Evidence which I wish to offer in favour of my own views as to ascending degeneration is divided into two parts. In this paper I wish to discuss the change in nerves themselves above the site of section, the anterior roots and the cord, with the exception of the nerve cells; and I propose, in another paper, to describe the changes induced in the nerve cells of the ganglia on the posterior nerve roots, and of the grey matter of the cord, as a result of my experimental work, and to preface these statements by a short critical digest of the most important researches on this subject.

The literature of ascending degeneration in nerves requires some mention, because opinions in the past have differed so idealy. Dickinson, in 1868, records wasting of the sciatic nerve after amputation. Hayem, in 1876, describes diminution of the anterior nerve roots, the nerve fibres being replaced by connective tissue, the myelin in some fibres being broken up, and the peripheral nerves undergoing parenchymatous neuritis.

Déjerine and Mayor, in 1873, describe no changes in the nerve in the connective tissue after amputation, save in the neuroma itself, in which they describe the fibres as growing in bundles, bent and twisted in many directions, some fibres shrivelled, and some granular, but the nerve fibres becoming more numerous as the nerve is traced upwards, and the connective tissue less and less marked. In some fibres only axis cylinders are left.

Hayem and Gilbert, in 1884, describe the nerves of stumps after amputation as very markedly reduced in size, the fibres diminished in number and also in calibre, not merely the myelin being less in amount, but the axis cylinders shrivelled, and connective tissue taking the place of degenerated fibres in the bundles. Some nerve fibres are very thick, the myelin being increased in amount, and the axis cylinders granular. The number of these vary in different bundles. There are also innumerable small fibres with little myelin and thick axis cylinders, the myelin staining well with osmic acid. These small fibres are grouped in bundles, or separated by connective tissue. The small bundles containing these fibres are in toto larger than an ordinary nerve fibre, each containing a varying

1 Dickinson, "On the Changes in the Nervous System which follow the Amputation of Limbs," Journ. Anat. and Physiol., London, 1868.
2 Déjerine et Mayor, "Recherches sur les altérations de la moelle et des nerfs du moignon chez les amputés d'ancienne date," Gaz. méd. de Paris, 1878.
3 Hayem et Gilbert, " Modifications du système nerveux chez un amputé," Arch. de physiol. norm. et path., Paris, 1884.
larger than an ordinary nerve fibre, each containing a very number of the small fibres. They think these are evidence of nerve regeneration. Krause and Friedländer,¹ in 1886, note in divided nerves of a stump after amputation simply atrophy, in addition, Wallerian degeneration; they incline to the view. They describe shrivelling and granularity of fibres, myelin staining badly by Weigert's method, and axis cylinders only faintly staining with aniline-blue. They think the atrophy of the axis cylinders and myelin is probably due to a chemical alteration, as the nuclei of the degenerated fibres also proliferate throughout the length of the nerves. The fibres are deficient in number, but at a greater distance from the neuroma they become more and more numerous. Generally the atrophied fibres for parts removed by the operation are centrally situated in the bundles, while the healthy fibres for the remaining parts of the limb are peripheral. The process begins three months after amputation, and goes on for several years. The anterior roots are normal, the posterior ones alter as far as the ganglia, but atrophied fibres are found passing through the ganglia. In other words, they think that sensory fibres have a peripheral trophic centre in the touch corpuscles.

Krause,² in 1887, states that in his opinion this degeneration of sensory fibres is due to the fibre losing its function by separation from the touch corpuscles, and that the touch corpuscles are trophic centres in themselves. Vanlair³ considers that atrophy of the anterior horn is only well marked in the young, or after a very long period of years. He gives much the same account as Hayem and Gilbert of the microscopical appearances of the nerves of the stump. The sheaths of Schwann become enormously distended by the fissiparously divided axis cylinders, each new fibre becoming surrounded by a small amount of protoplasm, but with no myelin sheath. He thinks that these fibres, which were considered by Krause, Gilbert, etc., to be due to shrivelling of original fibres, are in reality newly formed nerve fibres.

There seems to be no doubt about the powers of development of axis cylinders. Marinesco counted fibres and found them increased after amputation. Vanlair proved the capability of the growth of axis cylinders even into the Haversian canals of decalcified bone placed between the cut ends of a nerve, and Axel Key and Retzius found an isolated axis cylinder in the peripheral portion of the umbilical cord. Marinesco⁴ states that many fibres in the nerves of a stump appear to have very little myelin, and re-

¹ Krause u. Friedländer, "Über Veränderungen der Nerven und des Rückenmarkes nach Amputation," Fortschr. d. Med., Berlin, 1886.
² Krause, Arch. f. Anat. u. Physiol., 1887, Physiolog. Abth. s. 370.
³ Vanlair, "Alterations nerveuses centripètes consécutives à la névrotomie et aux amputations des membres," Bull. Acad. roy. de méd. de Belg., Bruxelles, 1891.
⁴ Marinesco, " Modifications des nerfs et de la moelle chez les amputés," Berl. klin. Wochenschr., 26th Sept. 1892; "Sur les altérations des nerfs et de la moelle après amputation et névrectomie," Neurol. Centralbl., Leipzig, 1898.
semblé naked axis cylinders. He notes greatly distended axis cylinders, 5 μ in size, taking on well the colour of the stain, hyaline in appearance, and with a chromatic substance disposed like a v, an x, or a star. Fibres may disappear and be replaced by connective tissue. With a high power he can see semilunar protoplasmic cells round some of these fibres within or outside the sheath of Schwann. There is a disappearance of fibres in the spinal ganglia, but no change in the cells. He thinks the greatest number of fibres actually degenerate, although they are connected with their trophic centre; it is not atrophy, but is Wallerian degeneration, from which he asserts it cannot be differentiated.

Marie,1 in 1892, records a case of amputation twenty years before death. He examined the stump and found few normal nerve fibres left, and these, he says, belong to the parts of the limb above the level of the amputation. Also, most of the fibres show a swollen sheath of Schwann, with the new formed axis cylinders described by Hayem, Krause, Friedländer, Vanlair, etc. There are numerous nuclei between the nerve fibres. He thinks these changes are secondary to degeneration, and are due to the fibres being cut off from their trophic centres in the skin. Where, on the other hand, the degeneration is found to be more universal than this, it points to an infective neuritis, resulting from a septic wound. Redlich (E.),2 in 1893, finds that seventeen to eighteen days after an experimental amputation of a guinea-pig's leg, there are degenerated fibres in the anterior roots and in the anterior cornua, near the lateral groups of ganglionic cells. In the opposite anterior root fibres are affected, but to a less extent. There is little change in the posterior roots. After thirty-six to seventy-six days, very intense degeneration is found in the anterior roots up to the cells in the anterior cornua; the posterior roots are also degenerated, but less marked than the anterior, and no degenerated tracts are found in the cord. He describes the same changes in man, but only noted in recent cases, and these central degenerations are much less marked than the peripheral. He thinks degeneration in the central end of a nerve mainly affects the motor fibres.

Darkschewitsch,3 in the same year, 1892, publishes the result of ligation of the sciatic, finding in both anterior and posterior root fibres some amount of degeneration or segmentation of the myelin. Moschaew, in 1893, finds, three weeks after experimental lesions of nerves, that both roots, and especially the posterior, are degenerated, and that degeneration, missing the ganglionic cells, passes up the posterior tracts of the cord. Feinberg,4 in 1894, and

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1 Marie, "Leçons sur les maladies de la moelle," 1892.
2 Redlich (E.), "Zur Kenntniss der Rückenmarks Veränderungen nach Amputation," Centralbl. f. Nervenhe. u. Psychiats., Coblenz u. Leipzig, 1893.
3 Darkschewitsch, "Lésions dans le bout central d'un nerf moteur sectionné," Neurol. Centralbl., Leipzig, 1892.
4 Feinberg, "Myélopathies post-névritiques," Ztschr. f. klin. Med., Berlin, 1894, Bd. xxv.
Grigoriew\(^1\) in the same year both find changes in the cord, an ascending degeneration causing breaking up of myelin being propagated into the posterior roots and so upwards to the cord. They both consider sensory fibres more markedly affected than motor. Homen,\(^2\) in the same year, as the result of amputating the limb in dogs at different ages, and killing at different intervals of time extending from days to years, finds that younger dogs show more marked changes than older. In a dog eight months old, and three weeks after amputation, the greatest number of sciatic nerve fibres were unaffected; amongst those altered some are of normal size, some smaller, some very specially so. Increase of nuclei in the sheath of Schwann was noted, especially in these small nerve fibres. Some loose connective tissue surrounding the fibres was found, and this might press seriously upon them. Many of the nuclei are broad, and seem to have a larger amount of surrounding protoplasm than usual. The myelin is granular, and the axis cylinders are often interrupted at several places. These changes appear in one to three weeks, and are found in small bundles of fibres. There is no change in the epineurium.

Let me sum up in a word the different theories formulated regarding ascending degeneration. Erlangmayer, Genzmer, Homen, Vanlair, Hayem and Gilbert, Cossy and Déjerine, amongst others, believe it is due to a simple atrophy, though some of these authors find empty sheaths, suggesting a very complete destruction of certain fibres. This, however, becomes less marked the further the central end of the divided nerve is traced. Other authorities, as Krause, Friedländer, etc., believe that the myelin suffers, while the axis cylinder is preserved more or less intact; while Marie holds that in the intramedullary fibres of the posterior roots, degenerative changes are specially well marked; although his theory that this is due to degeneration affecting collaterals, rather than the nerve fibres, seems rather strained.

In fact, we may state that, almost without exception, the degeneration differs from Wallerian, inasmuch as the myelin suffers most, and the axis cylinders, if they do become thinner, as Forel thinks, do not at least disappear. Purely motor nerves suffer more than mixed nerves, and the consensus of opinion is that the central end of such a nerve, as for example the seventh, may degenerate (Forel), and may do so by passing through a true Wallerian degeneration (Darkschewitsch). Darkschewitsch and Tichonow state that this is best marked at the anterior roots, while Brègmann and Bikèles consider that the lesion gets less marked towards the

\(^{1}\) Grigoriew, "Zur Kenntniss der Veränderungen des Ruckenmarkes beim Menschen nach Extremitätenamputationen," *Ztschr. f. Heilk.*, Berlin, 1894, Bd. xv.

\(^{2}\) Homen, "Die Krankhaften Veränderungen der Nerven nach Amputationen, Atlas der Pathologischen Histologie des Nervensystems," Von Babes u. Blocq, 1894, Lief. ii.
centre. Forel does not admit that in any sense of the term this is an ascending degeneration, but rather that it is a descending degeneration, consequent on the severe damage sustained by the trophic nerve cells from the effects of the nerve section; and in this opinion Golgi, Ramon-y-Cajal, Monakow, and Van Gehuchten join. Durante denies this descending theory, and brings in support the following arguments, that in ascending degeneration of the pyramidal tracts, degenerative changes cannot be traced beyond the decussation in the medulla; and that the changes become less and less marked, while the trophic cells need not suffer at all, and often do not. Further, he states that this "retrograde" degeneration can show itself in eighteen days in motor nerves, and in fifty to fifteen days for the posterior column; that it is therefore much slower than Wallerian, but that it is more rapid and more marked in the young of man and animals than in the old.

Leaving for later consideration an account of the changes in nerve cells, it seems self-evident that if the trophic cell itself suffers, the change cannot mean cell death, or an incurable loss of functional power, because certainly months and even years after a nerve section, the two ends may be made to reunite long after the peripheral end is degenerated, and when it can only serve as a guide for the actively growing axis cylinders from the central end.

The existence or non-existence of an ascending process, degenerative or inflammatory, is a momentous question to the surgeon who amputates or performs a neurotomy or neurectomy. Is there in reality an ascending (degenerative) process which may spread up the cord to the brain, and if so, is it a degeneration or an inflammation, or is there no such process at all?

There may be several explanations of the discrepancies. I suggest the following:

First, The methods adopted by certain observers for operation, and for fixing and hardening the specimens obtained, may be to blame. As regards operative methods, the most rigid antiseptic precautions are absolutely necessary, and in using dogs as subjects this presents extreme difficulty, because dogs pick out stitches, tear open wounds, and very readily induce suppuration.

If we consider what is really included in the term of traumatic ascending neuritis, we have something which will throw some light upon the discrepancies just referred to. An ascending neuritis may be very acute, rapidly spreading up to the cord, affecting ganglion cells in the grey matter, and even extending up to the medulla, producing a fatal result, or more commonly it may be an acute, subacute, or extraordinarily chronic but generally less disastrous inflammation, the result of a less potent agent. How to produce the severe form we do not know. Very probably it is microbic, but the less extensive variety is much more easily obtained than is often stated. Howell and Hubert rarely got it in dogs, as the result of nerve sections, crushing, etc. Hayem got it
by tearing out the sciatic of dogs, whereas other experimenters, as for example Vulpian, very seldom, if ever, found it at all.

A neuritis of this kind is never induced without some means of admitting an organism, or a more or less virulent toxin circulating in the blood, such as gout, rheumatism, and probably the toxic agent in diphtheria, influenza, etc.; and a septic wound is the common cause of the organismal variety. Although apparently a modified form of inflammation may be produced by pressure on a nerve, it is certain that if the nerve has been injured by section, a very severe inflammation may result; I have repeatedly seen this demonstrated in rabbits, where a haemorrhage starting after a wound was closed gave rise to tension with a very rapid ascending neuritis, and greatly hastened the degenerative changes in the peripheral portion of the severed nerve. As Strümpell, Kast, and Rosenbach consider most probable, the neuritis resulting from an extra nerve injury is due to thickening of connective tissue, and is a degeneration caused by pressure and not a true inflammatory condition at all. It is a well recognised fact that a nerve, if its sheath be not damaged, can, without much injury, be bathed in pus, and may in fact only eventually show evidence of involvement by the pressure of the resulting cicatricial tissue after the pus had become absorbed or is evacuated. I need not do more than refer to the fact that there is little and certainly no very distinct connection between the intra- and extra-funicular lymphatics.

My experience is that the slightest sepsis of a wound, if a nerve lie exposed in that wound and the nerve fibres have been divided, will lead to an ascending degeneration, and that this accounts for the phenomena of ascending degeneration up to the posterior ganglia, and involving often the posterior nerve roots, and may attack, though probably to a less extent, the anterior motor roots. If dogs are apt to tear out stitches, cats are far worse, and therefore the results obtained by experimental work on these animals are open to some comment. One observer states that, though the operations were strictly aseptic, his bandages were gnawed through, and that it was very difficult to keep up an antiseptic condition of the wounds, while he seems quite satisfied to find more or less pus below the sutures, if the sutures were allowed to remain at all.

In my own series of experiments I used no antiseptic except boiling water, and rejected every case where there was not union by first intention, or where there was any cheesy material in the wound. So far the operations on dogs’ sciatics which I have performed or seen performed number fifteen in all (on fifteen different animals), and of these—carried out with the utmost care so as to prevent the entrance of septic organisms—only about one in four of the wounds healed by first intention, and the central ends of most of the dogs’ nerves show far more signs of a degenerative or inflammatory
process than the rabbits'. A rabbit never picks out stitches from the hind-limb, and, if well cared for, there is little tendency to septic mischief. Hence, I believe, rabbits are more satisfactory for experimental work of this kind than dogs. All rabbits, however, are not equally good; short-haired species are by far the best.

It may seem a little over-confident to assert that every ascending degenerative change after a nerve section, other than the comparatively slow atrophic process about to be described, is of septic origin; the consensus of opinion appears to be shaping in that direction—and I cannot but believe that the opponents of this view might be led to modify their opinions were they to use rabbits more and dogs and cats less for experimental purposes.

As regards fixing and hardening, I need merely remark that something very like segmentation of myelin can be produced in an insufficiently hardened nerve by the action of ether in the celloidin process. It is inconceivable that a cell can have its axis cylinder process lopped off, and its function in the organism interfered with, either partially or totally, without suffering. The extent of the damage, so far as the fibres are concerned, may possibly include a greater tendency to segmentation of the myelin, under the influence of a reagent such as ether.

In not a few of the anterior roots of my rabbits, belonging to the side of the divided and therefore damaged sciatic nerve, this segmentation of myelin can be seen in celloidin specimens; perhaps in some fibres I should rather call it a series of bead-like swellings. As this can be produced artificially in a normal, badly-fixed fibre, it bears out my contention that a damaged—I do not say degenerated—nerve fibre, however slightly damaged, requires much longer and more careful hardening than a healthy fibre.

Second, An explanation may be sought for in the confusion of ideas which surround the question of what ascending degeneration really is. Is it an inflammation or a degeneration? There has been much discussion as to what the evidences of inflammation in peripheral nerves really amount to. I would say that one evidence is, an exudation with leucocytes, in addition to degenerated fibres, segmentation of myelin, etc.

The fact that several writers have changed their opinions from their original, and, as I would suggest, erroneous views, points to insufficient evidence in their first series of experiments or observations.

I propose to refer, first, to a specimen obtained from a patient, aged 27, whose right arm had been amputated above the elbow, at the age of 17, and who died of pernicious anaemia. Unfortunately, the only part I was able to secure was the terminal portion of the ulnar nerve, with the bulbous swelling thereon. The specimen was fixed and hardened in Müller's fluid and methylated spirit. The changes to which I wish specially to call attention are the evidences of ascending degeneration.
Specimens stained by the Weigert-Pal method show that immediately above the bulbous termination there are many funiculi with an apparently diminished number of nerve fibres, but the missing fibres were probably those nearest to the fibrous tissue, that is, the endoneurial septa and the perineurium. With the high power, many fibres show that the stained portion occupies an intermediate position, and not a peripheral one, as a healthy fibre generally does. This deeper peripheral staining in a healthy fibre with Weigert's haematoxylin stain is probably due to a condensation of the myelin, at or near the inner surface of the sheath of Schwann; and in a paraffin section, stained with haematoxylin and eosin, this condensation looks like a thickening of an inner stratum of the sheath of Schwann. These fibres do not stain uniformly, a condition which, in a well-fixed and stained Weigert-Pal specimen, indicates an early stage of degeneration; but the middle portion of the medullary sheath stains much more deeply than either the central or the peripheral.

While it is quite possible that this appearance is, to a certain extent, accidental (and I have no other amputation specimen of so many years' duration with which to compare it), the condition does suggest an early atrophic process.

In the neuroma itself the fibres are divided into smaller but more numerous bundles, many of the fibres being evidently subjected to considerable pressure, due to the great thickening of the endoneurium, perineurium, and the epineurium. Besides the bundles of fibres, which are very markedly twisted in all directions, there are a great number of medullated fibres, which course through the fibrous tissue in all directions, mostly running for a considerable distance in the direction of the main strands of this tissue, and then, suddenly branching to one or other side, force their way through a dense matting of fibres with apparently the greatest ease. This can be accomplished as well by a single fibre as by several fibres together. Isolated fibres can be often followed a long distance, and do not appear to suffer as regards thickness or capability of taking on the Weigert-Pal stain. These fibres are probably merely the terminal ends of the nerve fibres of the bundles, which have grown and twisted in all directions in their effort to find a new road towards the periphery. At first one almost agrees with Vanlair that these fibres are greatly increased in number, the result of the formation of new axis cylinders, and it is possible this may be so. My specimen does not demonstrate any proof of the accuracy of Vanlair's opinion, for there are sufficient nerve fibres in the original nerve to give the appearance of networks of fibres found in the neuroma.

Just above the terminal neuroma the fibrous tissue of the perineurium is greatly thickened, and in the endoneurium not merely are the broad septa increased, but between little groups of fibres numerous connective tissue nuclei may be seen. These little
groups of fibres are specially interesting. They do not occur in large numbers in all the bundles. I have endeavoured in Plate II., Figs. 1 and 2, to bring out the following points. Both figures are taken from the nerve a short distance above the neuroma. In Fig. 1 there are very few clusters or groups of connective tissue nuclei, and on careful observation nearly all the fibres are seen to be large fibres—where nuclei do occur in numbers a few fine fibres are seen. In Fig. 2—a funiculus very near the preceding one—there are a very great many more clusters of nuclei, and these are seen to be associated with fine medullated fibres arranged in groups. But in both of these funiculi the fine fibres are not normal when viewed with the high powers. The bundle represented by Fig. 2 ought to contain large numbers of fine fibres, the other bundle seen in Fig. 2 evidently ought not.

Weigert-Pal specimens show these little fibres manifestly degenerated, not the problematical degeneration described as effecting a change in the part of the medullary sheath taking on a particular stain, but the myelin is segmented or absorbed, the axis cylinder stained feebly or absent altogether, and the segmented nuclei proliferated.

The paraffin specimens stained with haematoxylin and eosin show the striking increase in nuclei where these little groups of fibres are situated, the disappearance of many of the fine fibres, the nuclei just referred to taking their place; and the loss of axis cylinders in not a few of these fibres, the outline of whose sheath is still distinguishable.

Longitudinal sections through these little groups demonstrate beyond any doubt that the bulk of these nuclei are, in reality, long spindle-shaped connective tissue nuclei, and not mainly proliferated segmental nuclei.

The obvious conclusion is that the connective tissue has replaced these fine fibres to a great extent—many of them being shrivelled beyond recognition, and many minus axis cylinders—and that most of the proliferated segmental nuclei have disappeared.

On careful examination of the various funiculi in the specimen, I was struck by the fact that, though in the ulnar nerve there are generally a considerable number of very fine medullated fibres, here there are extremely few; and we find funiculi composed almost exclusively of large fibres, which are much more healthy. These fine fibres, as Gaskell has pointed out, are probably designed for the supply of vessels, and they mostly leave the cerebro-spinal axis in certain regions alone, pass to the sympathetic ganglia, and from them return to peripheral nerves. The fine fibres are known to be medullated in a part of their course, and they are certainly medullated in many animals in their path along the peripheral nerves. The surrounding connective tissue acts normally as a protecting framework to fibres whose function of vasomotor is of the utmost importance.
These fibres, scattered all through the funiculi in which they occur, seem to find their way to the periphery of a funiculus at different levels, and probably do so near their points of distribution. In support of this theory, I find in my specimen the vessels in this nerve have suffered, right up to the highest part of the ulnar nerve. The perineurium is thickened apparently further than the limit of my specimen. There is a very slight diminution, but still a diminution in the total number of the degenerated groups in any funiculus, estimating the number from below upwards.

The smaller arterioles and capillaries in the endoneurium show most marked increase in number of the endothelial nuclei of the intima, of the nuclei of the media, and to a less extent of the adventitia. On careful observation, there is no question that, where in a funiculus the minute fibres are degenerated, the vessels in the neighbourhood have suffered in this way; whereas in the few funiculi which show most of these fibres unaffected the vessels have escaped. These nuclear changes are less marked in the vessels of the perineurium, and are only found in parts of the epineurium.

Longitudinal sections through the fine bundles in the nerve above the neuroma show that the nuclei are of far greater length than those found in grey fibres; and although it may be argued that the grey fibres have shared the fate of the white, still my experimental results tend rather to prove that most of the nucleated fibres are those of connective tissue.

Determined to ascertain if the central ends of the divided nerves in my experimental section of rabbits' sciatics would lead to the same conclusion with regard to these fine fibres, I made careful observations in each case. I examined the central end of the divided or ligatured nerves in about thirty rabbits, at periods varying from four to fifty days after the operation. My results are as follows:

There is a fairly rapid development of nuclei and of connective tissue fibres between and around the fine medullated fibres, and this change is recognisable by the end of four to seven days, and very marked by the end of the third week. In Plate II., Figs. 3, 4, 5, and 6, I have tried to illustrate the appearance of this connective tissue increase at the end of the twenty-third day after ligaturing a rabbit's sciatic.

Fig. 3 shows the sciatic on the affected side in transverse section, the connective tissue fibres forming strong septa, evidently capable of exerting hurtful pressure on the fine fibres around them; and note the thickened strands are confined to the site of the fine fibres.

Fig. 4 shows a longitudinal section of the same nerve, although possibly not the same funiculus. The fine fibres in the neighbourhood of the dense bands of connective tissue may be
made out, though many of them have suffered in myelin, axis cylinder, or both.

Fig. 5 represents a much higher power view of the thickened connective tissue trabeculae compressing the fine fibres.

Fig. 6 shows, by way of contrast, a longitudinal section of the same nerve on the healthy side. The relationship of the fine fibres to the connective tissue strands can be made out, but the strands are not nearly so thick as in the affected nerve.

Figs. 4 and 6 are taken from slightly diagrammatic sketches of the sciatic nerves.

A ligature twenty-three days old might give rise to the connective tissue increase, as the result of irritation, but it could not be due merely to the length of time. It is much more probable that it is because the fine fibres have sustained damage that this local connective tissue increase has made its appearance. Supposing, then, that a section of one of these fine fibres means that it is cut off from a trophic centre, not necessarily its only trophic centre, this might cause the change in question; or, another theory, these fine fibres are influenced from two stations, one at either end—that is to say, they conduct almost certainly both ways—therefore a peripheral stimulation or irritation of these fine fibres, produced at the site of section, may account for the phenomena.

There are no vessel changes in the central end of the rabbit's sciatic, as there were in the neuroma; but if the vessel changes depend on a nerve cause, the fine fibres supplying the vessels seen in the central end of the nerve are still undamaged; whereas, in the neuroma, the age of the lesion would mean contraction of the fibrous tissue replacing degenerated fine fibres. In this way previously healthy fine fibres in the immediate vicinity would be interfered with, and thus the vessel changes for some distance up the ulnar nerve in the neuroma might be produced. Probably similar changes would appear in time, after experimental section, but only after months or years.

To sum up: First, The fine fibres appear to suffer first and most, connective tissue replacing the fibres severed, and these fibres degenerate a certain distance upwards at least. Unfortunately, I cannot supply any exhaustive evidence as to how far these fine fibres degenerate. A research into the changes resulting in the cells of the chain of spinal sympathetic ganglia, of the cells in the medulla, where probably the vasomotor centre is situated, and finally, the presence of degenerative changes in the connecting links between sympathetic ganglia, the medulla, and the peripheral nerves, would require to be instituted before this question could be finally settled.

As far as my specimens go, the degeneration does not extend the entire length of these fibres; but I shall have other evidence to bring forward in my next paper.

Second, The larger fibres, though they do suffer to a certain
extent, only show a very slow and probably a simple atrophic change, and the myelin suffers more objectively than the axis cylinders. I have nothing fresh to state with regard to the larger-sized fibres, which undoubtedly do degenerate in the central end of a divided nerve. They are far from numerous, and often wanting almost altogether.

Third, There is a very gradual thickening of connective tissue, of perineurium, and also endoneurium, which takes the place of the atrophied fibres.

**DESCRIPTION OF PLATE II.**

**Figs. 1 & 2.**—Funiculus from ulnar nerve of human stump, ten years after amputation. Fig. 1 shows mostly large nerve fibres. Compare with Fig. 2, where there are more groups of fine medullated fibres, associated with numbers of nuclei. *n.m.* groups of fine medullated nerve fibres. *t.* connective tissue trabecule and nuclei. *s.* segmental nuclei. *per.* perineurium. *end.* endoneurium. *f.n.* nuclei of fibrous tissue.

**Fig. 3.**—Transverse section of funiculus from central end of sciatic of rabbit, twenty-three days after ligature, shows thickening of connective tissue strands between the fine fibres. *n.* ordinarily sized nerve fibres. (Other lettering, vide supra.)

**Fig. 4.**—Longitudinal section of same nerve (Fig. 3) shows same thickening of connective tissue, limited to neighbourhood of fine fibres. (Lettering, vide supra.)

**Fig. 5.**—A high-power view of one of these strands of connective tissue seen in Fig. 4. *a.x.* interrupted axis cylinder of a fine fibre.

**Fig. 6.**—Longitudinal section of funiculus from a normal sciatic of rabbit, showing connective tissue supporting fibres. (Lettering, vide supra.)

Figs. 4 & 6 are from slightly diagrammatic sketches.

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**Clinical Records**

**NOTES ON A CASE OF A PIN IN THE LARYNX, LOCALISED BY RÖNTGEN PHOTOGRAPHY, AND REMOVED BY EXTERNAL OPERATION.**

By Walker Downie, M.B., F.F.P.S.G., Lecturer on Diseases of the Throat and Nose, Glasgow University.

On 23rd March 1896, a lad from Milngavie, aged 19, presented himself at the throat and nose department of the Western Infirmary, complaining of pain in the throat, of three days’ duration. In giving an account of the possible cause of his discomfort, he stated that, while asleep in bed on the night of the 20th March, he seemed to discover a pin, either in his clothing or in the bedding, but where exactly he could not explain, and this he