglia, from which the compressed axis-cylinders arise, may not themselves become involved in the morbid process. When they do, however, it is through an ascending neuritis, the destructive inflammation extending towards the cerebrospinal axis and gradually destroying the ganglion itself.

That the axis-cylinders of a nerve receive a part of their blood through their cell-bodies in the organ from which they originate,* was shown in a preceding chapter. The importance of this fact finds its practical application in the present connection. It furnishes a sound foundation for the—now established—clinical difference between the neuritis of individual nerves and multiple neuritis.* This is clearly defined by McPhedran[100] in the following lines: "In isolated neuritis the disease begins in the nerve-sheath, constituting a 'perineuritis,' the inflammation extending to the nerve-fibers afterward. In the multiple forms the nerve-fibers themselves are the seat of the primary change, the sheath becoming affected later." The latter, as will be shown in the next heading, I do not regard as a form of "neuritis." The fact that a neuritis steadily ascends from the periphery has been noted by several investigators. Thus, Sydney Schwab,[101] after a study of 20 recorded instances, including two of his own in which the Gasserian ganglion had been examined microscopically after extirpation, concluded that disease of the nerve-cells did not exist as a primary parenchymatous affection, and that in trigeminal neuralgia two classes of phenomena could be distinguished, namely: (1) an ascending neuritis *beginning at the periphery* and having a tendency to ascend to the ganglion; (2) an interstitial inflammation, chronic and progressive, of the ganglion itself. Moreover, Rose[102] found the lesions more marked at the peripheral ends of diseased nerves than at their central ends. All these observations have been confirmed repeatedly.

* *Author's conclusion.*
[100] McPhedran: Med. News, Oct. 31, 1896.
[101] Sydney Schwab: Annals of Surg., June, 1901.
[102] Rose: *Loc. cit.*