nerve irritability is increased and the size of negative variation is increased. The same result is obtained if the nerve is tetanized; so Waller concludes that carbonic anhydride is produced during tetanization. As many other factors affect the negative variation in the same way, we cannot conclude from this observation that the conduction of the nerve impulse is accompanied by a metabolic change, leading to carbonic-anhydride formation.

The Action of Anæsthetics.—The anæsthetics, chloroform, ether, carbon dioxide, and alcohol all temporarily annihilate nerve conduction, although some observers state that a preliminary rise in excitability is their first effect. If not exposed too long to the action of the anæsthetic the nerve will recover; but if too large an amount is used, or if the exposure is too long, irritability and conductivity appear to be permanently lost. Chloroform is much more active than ether and the nerve recovers from it with much greater slowness. This may be due to its being less volatile than ether and hence escaping less readily from the nerve or to its having a more powerful action. The most probable explanation of the action of the anæsthetics is that they dissolve the lecithoproteids or colloids of the nerve. Mayer[35] and Overton[36] have pointed out the parallelism of the anæsthetic action to the fat-dissolving powers of the anæsthetics. The nerve is particularly rich in lecithin compounds, and it is not improbable that the anæsthetics act upon them. The dissolving action of these substances may be easily seen in blood corpuscles, the eggs of many marine forms and other organisms, so that it is probable that they act on nerve protoplasm in the same manner. There is, hence, nothing peculiar about the action of the anæsthetics. They produce the same kind of a change in protoplasm as do positive ions, the positive electrode, or warmth. They put the nerve in a condition of anelectrotonus. They are particularly valuable because they are so soluble in protoplasm, so volatile, and effective in such small amounts.

General Summary.—We are now in a position to see how far the foregoing facts enable us to understand the processes in the nerve which are represented in the nerve impulse. There have been several hypotheses thus far proposed to explain these phenomena. One of the earliest was that of Du Bois-Reymond. In this theory the nerve substance is supposed to be composed of bipolar electrical particles negative at each end and positive in the middle. The current of rest is obtained by connecting the middle or positive surface with the cut end or negative surface. As each portion of a magnet shows the polarity of the whole magnet, so each portion of a nerve shows the polarity of the whole nerve. The nerve impulse is simply a turning of these particles on their axes, so that the negative ends turn toward the surface. This will explain the action current.

Hermann believed that these particles did not pre-exist, but that the current of injury was due to catabolic changes taking place at the cut surface. This became negative to the rest in consequence of these chemical changes. A similar change occurred during conduction, and this change in each part of the nerve caused the part just following it to be put in a position of catelectrotonus. On this theory the negative variation stimulated each part of the nerve in turn and was itself regenerated by the change which it brought about.

Becquerel supposed that there were numerous electrocapillary couples in the nerve which gave rise to electric currents, each couple, consisting of two different liquids, being separated by a capillary opening or by an organic membrane. D'Arsonval, who has developed this theory, supposed the electrical phenomena to be due to modification of the surface of separation of the two liquids similar to the electrical phenomena shown by the capillary electrometer.

Loeb has suggested that conduction is due to a change in state of the colloids, but has furnished no evidence in support of this view. The author believes that the facts indicate the truth of this hypothesis and suggests the following more specific theory:

The protoplasm of the nerve is essentially a colloidal solution. The colloidal particles are proteid in nature and in all likelihood are lecithin proteids resembling the sheaths of the red blood corpuscles, as is indicated by the especial richness of the nerves in lecithin. These particles are of different sizes and are electropositive. They continually change their state of aggregation, being easily precipitated or brought into solution and easily coalescing with their neighbors or breaking up into a large number of smaller particles. Through these changes the surface separating each particle from the surrounding fluid augments or diminishes. When two particles coalesce the total surface is reduced; when one particle separates into two the total surface of separation is increased. Around each particle there are induced in the water electrical changes of an opposite sign. It will be seen that any change in the surface of separation must necessarily produce an electrical disturbance exactly in the same manner as do the movements of the capillary electrometer, and in this respect my suggestion harmonizes entirely with that of D'Arsonval.

Stimulation, whatever its character, whether mechanical, chemical, thermal, or electrical, brings about a change in the state of division of these colloidal particles. It produces either one of two effects, *i.e.*, a coalescence of the particles (gelation), or an increase in number of the particles (solution). According as a stimulus produces one or the other of these effects we say that it excites the nerve or anæsthetizes it. It may fairly be questioned which effect is the excitation and which the anæsthetization. This question may be answered, I believe, by the exciting action of drying the nerve and of applying cold. Both of these processes excite or generate nerve impulses. Since they can hardly be supposed to increase the solubility of the colloids, we may confidently assume that they congeal or precipitate the colloids, and hence that excitation is due to a diminution in the number of colloidal particles and a reduction in their total surfaces; and conversely, anæsthetization or inhibition is due to the reverse process. All the exciting agencies may be interpreted in this way. Thus mechanical shock which disturbs the hydrosol brings about such a condition of temporary coagulation or rigidity of the nerve protoplasm throwing the particles together. This interpretation is strengthened by Mrs. Andrews' observations on the effect of shock on the choano-flagellates, where the rigidity of the previously fluid protoplasm can be easily demonstrated, and by my own observations on other forms of protoplasm, notably eggs. Cold, as will be seen, diminishes the stability of the protoplasmic solution or hydrosol, while warmth increases it; negative ions precipitate positive colloidal solutions and they excite the nerve; excitation takes place at the cathode or negative electrode, where positive colloidal particles will be precipitated; the extraction of water acts in the same manner as cold. In fact all the phenomena of excitation are readily understood on this hypothesis. Similarly the action of all anæsthetizing agents becomes clear. Positive colloidal solutions are rendered more permanent by positive ions, and these annihilate nerve excitability; warmth of moderate amount increases the stability of nearly all solutions, and this diminishes excitability; ether and the anæsthetics dissolve the protoplasm of eggs and other cells and destroy irritability; the anode, which holds positive colloids in solution, abolishes excitability. We may sum up our conclusions in the general law that nerve excitability varies inversely with the stability of the protoplasmic hydrosol. The less stable the hydrosol, the more irritable the nerve. Irritability will be lost when the nerve is stable, either in the condition of solution or in that of total gelation. The rise in irritability at the anode on opening the current is due to the fact that, as already explained, by the action of the current the particles are greatly divided; and after the current is broken the diffusion outward of the positive ions reduces the stability of the hydrosol here and it returns back toward the normal. The electrotonic effects are due to the solution being made more stable near the anode and less stable near the cathode.

As readily as the facts of excitation are understood on this hypothesis, so many of the facts of the electrical phenomena of nerves may be explained. The electrical disturbances are the result of the alterations in the surface of separation of particles and liquid. Whenever these particles coalesce, a portion of the negative charges, formerly induced about each particle, are set free. The portion of the nerve where this is occurring becomes temporarily electronegative to the rest of the nerve. Thus the current of injury is due to the coalescence of particles at the injured end. This is always negative to the uninjured part.

If this is true, the exposure of the end of the nerve to acids or anæsthetics should diminish the current of injury, whereas alkalies should increase it. Such I have found to be the case. The current of action is the result of the progressive precipitation of the colloids and a progressive setting free of negative charges. It is, however, impossible within the limits of this article to discuss the bearing of this hypothesis on all the numerous electrical phenomena of nerves. It may be stated, however, that a warmed or etherized portion of a nerve is electropositive; a cooled portion electronegative to the normal nerve.

The conduction of the nerve impulse may be understood on this hypothesis as follows: Each precipitation of colloidal particles sets free by the accompanying reduction in surface negative charges formerly induced in the water about each particle; these charges at once precipitate the next layer of particles, and so on. Thus the negative variation successively stimulates each following segment of the nerve, as Hermann supposed, and it is regenerated by the change which it itself has produced. The sheath and peculiar structure of the nerve probably, as Boruttau supposes, plays an important part in the electrical phenomena of polarization and stimulation, and possibly in determining the speed of transmission, but the change in the protoplasm itself is the most important factor in conduction. Finally, it should in all fairness be stated that among the difficulties or exceptions to this hypothesis are the statements that the anæsthetics bring about a preliminary rise in irritability, and that conductivity and excitability may vary somewhat independently of each other. Whether these facts can be harmonized with the explanation already offered remains for the present unknown.

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NERVES, PATHOLOGICAL CHANGES IN. See *Neurone, etc.*

NERVE TISSUE, HISTOLOGY OF.—According to the fundamental conception of neurology the entire nervous system, central as well as peripheral, has been regarded as composed of morphological units, the neurones, held together by the supporting tissues, the neuroglia. The term neurone was suggested by Waldeyer in 1891, and was accorded almost universally an international acceptance by anatomists, physiologists, pathologists, and clinicians. The term neura, proposed by Rauber, and neurodendron by Kölliker, to designate the same unit, have not met with similar favor. More recent investigations, however (Apàthy, Betha), have thrown some doubt upon the neurone doctrine as formerly held. The neurone consists of a cell body, dendritic processes, and an axis-cylinder process (axone with its terminal ramifications). As the neurone does not consist only of the cell body, but also has processes, some of which are of extreme length, it is impossible to see the entire neurone in the majority of cases. As a matter of convenience, therefore, the description of the neurone may fall under two headings—the nerve cells or nerve-cell bodies, and the nerve fibres.

THE NERVE CELL.—The essential part of a neurone originating the nerve impulse is the cell body. Nerve cells or ganglion cells, as they are generally called, occur in groups known as ganglia in the cerebrospinal system, the sympathetic system, and in the organs of special sense. While variable in size, they are among the largest cells in the body, often, as in some of the ganglion cells in the anterior horns of the spinal cord, reaching a size of from 90 to 135 μ, the cells of Betz in the paracentral lobule being especially large. Many nerve cells, however, are much smaller in size, the cells of the granular layer of the cerebellum being only from 4 to 8 μ in diameter.

Study of the morphology of the neurones requires the consideration of their external peculiarities as well as of their internal architecture. The former are best revealed by the methods of Golgi and Ehrlich, and the latter by the methods of Nissl and Held.

Morphology of the Nerve Cells.—Nerve cells vary greatly in shape. Starting originally as spherical cells, some may retain this shape as in the spinal, Gasserian, or other ganglia; others may become ellipsoidal, as in the spinal cord, pyriform as the cells of Purkinje in the cerebellum, pyramidal as the cells in the gray matter of the cerebrum, or stellated as the multipolar ganglion cells of the spinal cord. The most conspicuous peculiarity of the nerve cells is the branching. This may take place only on one side leading to a prolongation of the protoplasm into a single pole, such cells being known as unipolar nerve cells; when the protoplasm is prolonged into two, usually opposite, poles, the cells are appropriately designated as bipolar; when the protoplasm extends in several directions multipolar cells are formed. Each polar prolongation is continued to form a nerve-cell process. Of such processes two kinds are recognized, the branched *protoplasmic processes* and the *axis-cylinder process.*

The branched protoplasmic processes, now usually called the *dendrites*, form prolongations of the protoplasm from the cell body, hence the old name of protoplasmic process. They are always broader and thicker at their origin, becoming gradually narrower as they divide, splitting up and subdividing in an antler-like fashion until a rich twig-work or arborization results. The group of terminal end-branches of the dendrites is known as the *telodendrion.* The character of the dendrites, which result from the branching of the protoplasmic processes, varies much in different parts of the central nervous system. In some cells the branching commences a short distance from the origin of the process, while in other cells the process continues for some distance from the cell body before undergoing division, and then suddenly breaks up into a large number of dendritic branches.

The cerebellar cells of Purkinje are instances of the former type, the apical dendrites of the pyramidal cells

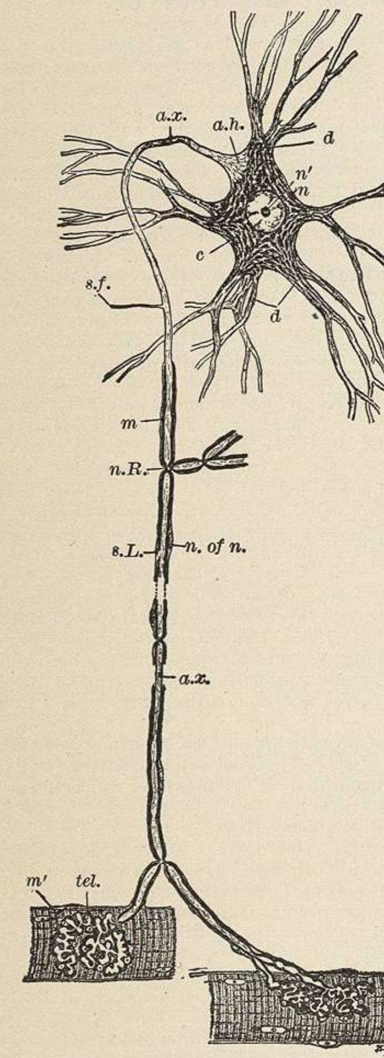

FIG. 3572.—Schematic Representation of a Lower Motor Neurone from the Ventral Horn of the Spinal Cord, together with all its protoplasmic processes and their divisions. The axis-cylinder process with its divisions, side fibrils, or collaterals, and the end ramifications (telodendrions or motor end-plates) in the muscle, represent parts of a single cell or neurone. *a.h.*, Axone hillock devoid of Nissl bodies, and showing a tendency to fibrillation; *a.x.*, axis cylinder or axone, also indistinctly fibrillated. This process, at a short distance from the cell body, becomes surrounded by a myelin sheath, *m*, and a cellular sheath, the neurilemma, the latter not being an integral part of the neurone; *c*, cytoplasm showing the dark-colored Nissl bodies, separated from one another by the lighter ground substance; *d*, protoplasmic processes (dendrites) containing Nissl bodies; *n*, nucleus; *n'*, nucleolus; *n.R.*, nodes of Ranvier; *s.f.*, side fibril; *n. of n*, nucleus of neurilemma; *tel.*, motor end-plate or telodendrion; *m'*, striped muscle fibre; *s.L.*, segmentation of Schmidt-Lantermann. (From "The Nervous System and Its Constituent Neurones," by Lewellys F. Barker. D. Appleton & Co., New York, 1899.)

of the cerebral cortex of the latter. The extent and complexity of arborization is also variable, being comparatively simple and with little branching in some cells,

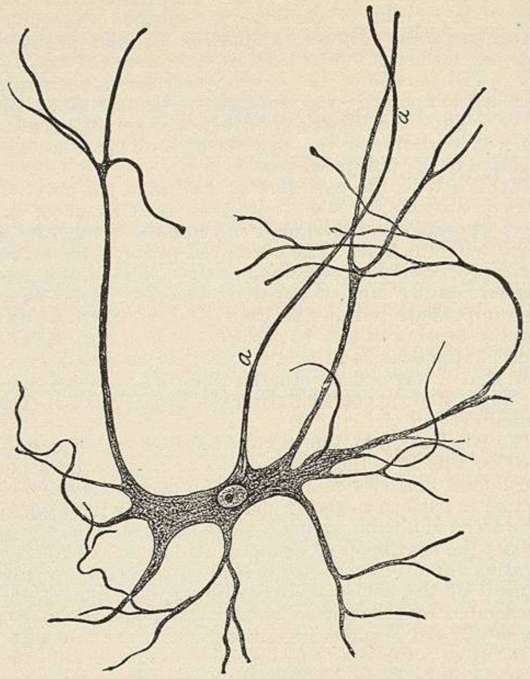

FIG. 3573.—Multipolar Ganglion Cell from the Anterior Horns of the Spinal Cord, Isolated by Maceration and Teasing, showing that the numerous branched protopasmic processes are somewhat displaced and distorted, owing to manipulation. *a*, Axis-cylinder process; cytoplasm granular; nucleus large, distinct; nucleolus darker than nucleus. (Piersol.)

while in others a complete arborization exists, forming a dense forest which extends over a wide territory. Besides the degree of complexity of arborization the relation of the dendrites to the surface of the cells is of interest, since this branching may arise from only one or two dendritic processes as in the cells of the hippocampus, or it may originate from all sides of the cell like

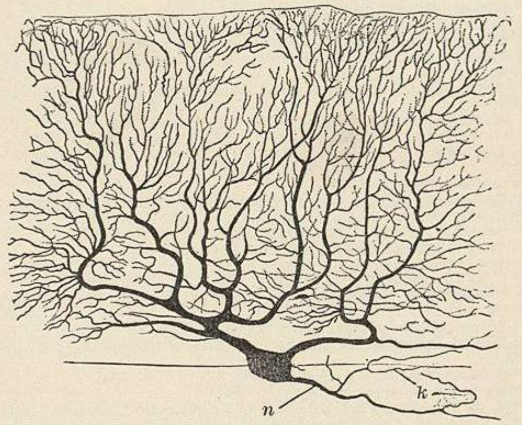

FIG. 3574.—Cell of Purkinje from the Cerebellum of Man. Showing pyriform cell body, large arborescent protoplasmic process with gemmules forming the typical telodendria of the dendrite. *n*, Axis-cylinder process; *k*, collateral fibrils. (Kölliker.)

a radiation as in the ventral horns of the spinal cord. Rarely neurones are characterized by entire absence of dendrites; such *adendritic* elements have been observed in the nervous system of invertebrates and also in the

spinal ganglia of man. The contours of many lateral dendrites exhibit the presence of small buds known under the name of *gemmules. The axis-cylinder process, neu-*

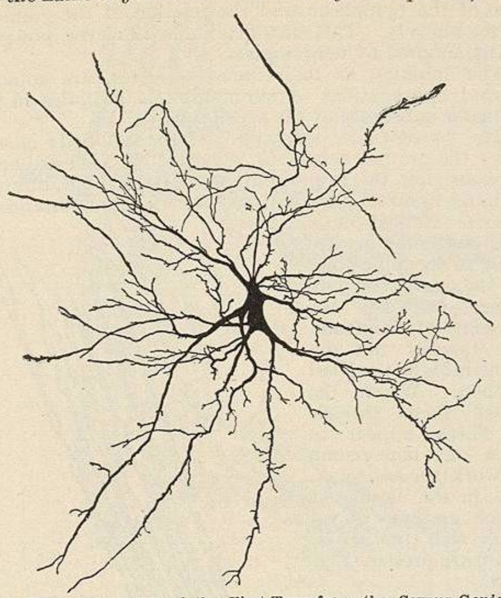

FIG. 3575.—Golgi's Cell of the First Type from the Corpus Geniculatum of a Cat. Showing numerous richly branched dendrites, and the very fine axone with its collateral branches. (Kölliker.)

rite or *axone*, unlike the dendrite, is thin, slender, inconspicuous, straighter in its course and smooth in outline. It was formerly described as an unbranching single process, and was supposed to be always the continuation of the axis cylinder of a nerve fibre. Golgi's investiga-

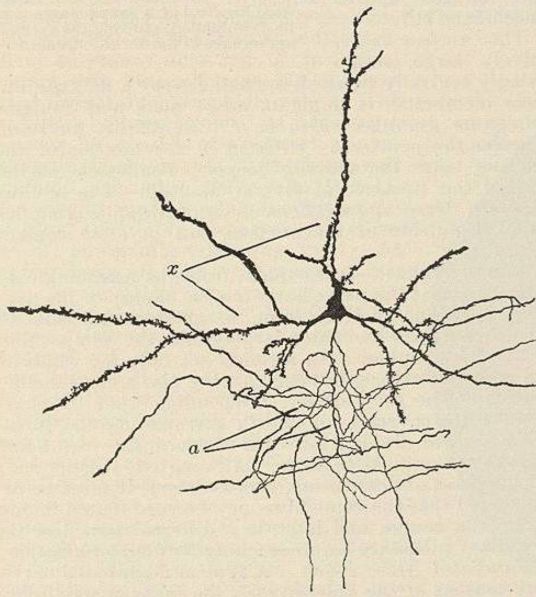

FIG. 3576.—Golgi's Cell of the Second Type from the Cerebrum of a Cat. Showing *x*, the coarse protoplasmic processes easily distinguishable from the more delicate axis-cylinder process *a*, forming the rich telodendrion of the axone. (Kölliker.)

tions have shown, on the contrary, the existence of nerve cells in which the axone is branched and does not become the axis cylinder of a nerve fibre. Hence nerve cells are arranged into two types—cells of the *first type*, in which the single non-branching axone becomes the axis cylinder of a medullated nerve fibre, and those of the *second type* in which the axone does not become the axis-cylinder process of a nerve fibre but undergoes branching, forming a telodendrion of the axone to which the name of

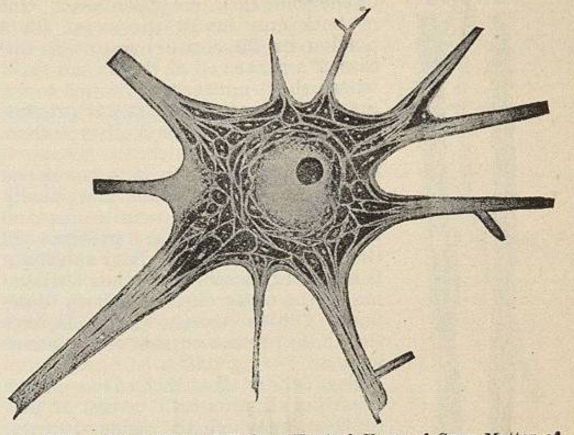

FIG. 3577.—Motor Nerve Cell from Ventral Horn of Gray Matter of Spinal Cord of Rabbit. Of the three lower processes, the middle one represents the axone; all the other processes are dendrites. The margin of the cells and of the masses of stainable substance appear too sharp in the reproduction. At the angle of division of the large dendrite at the left superior angle of the cell is shown one of the wedges of division. The spindle-shaped Nissl bodies are well shown, especially in the dendrites. (From "The Nervous System and Its Constituent Neurones," by Lewellys F. Barker. D. Appleton & Co., New York, 1899.)

dendraxone or neuropodion (Kölliker) is applied. The termination of the dendraxone usually takes place by exhaustion of repeated division. Very rarely and only in exceptional cases the terminal branches are interwoven to form a basket-like meshwork surrounding the cell body of a second neurone. Axones vary greatly in length, being very short, often only a few millimetres long; in nerve cells of the second type the dendraxone never leaves the gray substance. In nerve cells of the first type, on the contrary, the monaxone may be exceedingly long, some extending, as spinal nerve fibres, fully half the length of the body. Monaxones are frequently provided with collateral branches or *paraxones.* These collateral branches should not be mistaken either for the arborization which takes place in the dendraxone or for the true division of the axone into two branches, forming a right or an obtuse angle resulting in the T- or Y-shaped branches described by Ranvier.

In addition to the monaxone neurones, *diaxone* as well as *polyaxone* neurones have been observed. Ramòn y Cajal describes also *anaxone* neurones in the retina.

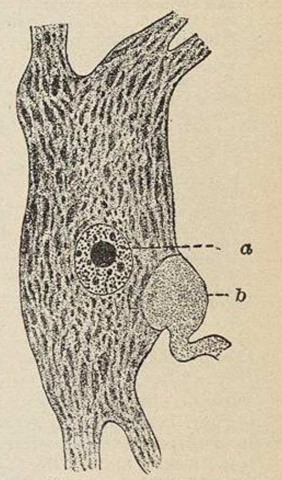

FIG. 3578.—Nerve Cell from the Anterior Horn of the Spinal Cord of an Ox. Showing coarse chromotophile flakes, nucleus (*a*), nucleolus, and the implantation cone or axone hillock (*b*), devoid of chromatophile granules. (Böhm-Davidoff.)

The mode of origin of the axone also claims attention. The axone may arise from the cell body directly or else from the dendrite; in this case the origin is usually near the cell body, while more rarely it is situated at some distance from the cell body. At its origin the axone is wedge-shaped and hence is called the *implantation cone.* It possesses certain characteristics in its internal structure and will be referred to later. The axone may have protecting coverings or a sheath. When no envelope is present it is customary to speak of *naked axones*. The coverings are the *neurilemma* or the *sheath of Schwann*, and the *medullary substance* or the *white substance of Schwann*. One or both may invest the axone for a portion or its entire length. In the case of a nerve cell of the second type, where the dendraxone is limited to the gray substance, no sheath is present. In nerve cells of the second type there are stretches in which the axone is naked, those in which it is enveloped only by the neurilemma, and finally tracts in which both neurilemma and medullary substance are present. In the latter case the medullary substance is the inner sheath, while the neurilemma is the outer one. At times ill-defined fibrous tissue, called Henle's sheath, is present outside of the neurilemma.

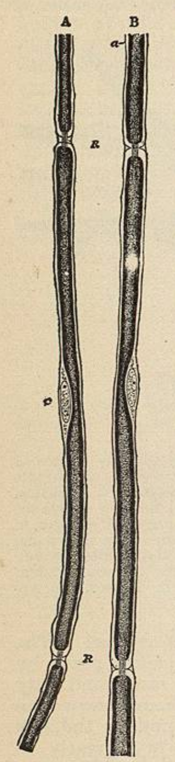

FIG. 3579.—Portions of Two Medullated Nerve Fibres Stained with Osmic Acid. (From a young rabbit.) × 425 diameters. *R*, *R*, Nodes of Ranvier, with axis cylinder passing through; *a*, primitive sheath of the nerve or neurilemma; *c*, opposite the middle of the segment, indicates the nucleus and protoplasm lying between the neurilemma and the medullary substance. In *A* the nodes are wider and the intersegmented substance is more apparent than in *B*. (Quain.)

Structure of the Nerve Cell.—Nerve cells like all other cells consist of protoplasm, to which the name of *neuroplasm* has been applied by Kölliker. They contain an attraction sphere, within which one or more centrosomes are situated; a nucleus, which is larger than in most other cells of the body; usually a single nucleolus, more rarely several nucleoli; an implantation cone; several nuclear caps, and sometimes several spindles. A variable amount of pigment granules is also present, depending upon the age of the cell. Not every nerve cell, however, contains the above-enumerated parts. In fresh preparations, without the use of reagents, the protoplasm appears more or less homogeneous. With the use of reagents and different methods of fixation the protoplasm varies in appearance, being fibrillar, granular, or vacuolated. The latter two conditions at least may be regarded as probably artefacts, produced by reagents. The granules have a special affinity for certain aniline stains. They appear very distinctly when stained by the Nissl method and are generally known as *Nissl bodies*, *chromatophile* or *tigroid* granules (Fig. 3577). The granules are variable in size, regular or irregular in shape, and are arranged in groups, rows, or irregularly; sometimes simulating rods of variable thickness and constituting the so-called "*stainable substance*" of Nissl. That portion of the protoplasm which has no affinity for stains is known as the "*unstainable substance*" of Nissl. The granules are more concentrated in the inner portion (or the *entoplasm*) of the cell, while in the outer portion (or the *ectoplasm*) they are more rod-shaped. The rod-shaped elements are present in the dendrite, but are not found in the axone. Nissl suggested an elaborate classification of nerve cells, depending upon the amount, the arrangement, and the proportion of the granular substance to that of the cytoplasm and the relation of the granules to the nucleus. This classification is, however, not generally adopted by neurologists.

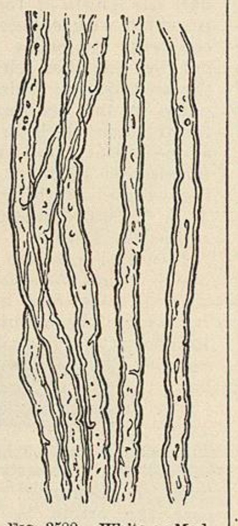
FIG. 3580.—White or Medullated Nerve Fibres (shortly after death), showing the sinuous outlines and double contours. (Quain.)

The spindles, as their name indicates, are spindle-shaped aggregations of chromatophile granules in the stainable substance of the nerve cells.

The "*unstainable substance*" of the cell body constitutes the ground substance, regarded by Nissl as homogeneous; but the investigations of Held (by a different staining method) have not only shown this substance to become stained of a deep red color in contrast to the blue color of the stainable substance, but also the presence of longitudinal threads not demonstrable by the Nissl method. Within the axone these threads or fibres appear to form a honeycomb network (the *axospongium*), in the meshes of which granules of variable size (the *neurosomes*) are present (Fig. 3578).

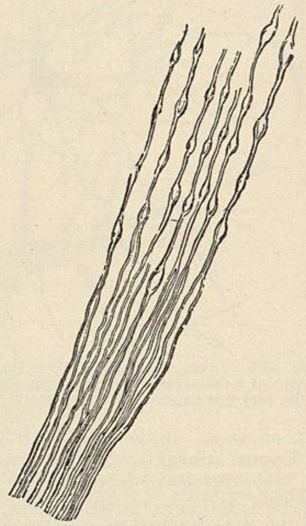
FIG. 3581. — Medullated Nerve Fibres from the Root of a Spinal Nerve near its Termination, showing the varicose appearance of the fibres. (Quain.)

The *implantation cone* or *axone hillock* is free from chromatophile granules, and stands out in marked contrast to the rest of the protoplasm of the cell body on account of the mottled appearance of the latter.

The *nucleus* is relatively large, round, usually centrally situated, surrounded by a delicate nuclear membrane, is single in adult man, and contains numerous granules which have little affinity for stain, whereas the neucleolus, situated in the interior of the nucleus, takes the staining deeply. Lenhossek has described the presence of several nucleoli. The nuclear caps are dense aggregations of chromatophile granules situated outside of the nucleus, but in close contact with it.

NERVE FIBRES.—It is evident from the foregoing consideration that the nerve fibres are the axones of the neurones. The nerve fibres form the chief constituents of all nerve trunks and enter largely into the composition of the cerebrospinal axis, forming not only the whole of the white substance, but constituting also a considerable portion of the gray matter. Depending upon the character of the coverings or sheath surrounding the fibres, the latter are divided into two varieties, the *medullated* and the *non-medullated* fibres. Although this distinction, for purposes of description, is convenient, it must be remembered that the same fibre may be medullated in one part of its course, and later, in a different part, lose its medullary substance before reaching its final termination.

Medullated Nerve Fibres.—A typical medullated nerve fibre consists of the *axis cylinder*, the inner or axial portion of the fibre, the *medullary substance*, or the white substance of Schwann, surrounding the axis cylinder, the *neurilemma* or *sheath of Schwann*, the outer covering surrounding the medullary substance, and the nerve corpuscles or nuclei. The axis cylinder is the most important part of the nerve fibre, conveying as it does the nerve impulse and constituting the only part which is never absent in the nerve fibre. The axis cylinder originates in the cell body of the neurone as its axone and terminates in the tissue to be controlled by that element.

It appears as a thread, running through the centre of the fibre, but consists of a bundle of very delicate nerve fibrillæ, called the ultimate nerve fibrillæ, held together by a homogeneous cement substance and surrounded by a delicate and closely adherent membrane, the *axilemma*.

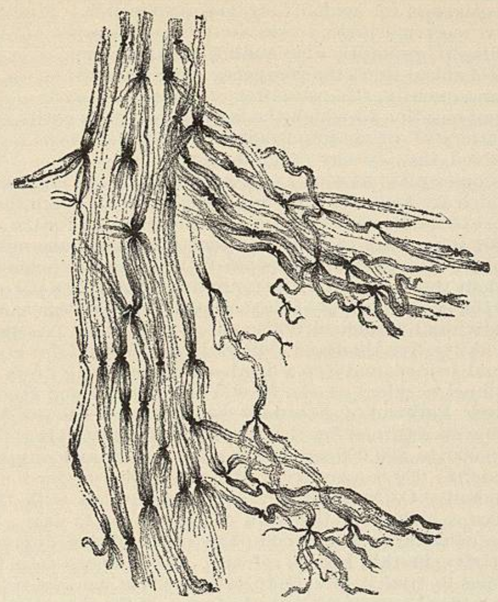
FIG. 3582.—Small Branch of a Muscular Nerve of the Frog, Near its Termination. Showing the well-marked nodes of Ranvier, the axis cylinder, and the division of the fibres at the nodes. (Kölliker.)

Surrounding the axilemma is the medullary substance, much thicker than the axilemma, of soft fatty consistency, and acting as a protecting medium to the delicate axis cylinder. In the fresh state the medullary substance does not lie in actual contact with the axilemma, but is separated from it by a lymphatic space. The medullary substance itself is not homogeneous, but consists of a network of neurokeratin, in the meshes of which the soft semifluid substance, the myelin, is held. The myelin is of an albumino-fatty composition, containing protagon, and capable of powerfully refracting light. At regular intervals, along the course of the fibre, symmetrical constrictions of the medullary substance occur, known as the *nodes of Ranvier*. These nodes are constrictions of the neurilemma and complete interruptions of the continuity of the medullary substance, but not affecting the axis cylinder, which at these points is in contact with the neurilemma. That portion of the nerve fibre which is situated between two adjacent nodes is known as the *internode*. In fine nerve fibres the internodes are shorter than in those of greater diameter, and in fibres of the same thickness they are shorter in warm-blooded than in cold-blooded animals. Near the termination of the fibre the internodes are also shorter. At the constrictions the axis cylinder is accessible to various reagents which cannot reach it at other points, as they cannot penetrate the medullary substance. Actual breaks in the medullary substance are artificial markings—the Schmidt-Lantermann segments as they are called,—resulting from the use of reagents. These interruptions may be distinguished from the true nodes by their irregular character, their asymmetry, and by the fact that no constrictions of the neurilemma take place in these locations. Nerve fibres are not uniform in diameter, but vary greatly; according to Kölliker, the finest fibres measure from 2 to 4 μ, those of medium size from 4 to 9 μ, while the largest possess a diameter from 9 to 20 μ. *The neurilemma* or *sheath of Schwann* is the outermost covering of the nerve fibre, and consists of a structureless or hyaline membrane, surrounding the medullary substance. Oval *nerve nuclei*, or nerve corpuscles, lie just beneath the neurilemma in depressions on the outer surface of the medullary substance. Only one such corpuscle is present in each internode and is usually placed in the middle of the internode. The medullated nerve fibres in the central nervous system have *no* neurilemma, nodes of Ranvier, or corpuscles. In the fresh state the medullated nerve fibre has a glistening, homogeneous appearance. After death the fibre appears to have a double contour, but later becomes mottled, as the result of rapid disintegration. Osmic acid stains the medullary substance black.

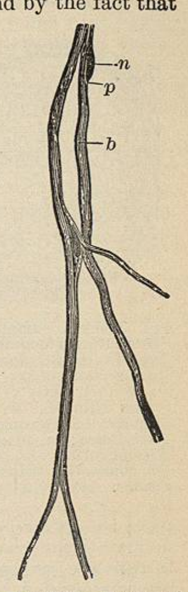

FIG. 3583.—Portion of the Network of the Fibres of Remak from the Pneumogastric of a Dog. Showing *n*, nucleus; *p*, protoplasm surrounding it and the faint striation caused by the fibrils. (Quain.)

Non-Medullated Nerve Fibres.—The *non-medullated* nerve fibres or the *fibres of Remak* are nothing more than axones or axis-cylinder processes of neurones devoid of medullary substance and neurilemma. The latter, however, is replaced by a delicate sheath, beneath which the small nerve nuclei are located. The nuclei are more numerous in these than they are in

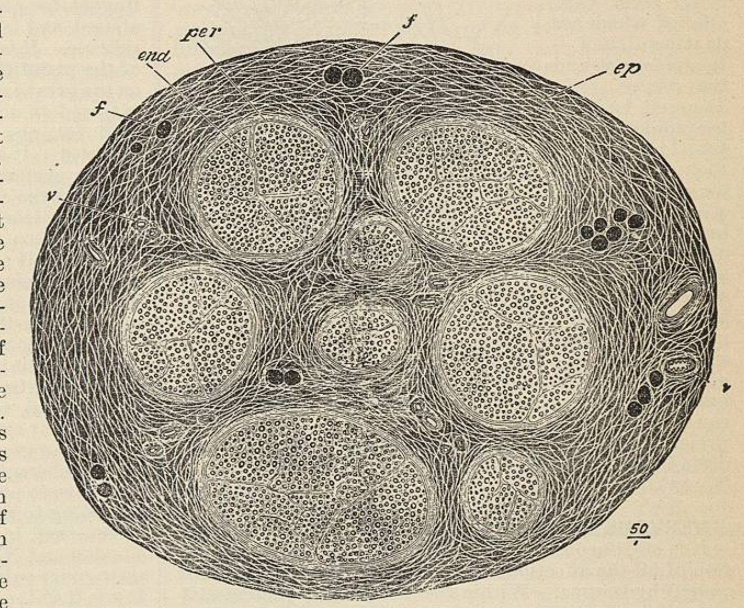

FIG. 3584.—Transverse Section of the Internal Saphenous Nerve of Man, made after being stained in osmic acid and subsequently hardened in alcohol. Drawn as seen under very low magnifying power. Showing *ep*, epineurium or the general sheath of the nerve, consisting of connective-tissue bundles of variable size separated by cleft-like areolæ, which appear as a network of clear lines with here and there fat cells, *f*, and blood-vessels, *v*; *per*, perineurium, the lamellated connective tissue, enclosing the funiculus; *end*, interior of the funiculus, showing the cut ends of the medullated nerve fibres which are embedded in the connective tissue within the funiculus (endoneurium). The fat cells and the nerve fibres are darkly stained, but the connective tissue of the nerve is only slightly stained. (Quain.)

the medullated nerve fibres. Non-medullated nerves often appear varicose and exhibit a marked tendency to branch and form plexuses. Their ultimate fibrillæ are more widely separated than are those of the medullated fibres, protoplasm in the former taking the place of the cement substance in the latter. As independent fibres they occur principally in the sympathetic system, but not infrequently they are associated with medullated fibres in nerve trunks, as in the vagus.

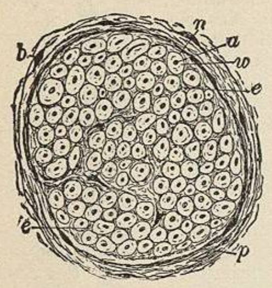

FIG. 3585.—A Simple Funiculus More Highly Magnified. The apparent small nucleated cells are sections of the nerve fibres and their axis cylinders. *a*, Axis cylinder; *w*, white substance of Schwann or medullary substance; *n*, neurilemma; *e*, endoneurium; *p*, perineurium; *b*, connective-tissue cells of the same. (Piersol.)

Nerve Trunks.—The nerve fibres are usually collected in bundles or funiculi, several of which constitute the nerve trunk. The individual fibres are held together by a delicate connective tissue, the *endoneurium*. A certain number of the fibres are grouped to form a funiculus, the latter being surrounded by a more dense connective-tissue envelope, the *perineurium*. The funiculi in turn are grouped together to form a nerve trunk, and are surrounded by a larger amount of loosely arranged connective tissue, the *epineurium*. This tissue supports the blood-vessels and the lymphatics, which invariably are present in the interior of the nerve trunk, as well as the adipose tissue often present in the larger nerve trunks.

The Neuroglia.—The supporting substance in the white matter of the brain and cord, as well as a considerable portion of the matrix of the gray substance, is made up of a network of exceedingly delicate fibres, the *neuroglia* fibres, and the neuroglia cells, two varieties of which are distinguished—the *spider cells* and the *mossy cells*. The cell body of the spider cell is smaller, while their processes are long, thin, rigid, with very little branching. They occur chiefly in the white substance of the brain and cord. The mossy cells have a larger cell body, short, richly branched processes, and are principally found in the gray substance, where they are often in intimate relation with the walls of blood-vessels.

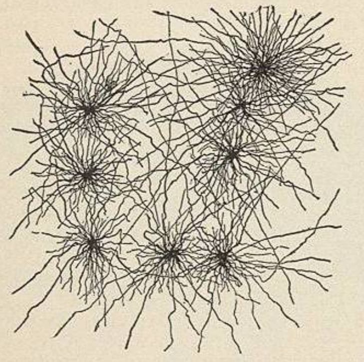
FIG. 3586.—A Group of Spider Cells from the White Substance of the Brain of Man, stained by Golgi's method. Drawn as seen under high magnifying power. (Kölliker.)

Robert Formad.

NERVOUS SYSTEM, TRAUMATIC AFFECTIONS OF.—It is not purposed here to attempt a detailed description of all the affections of the nervous system which are caused by trauma. Within the limits of the present article nothing further can be attempted than a brief analysis of the causal relations in which trauma stands to nervous diseases, with especial consideration of the place which nervous diseases, when caused in this manner, occupy at law. What is to be said, therefore, will be chiefly interesting to the medical man who is brought in contact with injuries to the nervous system and their legal complications. Personal-injury claims form a very important feature of modern life. Not only transportation companies, but private individuals as well, fully expect to pay for injuries which are received through actionable negligence for which they are responsible. Similarly, few receive injuries traceable to the negligence of others without promptly demanding compensation. In our mechanical times the frequency of accidents is enormous. Consequently, the evaluation of injuries received and the compensation to which the injured person is entitled are matters of prime importance. Greater interests are involved than in any other medico-legal question. This becomes plain as soon as we reflect upon the large sums which are annually paid out in such cases. From the report of the Brooklyn Rapid Transit Company for the year 1901 it appears that in that year more than one million dollars was paid for personal injuries and expenses incident thereto. This sum represented nearly ten per cent. of the gross receipts of the company for the year named. Individual verdicts are also often very high. As much as thirty-five thousand dollars has been paid for a personal injury, and for a death claim resulting from the Tunnel accident of the New York Central and Hudson River Railroad of February 8th, 1902, a verdict of $60,000 was returned by the jury. Verdicts varying from $10,000 to $20,000 are not at all unusual, and anything under $1,000 is considered virtually a victory for the defendant. Court calendars are overcrowded with these cases, which form the bulk of jury trials to-day. But the calendar is not a complete index of the degree of activity in this branch of law, as for every case that comes to trial it is safe to estimate that ten are settled by mutual agreement out of court. If the magnitude of the interests at stake are taken into consideration, it is not surprising that trial lawyers should be on the alert, or that there should be great competition for plaintiff's cases. As a result, "runners" or "ambulance chasers," representing legal firms which specialize in accident cases, are constantly stationed about centres of traffic; they rush to the scene of accident, and make their appearance at the hospital door almost simultaneously with the injured person. Thenceforth the claim is prosecuted on the contingent fee plan. The system has doubtless been much abused, and has been made the object of much attack and ridicule. It is made possible solely through the poverty of the plaintiff, who is generally unable himself to carry on the great expense of trial at law, and who consequently is forced to accept professional services which are to be paid for, on a percentage basis, out of the damages awarded. It has many very objectionable features. By such a system the lawyer is made more than an advocate, and the expert medical witness more than a mouthpiece of science. But no practical and better substitute has yet been suggested. The question will probably resolve itself eventually by fewer claims being litigated, and more being settled by mutual agreement. Mr. Herbert W. Page, whose book, "Injuries of the Spine," published in 1882, marked a distinct epoch in the history of this subject, told me a year or two ago that litigation of personal-injury claims in England was becoming more and more infrequent. Erichsen's book, which appeared in 1866, and which furnished the original description of the peculiar symptoms resulting from railway and allied injuries, gave the first effective impetus to litigation of this character. For years afterward personal-injury claims were prominent in the English courts. But now, according to the statement of Mr. Page, they are so infrequent that, in his position as consulting surgeon to the London and Northwestern Railway, he is called upon to go to court only three or four times a year. It has seemed to me that the willingness for compromise is growing in this country also. Among the litigated cases those hardest to compromise are the ones in which injury to the nervous system is alleged. In purely surgical injuries, such as the loss of a limb or of an eye, the cause is definite, and the question quickly resolves itself into one of liability and the appraisal of the value, as far as such an appraisal is possible, of the injured or missing member. But in nervous affections, and especially in the functional affections, with their obscure causation, their indefinite and often bizarre symptomatology, agreement is much more difficult. The contending parties are often at variance in regard to every particular. Neither is inclined to give in, and the case, if it is a case, goes to the jury.

Nervous diseases are divided into two great classes, organic and functional. A functional, as opposed to an organic disease, is one in which the anatomical integrity of nervous structure remains unimpaired. Functional diseases doubtless have a material pathology. But such a pathology remains inaccessible by any methods of investigation at present at our disposal, and we are therefore obliged to retain this classification, artificial as we know it to be. Of the organic nervous affections caused by trauma, the vast majority are definite surgical injuries to the central or peripheral nervous system. Thus, injuries of all kinds to the head, with injury of the brain; to the back, with injury to the spinal cord; or to the peripheral nerves, are causes. When, in addition to the cause, we can demonstrate certain cardinal abrogations of function of these organs, which we have learned to rely upon as indications of structural alterations in them, the diagnosis of organic injury is justifiable. Thus, after head injuries, paralysis of one or more cranial nerves, or of the extremities, together with other general symptoms, speaks for injury to the brain; paralysis, with anæsthesia in characteristic areas, and loss of control of the sphincters, speaks for injury to the spinal cord; paralysis, with degenerative electrical reactions, speaks for injury to a peripheral nerve. Injuries of this character are ordinarily easy to recognize, and the prognosis in regard to them can usually be formulated with considerable precision. Consequently, in common with other surgical injuries, when they are seen in court, which they rarely are, the questions for the jury to decide concern the legal aspects of the accident rather than its surgical results.

There is a group of chronic organic diseases, with uncertain and indefinite causation, which are not infrequently the subjects of litigation. The most important of these are locomotor ataxia and general paralysis of the insane, or general paresis. Others of this class are ataxic paraplegia, progressive muscular atrophy, paralysis agitans, syringomyelia, multiple sclerosis, etc. These latter are, however, much rarer diseases than the two first mentioned, and consequently of much less importance. Both locomotor ataxia and general paresis are comparatively common (the latter chiefly in cities). As has been said, their causes are obscure and undetermined. It is possible, and indeed probable, that injury can act as a contributing cause in their development. But the weight of scientific evidence is against their ever occurring solely as the result of trauma. Both diseases are often latent for a long time, and both may undergo a sudden outbreak of symptoms as the result of disease or injury. Both diseases, by their symptoms, expose the victims of them to accidents. It is consequently not surprising that both are frequently made the subjects of personal-injury claims. Juries often award verdicts in such cases, in view of the fact that sworn experts, who frequently do not at all understand the condition about which they testify, affirm that the injury was the sole cause of the trouble.

Epilepsy is another disease, which in this connection can be considered organic, and about which legal interest frequently centres. That typical epileptic convulsions follow head injuries, even when there is no discoverable injury to the brain, is an incontestable fact. In order to establish a reasonable support for such a contention in any given case, it is necessary to prove that the patient had not had epilepsy before the accident, and that the accident, in character and severity, was of a nature to produce such a result.

While the three diseases named above not infrequently figure in litigated cases, the chief interest, both legal and scientific, in traumatic affections of the nervous system, centres about the functional disorders known, since the appearance in 1889 of Oppenheim's monograph, as the traumatic neuroses. In the earlier treatises, and especially in Erichsen's, these neuroses were totally misunderstood and were classified with organic injuries. Progress throughout the whole field of neurology has now made it possible, in most cases at least, to distinguish these two great classes. As originally described by Oppenheim, the traumatic neuroses present chiefly the symptoms of neurasthenia and hysteria, but also some which indicate structural lesions. The term was a taking one, and has attained a rather different meaning from that which Oppenheim intended. To-day, by a traumatic neurosis is understood a simple neurosis, without known organic basis, plus such characteristics as its traumatic origin has added to it. Thus considered, the traumatic neuroses are composed of symptom groups which can, in nearly all cases, be brought under the rubric of neurasthenia or hysteria. In causation, they have many points in common. They have both attained their prominence through railway accidents. This is partly due to railway accidents so often being due to actionable negligence, and partly to the fact that in such accidents physical injury and mental shock are conspicuously combined. Both mental and physical elements are present in nearly all accidents. In most cases of neurasthenia the bruising and shaking up have been considerable, although severe surgical injuries are usually absent. Hysteria, on the other hand, is a fright neurosis above all else, and the history of injury in its causation is often very inconspicuous. It is well to observe in this connection that in the State of New York there can be no recovery of damages unless there has been a definite physical injury. Injuries resulting from fright alone do not constitute a cause of action.

Much has been written and much said about litigation as a cause of functional nervous diseases following trauma. If one were to be guided by the fluent generalizations of some railway claim agents, one would have to believe that any real injury to the nervous system could not occur on a railway; that all persons who allege such injuries either deceive themselves or wish to defraud the company. Certain experts, on the other hand, who are especially prominent in plaintiff's cases, are not inclined to accord much importance to the financial side of the question. Leaving aside actual simulators and impostors, who are very rare, I may say that my experience has taught me that the question of damages has a great influence on both neurasthenia and hysteria, and that in neither disease is restoration of health probable while litigation is pending. This baneful effect is due to the difficulty of carrying out proper treatment so long as legal questions are pending. Were the treatment for such cases simply medicinal, such a statement would naturally appear absurd. Medicines, however, play a very insignificant rôle in the treatment. They are of some indirect service, but far more important is the psychological direction of the patient. The diversion of the patient's thoughts away from morbid channels, the arousing of his interest in matters not connected with himself or his troubles, the exclusion from his consciousness of suggestions which may magnify or create symptoms,—these are the keys to the successful treatment of the traumatic neuroses. They are rendered powerless by the damage claim. The frequent examinations by experts (in some cases as many as five or six doctors examine a plaintiff), the law's delays, the legal inadvisability of the patient's returning to work, and the thousand and one annoyances inevitable to litigation, render futile any attempt to control the patient psychologically. These factors, in my opinion, are much more responsible for the continuance of symptoms than is any desire which the patient may possess to profit by his misfortune. This is especially true for traumatic hysteria, in which disease, aside from its being an agent in suggestion, the money question has little or no influence. The question of litigation as a cause of the traumatic neuroses must be kept separate from the question of voluntary exaggeration of symptoms actually present, and of simulation or fraud pure and simple. As far as actual simulation is concerned, it is very rare, and should not pass undetected by a phy-