On certain changes in sensation associated with gross lesions of the spinal cord : Thesis for the degree of M.D. in the University of Cambridge.

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To which the Raymond Horton-Smith Prize was awarded in 1906.

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Medical Registrar at the London Hospital, E., 1905 and 1906.

Assistant Physician to the Hospital for Sick Children, Great Ormond Street, W.C.

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PREFACE.

The effects upon sensation produced by lesions of the spinal cord have been the subject of much recent work, and the conclusions drawn by various investigators differ considerably from one another. The fact that a lesion in one part of the spinal cord produces sensory changes differing from those which occur when the lesion occupies another position has been known for many years, and the conclusion has been drawn that the various sensory impulses pass up in the spinal cord along distinct paths. But the exact situation of these paths and the nature of the sensory impulses they conduct have been the subject of much controversy.

Thus among recent authors, Ziehen (1) is of the opinion that all forms of sensory impulses pursue in the spinal cord a path which is partly crossed and partly uncrossed.

Most authors however, following Brown-Séquard (2), consider that the path of these impulses within the spinal cord is wholly a crossed one.

Van Gehuchten (3) and others consider that impulses of pain and temperature pass up in the white matter, while Déjerine (4) agrees with Schiff in thinking that these forms of sensory impulses pass up in the grey matter of the same side.

There must be reasons for conclusions so discordant. One reason may be found in the fact that many of the conclusions are based upon the results of animal experiments. In matters of sensation it must however be clear that these experiments cannot have the same weight as carefully observed clinical cases in man. Those cases in which the spinal cord has been divided by a stab from a knife have all the accuracy of animal experiments, and in addition sensation can be tested in subjects who are able to describe their sensations by means of language. Another reason for these diverse conclusions lies in the great variety of the methods which have been used to test sensation, and the often totally inadequate definition which has been given to these methods. Thus it has recently been shown that of the stimuli used to determine "light touch" the greater number were quite useless for this purpose, as they aroused deep sensibility as well.

Further uncertainty was caused by the very imperfect understanding of the way in which sensory impulses were conducted to the spinal cord by the peripheral nerves.

The recent work of Head, Sherren, and Rivers, upon the conduction of sensory impulses in the peripheral nerves, has been freely used in this Thesis; and without this aid it would have been impossible to unravel the complexities of sensory conduction in the spinal cord.

CHAPTER I.

The Methods Used for Testing Sensation.

No work on the sensory nerves can be considered complete without a clear account of the methods that were used for testing sensation. Unfortunately many authors neglect to give this, and the value of their observations is therefore greatly diminished.

The following methods were rigorously applied to all the cases described in this Thesis.

During the testing of sensation, the patient's eyes were closed, and when necessary bandaged. He was told to call out whenever he experienced a touch, a prick, or any other sensation, and to say what kind of sensation he felt. At the same time he was asked to point to the place on the skin from which the sensation seemed to proceed. No further questions were asked.

While sensation is being tested it is of the utmost importance to ensure that the patient should not be distracted by any external auditory or visual stimuli. Consequently the majority of these observations were made in a small room, containing only a bed and a small table. The patient was made as comfortable as possible upon the bed, and in no case was the testing proceeded with until the patient was perfectly composed. In some few of the cases, the testing had to be done in the hospital ward, because the patient was unable to walk; but in these cases, by a suitable arrangement of screens, every effort was made to secure his undivided attention.

Another factor which has been found greatly to influence the condition of the patient is the state of the weather. In winter time, the accuracy with which replies were given was slightly, though perceptibly, less than in the summer, and the limits of differentiation between slight variations of sensation were distinctly altered. This is not altogether a matter of external temperature, for it was found that a damp, misty, or foggy day was a peculiarly unfavourable time for testing sensation.

Such variations with weather were found to exist not only on parts where the sensibility had been altered by disease, but also over skin where the sensibility was perfectly normal.

Superficial or "light" touch was tested by means of cotton wool stroked gently across the skin.

The majority of the more commonly used tests for light touch are useless for the purpose, as they stimulate deep sensibility. Thus when all the cutaneous nerves supplying a certain area of skin have been divided, the touch of a finger, or of the feathers of a quill, is easily recognised and well localised. If however the skin is lifted up these methods of stimulation fail to evoke any response, proving that whatever sensation had been previously present was due to the stimulation of the end organs in underlying structures.

Moreover the cotton wool test needs to be applied with extreme care. As Head and Sherren (5) have pointed out, many brands of wool, when rolled into a wisp, form so stiff a mass that deep sensibility is evoked. This is especially noticeable with regard to the cotton wool of short staple, which, being rendered absorbent by potash for use as a surgical dressing, is much too stiff to afford a certain stimulus for superficial touch alone. The untreated cotton wool used by jewellers should always be employed.

Moreover the application of this test over hairy parts may be vitiated by the peculiar sensibility of the hairs themselves. Consequently, if it be desired to test the sensation of the skin to light touch over a hairy part, it is essential to shave the part before doing so.

This fallacy is easily exposed by experiments on hairy and hairless areas of the skin of a normal person. Over the hairless palm of the hand, cotton wool produces a very slight though definite and well localised sensation. Passing however to the hairy parts on the back of the hand, a very marked and definite sensation is produced as soon as the hairs are reached. This so greatly exceeds the sensation produced on the palm that, if the testing is carried out in the reverse order, the stimuli on the palm may not be recognised at all, especially if the weather is cold.

Again, certain parts of the palm of even a well kept hand, for example the inner side of the thenar eminence and the front of the wrist, are relatively insensitive to stimulation by cotton wool.

Remembering this, one is not surprised that as a rule stimulation of the "horny hand" of the workman fails to evoke any response. Enough has been said to indicate the fallacies of this method of stimulation, and one must conclude that most of the statements in the literature as to the presence or absence of "light" touch sensibility were in all probability only the records of the sensibility to pressure.

The power of discriminating two points, simultaneously applied to the skin, was tested by means of a pair of compasses with blunted ends. It is of the utmost importance that the two points should be applied simultaneously: for if one point is applied even a fraction of a second after the other, the patient will reply that he feels two points; whereas he may be quite unable to recognise them as two when the ends of the compasses are applied exactly together. The method used for recording the results obtained is that of McDougall (6). The part of the skin tested is accurately noted. The relation of the line joining the two points of the compasses to the axis of the limb or trunk, whether transverse or longitudinal, must be recorded, as the liminal sensation varies considerably with the angle between these two lines. The blunt ends of the compasses are then separated for a fixed distance measured in centimetres. The skin of the part is touched, and the patient, whose eyes are closed, is asked to say after each application whether he has been touched by one or two points. When the points of the compasses are separated by a certain distance, which varies at different parts of the body, the points no longer appear as two on the normal skin. The patient is touched ten times with the two points and ten times with one point only, each form of application being applied at random in the series. The results are recorded graphically in the following manner:—

Every time the patient answers correctly a stroke is made, above a horizontal line, if touched with one point, below it if touched with two points. An incorrect answer is recorded by a cross, which is placed above or below the horizontal line according to the stimulus which is applied. If the patient is in doubt the letter D is substituted, while O is used if he fails to answer at all. The following records taken from one of my cases will illustrate the method :—

Ulnar half of right hand.

Transverse.

2 cm. apart.	1 | III II IIIII	1 | 100 % Right.
	2 | IIII III III	2 | 100 % Right.

This record indicates that the patient is perfectly able to discriminate the compass points at 2 cm. apart in the area of skin indicated.

1 cm. apart.	1 | IXII IXI IIX	1 | 70 % Right.
	2 | IXII IXI III	2 | 80 % Right.

Such a record indicates that the threshold of sensation is nearly approached, and it will be noticed that there is a tendency to estimate one point as two, since 30 % of the answers were wrong when the patient was stimulated with one point.

This curious phenomenon, noticed by Head and Sherren in their studies on the peripheral nerves, was also frequently observed in my cases when the limits of accurate discrimination had been reached, and it occurred on areas where sensibility was normal as well as on areas where it was defective.

·75 cm.	1 | III II IIIII	
	2 | XXXX XXX XXX	

Such a record shows that the threshold of sensation has been completely passed. Here every double stimulus causes the patient to answer as if it were single.

It is noteworthy that while on the palm the threshold for "compass sensation" is higher when the points are applied longitudinally than when they are applied transversely, yet on the trunk the threshold is lower when the points are applied longitudinally than when they are applied transversely to the axis of the body.

It may be of interest to recall the magnitude of the threshold distance for the discrimination of the compass points in various regions of the skin.

On the palm the average liminal distance was 1 cm. : on the forearm and leg, 4 cm. : on the trunk, 5 cm. : and on the arm and thigh, 6—7 cm.

Sherrington (7) quoting Czermak gives the following numbers :—

FOR ADULT MEN.

Palm	1.13 cm.
Forehead	2.26 cm.
Forearm	4.06 cm.
Sternum	4.51 cm.
Thigh	6.71 cm.

When the power of discriminating the two points of a pair of compasses becomes lost in disease of the spinal cord, the distinctness of the loss is exceedingly striking. Thus in one case,

while the patient was almost perfect at 1 cm. on the palm of the right hand, he was totally unable to distinguish the two points on the left palm, even when 5 cm. apart, every stimulus evoking the answer "one." It is only upon such large differences that stress is laid in this Thesis. It must be remembered that the power of discriminating the two points of the compasses will depend not only upon the power of localisation, but also upon the integrity of tactile sensibility.

The sensation of pressure was tested by means of the blunt end of a pencil or the head of a pin. The locality on the skin to which the sensation was referred was carefully recorded.

The *pain* produced by a firm pressure or crush was also tested. This is the sickening sensation that is produced when a bone is violently squeezed, and the sensation is very characteristic. It need scarcely be said that it is impossible to make this test satisfactorily unless the skin is analgesic.

Another test applied was that of *tuning fork vibration*. A long tuning fork vibrating 128 times a second was used. This method was found to be very untrustworthy. Not only did error arise owing to vibrations being audible to the patient, but also from the conduction by the tissues of vibrations from an insensitive to a sensitive part at a distance from the point stimulated.

Attempts were made to test the *sense of area*. Thus, round discs of 2 cm., 1 cm. and 2 mm. diameter were applied successively to the skin, and it was found that on normal skin the patients were able to differentiate between the areas of these three discs, but that this power was often lost in diseases of the spinal cord. This form of sensibility will hereafter be referred to as the "sense of area." It was however found that the discs had to be applied with an exactly equal pressure to the skin, for if the disc of 1 cm. was applied firmly while the 2 cm. disc was only placed lightly on the skin, the patient would declare that the former was larger than the latter.

The *sense of form* was tested by applying a rounded or convex and a flat object to the patient's skin, and asking him to differentiate between them. The usual test of asking the patient to distinguish between coins of various sizes laid upon the skin is fallacious; it is a test only for the sense of area.

Pain was tested by means of a sharp needle or pin, and care was taken to differentiate between the sense of pressure so produced and true pain. Thus, when a report of a case reads that "a prick was well felt and localised," it is of importance to know whether this was an actual sensation of cutaneous pain, or was merely a sensation produced by the stimulation of end organs in deeper structures. Thus, over an analgesic area, a patient may not only be able to appreciate and localise a pin-prick, but may also be able to distinguish the point from the head, and say that the former is "sharp" (acuæsthesia). Consequently, unless great care be taken, analgesia may be overlooked. In cases of doubt, the application of a painful interrupted current distinguished with great certainty whether the patient's replies were based on sensations of deep pressure, or upon a true appreciation of pain as such.

Sensibility to heat and cold was tested by applying to the skin test-tubes filled with water at various temperatures. The objections that have been raised to the use of glass tubes fortunately did not hold good in our investigations. It has been urged that the glass wall of the test-tube is hotter than the water it contains, if the test-tube has been heated in a Bunsen flame. The outer surface of the test-tube will be colder than the water it contains, if hot water has been poured into the cold test-tube. Early in our investigations however it became clear that, when the sense of heat or cold was lost in cases of gross lesions within the spinal cord, it was absent whatever the temperature of the water which was used as a stimulus. Consequently a difference of a degree or two between the actual temperature applied, and that registered by the thermometer within the test-

tube, is not of great importance. It must be noted that stimulation by temperatures over 50° C gives rise to painful sensations as well as to sensations of heat.

The sense of passive position, or the ability to appreciate the position into which a joint has been passively placed, was tested as follows:—

In the case of the hand, the patient was told that the fingers of one hand would be moved, and that he was to imitate with the sound hand the position in which they had been placed. In the case of the great toe he was told that the toe would be placed pointing upwards or downwards, and that he was to indicate his sensation by the words "up" or "down." A similar method was used when the patient's hands were paralysed. In some of the later observations I used a method of recording the results of this form of stimulation analogous to that of McDougall for the discrimination of the two points of the compasses. When the sense of passive position is seriously affected in gross lesions of the spinal cord, the patient often says that he is quite unable to tell in what position his limb is placed. If however the patient attempted to guess, it was found to be an excellent plan to allow him to open his eyes after he had made a wrong answer, when the look of surprise on his features, on seeing the real position of his limb, was conclusive evidence that his sensibility for passive position had been lost.

Electrical Sensibility. This was tested by using the painless interrupted current, a faradic current of so small a strength that the tingling sensation alone was appreciated by normal persons. The painful interrupted current was found to be of the greatest value in this work. In some cases it was difficult to delimit an analgesic area owing to the pressure of acuæsthesia, but the application of the painful interrupted current at once enabled me to define the limits of the analgesic area with the greatest precision.

CHAPTER II.

The Kind of Cases Investigated.

Gross lesions of the spinal cord which produce marked changes in sensation are rare. I have however been able to bring forward in this Thesis, and to publish for the first time, no less than twenty cases. The majority of these cases have occurred in the practice at the London Hospital, but for the remainder I am indebted to the kindness of Dr. Henry Head, who has been good enough to allow me to examine cases under his private care and to use the results in this Thesis. Of the twenty cases, eight are cases of "syringomyelia." These cases are all of long duration, and on some of them I have been able to make observations, more or less continuously, during the period of three years. In others I have had the great advantage of consulting Dr. Henry Head's observations upon them, made previously to my first examination; and, in one case, his observations extend back as far as the year 1899. I have had the opportunity of examining two cases of the special form of syringomyelia known as "Morvan's disease," where trophic changes are very marked. Four other cases are in all probability cases of central glioma of the spinal cord. In one of these cases, in which the symptoms lasted for one year, I was enabled to make a complete examination of the brain and spinal cord. In another case also I was able to examine the spinal cord. Five cases are instances of the so-called "Brown-Séquard symptom complex." Two of these latter cases were due to falls upon the back, the symptoms and signs being probably produced by hæmorrhage into one half of the spinal cord. In two other cases the onset occurred spontaneously and during sleep; the one in a powerful labourer who suffered from chronic rheumatic endocarditis; the other in a young married

woman, the onset being coincident with the first day of a menstrual period. The fifth and last case was due to a fracture of the cervical vertebræ. A case of syphilitic disease of the spinal cord, producing changes in sensation, was also studied.

The relative value of gross lesions of the spinal cord, as regards the determination of the paths of sensory impulses, varies considerably. Undoubtedly the cases most valuable for the determination of the paths of sensory impulses are those in which the spinal cord has been partially divided by a knife stab in the back. Unfortunately, for the purpose of this Thesis, these cases are uncommon in England, though a number of them have been carefully recorded in foreign literature. The cases due to a local hæmorrhage into the substance of the spinal cord, of sufficient extent to produce destruction of a limited area, are undoubtedly of great value. Cases of syringomyelia are also of great importance in the determination of the grouping of the various paths of sensory impulses within the spinal cord, and they are especially valuable because, in the greater number of cases, the disease spreads from the central canal of the cord towards its periphery.

Cases of rapidly growing tumour within the spinal cord should be carefully examined after death both macroscopically and microscopically. This is of importance because a rapidly growing infiltrating tumour may appear, on superficial examination, to have destroyed a certain portion of the spinal cord entirely, yet in that portion nerve fibres may be still intact.

In cases where limited portions of the spinal cord have been destroyed by tubercle or syphilis the same caution must be observed, for in many of these the pathological lesion is undoubtedly a diffuse one; and in interpreting the result of *post-mortem* examination such cases must be placed on a lower level of importance than those first described.

Summary of Cases Examined.

Syringomyelia	8 cases.
Morvan's Disease	2 cases.
Unilateral Lesions (Brown-Séquard type)	5 cases.
Tumours of the Spinal Cord	4 cases.
Syphilis of the Spinal Cord	1 case.

CHAPTER III.

The Effect of a Local Lesion of the Spinal Cord.

The effect of a local lesion of the spinal cord is twofold. Changes, both sensory and motor, are produced in the part of the body supplied by the segment or segments of the cord which have been destroyed by the lesion, and changes of a totally different character are produced in parts of the body supplied by the spinal cord below the lesion.

In reading the literature of the subject I have been struck by the inability of many authors to recognise this fundamental fact. This is not so noticeable where the lesion has been limited to one or two segments of the spinal cord, but it is especially seen in cases of syringomyelia in which the lesion extended for a considerable length of the cord.

Thus Mott (8) instances cases of syringomyelia as proving that sensory impulses pass up in the grey matter. But, as I shall show, this lesion affects the grey matter of the posterior horns and interrupts the fibres of the sensory path almost immediately after they have entered the spinal cord.

Consequently we must divide the effect of a lesion of the cord into:—

(A) The local effects.

(B) The effects upon the parts below the lesion.

(A) The *motor* changes in the part of the body supplied by the affected segment of the cord are due to implication of the anterior horn cells situated in this region. In cases of syringomyelia, or of tumours within the spinal cord, the anterior horn cells are slowly compressed. The muscles supplied by these cells slowly waste and become weak; but it is a curious fact, borne out by my cases, that electrical changes are not apparent until very late. The muscles seem to react normally to faradism and galvanism for a very considerable period after wasting is manifested. The normal polar reactions are preserved until very late, and it sometimes happens that the normal relations of these reactions are preserved until the muscles cease to react to any electrical stimulus at all. In some cases, however, where the destruction of the anterior horn cell had been more rapid, the reaction of degeneration was found. It would seem then, that pressure upon anterior horn cells, when applied gradually, does not alter the electrical reactions of the muscle supplied. It is only when the cell is completely and rapidly destroyed, or is itself the seat of a degenerative process, that the reaction of degeneration appears.

Another very remarkable feature in cases of syringomyelia is that the first muscles to waste are the small muscles of the hand. This cannot be due to the coincidence that the cavity in the spinal cord always attains its greatest dimensions in the first dorsal segment, from which the intrinsic hand muscles are supplied. Probably the explanation must be sought in the fact that the movements of the hand muscles are the most complex and the most lately developed in the body, and that therefore the anterior horn cells, which preside over them, are more susceptible to pressure than the other cells in the anterior horns.

The *sensory* changes produced in the part of the body supplied by the affected segment of the cord differ fundamentally from those produced in the parts below the lesion; hence the sensory changes in these cases can be used for the study of the paths of sensation only at the points where these enter the spinal cord. Thus in cases of syringomyelia the paths of sensation are interrupted as they enter the cord from the posterior roots. This is shown by the fact that the areas of skin affected correspond in their distribution to the areas supplied by the posterior spinal roots. These areas correspond with those found by Head to be affected in cases of Herpes Zoster, and with those which are found to be affected after division of the posterior spinal roots. It must be noted, however, that the extent of these areas differs considerably according as they are ascertained by different methods. Thus the area of loss of sensation which occurs when a given posterior root is divided does not represent the entire cutaneous distribution of that posterior root. For, as Sherrington has shown, there is a considerable overlapping of the skin areas supplied by different posterior roots. There is however another method by which these areas may be determined, known as the method of residual sensibility, and this method reveals the full cutaneous distribution of the divided root. When a number of roots have been destroyed above and below a given root, the area of skin which remains sensitive represents the greatest cutaneous distribution of the intact posterior root. In this Thesis however I am not so much concerned with the cutaneous distribution of sensation as with the qualitative changes which follow lesions of the spinal cord. It is these latter changes which are so important for the discovery of the paths of sensory impulses within the cord. However, I may say that the peripheral distribution of the posterior roots worked out by Head, Allen, Starr, and Thorburn, has been confirmed by the investigations made in the course of the researches undertaken in preparing this Thesis.

In syringomyelia the *boundaries* of the areas of loss of sensation are often indefinite. This is due to the fact that the unaffected segments of the cord above and below the lesion supply parts

of the skin within the boundary of loss of sensation. It was no uncommon occurrence to find that when a patient was tested after a lapse of three or four weeks, the area of anæsthesia had extended so as to correspond to a segment further, especially if the conditions for testing were less favourable, while if the general state of the patient was better, the area of anæsthesia might even correspond to one segment less than that obtained on a previous occasion. This "variable area" however never exceeded that supplied by one segment of the cord, and these results would agree with those of Sherrington on animals. He found that the overlap of skin sensibility rarely extended beyond that corresponding to the adjoining segments.

In certain positions the boundaries became very clearly defined. These well marked and sharply differentiated areas were bounded by the lines of fusion of posterior root areas and corresponded with the preaxial and postaxial borders of the arm and leg. Thus a sharp boundary line was found extending along the second rib outwards, and this was continued down the anterior surface of the arm. This line separates the cervical and thoracic areas: on the chest, above the line, lies the area supplied by the fourth cervical root. Below the line lies the area supplied by the third thoracic. A similar separating line is found behind, beginning at the first thoracic spine and passing outwards in a slightly curved manner down the posterior border of the arm.

For the lower limb these lines pass, the one from the first sacral spine outwards in a curved manner down the outer side of the leg; while the other begins in the groin and passing backwards runs down the inner and posterior aspect of the legs; these lines are marked in the following diagram.

The sensory nucleus of the fifth cranial nerve which supplies the face and the anterior part of the scalp seems to be continuous with those posterior horn cells which subserve sensory functions; for the area of loss of sensation has been observed to spread gradually upwards until the greater part of the face became analgesic.

(B) The motor effects in the parts below the lesion are due to implication of the pyramidal tract: this brings about a spastic condition on the same side of the body as the lesion in the spinal cord.

Sensory changes occur on both sides of the body below the lesion, for, as I shall show later, certain forms of sensory impulses are conducted up the spinal cord on the same side, while certain others cross the middle line soon after their entrance and pass up the cord on the opposite side.

The local and remote effects of a lesion within the spinal cord are very well illustrated by the case of J. F., in which, after having ample opportunities for making complete observations during life, I was enabled to obtain a *post-mortem* examination, and to make a complete macroscopical and microscopical study of the lesion in the spinal cord.

J. F., *aet.* 44, an actor and vocalist, was admitted to the London Hospital on July 6th, 1905, under the care of Dr. Dawson.

His illness commenced in November, 1904, when he noticed a slight weakness in the left arm. Soon afterwards his neck became stiff and he felt tingling sensations in the fingers of the left hand. The weakness in the left arm gradually increased, but he continued to play the piano with both hands until February, 1905. He was able to play the oboe, his favourite instrument, for a few weeks longer, but his left arm then became completely paralysed. During March and April, 1905, he became progressively weaker and his legs began to give way. He took to his bed fourteen days before his admission, and during this time he had had incontinence of urine on two occasions, and of fæces once, when his bowels were loose. His previous health had been excellent, and with the exception of a slight attack of gonorrhœa, twenty years before, he had never been ill. He had never suffered from syphilis. His condition on July 20th was as follows:—

No headache. No fits. Speech perfect. The left arm lay motionless on the bed by his side: measurements showed that the left forearm was half an inch smaller in circumference than the right forearm at a corresponding level. No voluntary movement took place in the left arm, and there was marked wasting of all the small muscles of the hand and also of the arm and forearm muscles. The electrical reactions of the muscles, though somewhat diminished both to faradism and galvanism, were normal. There was a good deal of pain when an attempt was made to flex the elbow joint beyond a right angle. He stated that he felt this pain both in the elbow and over the lower ribs on the left side. The skin of the fingers

was thin, white, and glossy, but the nails were normal. On the right side, movement was good and the hand grip fair, but there was some wasting of the small muscles of the hand.

Both legs were thin, but equally so. The leg muscles were flabby to the touch, and were distinctly spastic.

There was no fibrillary twitching in either arms or legs. The knee jerks were exaggerated and equal. The plantar reflex gave a distinct extensor response on both sides, and ankle clonus was easily obtainable. Co-ordination, as tested by his ability to touch a given object with the great toe, was good. The joints, skin, and nails of the lower limbs were normal. The neck was held rigidly extended and any attempt either to flex or to rotate the head was resisted, and seemed to cause the patient considerable pain. There was no pain, however, on jarring the head, and a radiograph of the cervical vertebræ showed that the bone was free from pathological change.

The cranial nerves were normal, the pupils being small but reacting normally to light, to shade, and to accommodation. Examination of the fundi revealed no change in either discs, retinae, or retinal vessels.

SENSATION.

There were no marked subjective sensations; the patient merely complained of some slight discomfort and tingling over the left lower ribs.

Light touch was perfect over the whole body and was extremely well localised.

Pressure was recognised over the whole body and was everywhere well localised, except over the left arm where its localisation was defective. Firm pressure on the left arm did not produce the sickening sensation that is usually produced by deep pressure.

The *sense of area*, as tested by applying discs of various sizes to the arms, was normal over the right arm, but on the left was very defective.

Compass Sensation. This was completely lost on the left hand, but on the right hand was normal.

The records, when the compasses were applied transversely, were :—

	Left hand. Ulnar side.	*Right hand.* Ulnar side.
At 2 cm. apart.	1. | IIII III III 2. | xx xxxx xxxx	2 cm. 1. | III IIII III 2. | IIII III III
3 cm.	All stimuli called "one"	3 cm. perfect.
1 cm.		1. | IxII III III 2. | IIII IxI III
5 cm.	All stimuli called "one."	Nearly perfect.
Absolute loss of power to distinguish the two points of the compass.		Normal condition.

The *sense of passive position* was absolutely lost in the left upper limb, and he was quite unable to indicate the position of his fingers in space or the direction in which they were being moved. He had also lost this sensation at the wrist, elbow, and shoulder joints absolutely. The contrast between right and left arm in this respect was most striking. The patient stated that he was quite unable to tell the position in space of his left arm, while as regards the right side his answers to similar questions were immediate and invariably accurate.

In the right great toe the sense of passive position was perfect, and also in the right ankle, knee, and hip; but in the left great toe this sensation was extremely defective. On testing this sensation at the left ankle and knee joints, however, his replies were usually accurate.

Sensibility to pain was lost on the right side below the fifth cervical area, and on the left side over the left upper forequarter and the adjacent skin of the neck and trunk.

A pin-prick or indeed any form of pressure over the sole of the foot caused a peculiar sensation. The patient stated that this was not a painful sensation, but was a very disagreeable one.

Cold sensation was lost over a very similar area to pain.

Heat sensation was lost over a slightly larger area; and over the right side of the neck, a test-tube at 50°C was called "cold." Over this area heat and cold were unrecognised whatever the temperature of the water in the test-tube.

Electrical sensation of a painful interrupted current was lost over an area exactly corresponding with the area in which cutaneous pain was lost. The painless interrupted current was recognised as "pins and needles" all over the body.

SUBSEQUENT PROGRESS.

The patient was vigorously treated with iodide and mercury. One drachm of mercury ointment was rubbed in daily, and 20 grains of potassium iodide were given three times a day. Under this treatment he was salivated twice, and had two attacks of diarrhœa, during which the treatment was temporarily omitted.

On August 5th, 1905, he seemed to be slightly drowsy, although he was quite intelligent and was able to give clear replies to questions, and to respond to cutaneous stimuli.

During the fortnight he had been steadily losing power in the right arm, and he was now unable to feed himself. He had complete incontinence of fæces and urine.

All forms of sensation were again tested, and a remarkable change was found to have occurred in the left arm.

Deep tactile sensibility was now completely lost over the left arm, and the sensation of light touch was very much diminished over this area. Thus, over the left arm no response was made; over the upper part of the left side of the chest, and over the right side of the body, the patient replied that he was being touched; while over the rest of the body he stated that the stimulus was a pin-prick, which hurt him.

On August 9th he was semi-comatose. He spoke in a whisper. Swallowing was difficult, and he could take liquids only. The other cranial nerves were unaffected. The following morning the right arm was found to be completely paralysed, and in the evening the patient suddenly died.

A *post-mortem* examination was performed on August 11th, 1905 (twenty-four hours after death).

The body weighed 6 stone 11 pounds; rigor mortis was still present. The pupils were equal, and of moderate size.

In the larynx the vocal cords were found in the usual cadaveric position. The tongue, tonsils, and thyroid were normal, and no trace of the thymus gland was found.

The pleuræ contained no liquid and showed no adhesions.

The lungs were aërated throughout. There was some emphysema. There was great engorgement and œdema of the lung bases, and muco-purulent secretion was found in the trachea and larger bronchioles.

The pericardium and heart (7½ ounces) were normal. The coronary arteries were patent, but the aorta showed scattered patches of atheroma all along its course.

The abdominal cavity showed no adhesions and no excess of liquid. The spleen weighed three-and-a-half ounces, was small, and showed wrinkling of the capsule.

The kidneys weighed five ounces each, and presented no pathological appearances.

The bladder and testicles were healthy, as were also the stomach and intestines.

The liver presented no abnormality except the presence of a small Riedel's lobe.

The brain and spinal cord were removed together by sawing through the occipital bone down to the foramen magnum on either side. After removal of the posterior parts of the vertebræ, these bones were examined and were found to be perfectly healthy.

On dissecting away the cauda equina a large quantity of clear cerebro-spinal liquid escaped, and the spinal cord which had previously been much distended became somewhat smaller. In the cervical region the circumference of the cord was greater than normal, and this increase in size extended up to the bulb. The cord and brain were removed together, a considerable quantity of clear liquid escaping when the dura covering the cerebellum was opened. The cerebral hemispheres were separated by cutting through the crura, and the meninges were seen to be healthy. The cerebral convolutions were flattened and the lateral ventricles contained an excess of liquid, their walls having a "sanded" appearance.

The pituitary body was firm, white, and slightly larger than normal.

The spinal cord, pons, and cerebellum were hardened in formol for six days, and were then placed in Müller's fluid or two months. Sections were then made, commencing at the crura and passing down the cord.

A section through the pons showed marked dilatation of the aqueduct of Sylvius, which measured 10×7.5 mm.

Section at the region of the olivary nucleus showed some increase in the gelatinous material round the central canal. This was much more marked in a section at the level of the decussation of the pyramids. Between Cervical 1 and 2, the section showed a clearly-defined round tumour in the centre of the spinal cord. At the level of C 3, the whole of the left side of the cord was occupied by a large tumour mass, with a yellow area in its centre. Similar appearances were seen at the level of each root down to C 7, where the tumour, though still lying to the left, occupied a more central position. Below this the tumour became much smaller, occupying the grey matter around the central canal, and disappearing at the level of D 7. Below this the cord appeared normal to the naked eye.

On microscopical examination, the tumour was found to be a gliosarcoma. The tumour consisted of cells with a large deeply staining nucleus and a small amount of protoplasm. Heidenheim's iron-hæmatoxylin method showed that many of these cells possessed processes which interlaced.

At the level of the third cervical segment the tumour attained its greatest dimensions.

The following parts of the cord were destroyed at this level:—

(1) The whole of the left side of the cord with the exception of the left anterior column.

(2) The right posterior column was compressed.

Sections through the pons, medulla, and cervical cord above the tumour showed ascending degeneration in:—

(1) Left antero-lateral tract of Gowers.

(2) Left direct cerebellar tract.

(3) Left posterior column, and

(4) Some degeneration in the right posterior column.

Below the lesion Weigert's method showed a very marked degeneration in the left pyramidal tract.

In this case a glioma was found involving the left half of the spinal cord in the cervical region between the second and fifth segments. The local effects produced by this lesion were both motor and sensory, and were limited to the left arm.

Wasting, weakness, and finally total flaccid paralysis occurred in the left arm. The electrical reactions to both faradism and galvanism were normal, though slightly diminished.

The sensory changes corresponding with the level of the lesions were also found in the left arm and the left side of the neck. When the patient was first observed, these changes consisted of loss of painful and thermal sensibility, and of the muscular sense. Later, tactile sensation was also destroyed almost completely, leaving the left arm totally anæsthetic.

The motor effects on the parts below the lesion consisted of a spastic paralysis of the left leg, and, to a less extent, of the right leg also. The deep reflexes were increased, ankle clonus was obtained, and the plantar response was of the extensor type. Loss of sensation below the lesion occurred in both legs. On the right side below the lesion there was total analgesia and thermo-anæsthesia, while the muscular sense was normal. On the left side the muscular sense alone was affected. There was no loss of tactile sensibility in either leg.

(c) *The effects of extension of the lesion.*

When an intramedullary lesion is progressive the area of skin over which sensation is lost gradually increases. This may occur in two different ways. In one—the more usual method—successive posterior roots, both above and below the lesion, are destroyed as they enter the spinal cord, and the area of skin over which sensation is lost increases in size by the addition of successive segments of the skin corresponding to posterior roots. In the other—a far rarer method—a lesion high up in the cervical region or in the pons in its spread gradually encroaches upon the paths transmitting sensory impulses from below, so that the anæsthetic areas increase in size in a totally different manner from that described in the first method.

An excellent example of the first method of spread is furnished in the case of F. A. R., *aet.* 17, who in 1899 began to be troubled with pains in her left shoulder, and weakness of the left hand. On October 16th, 1899, there was distinct wasting of the small muscles of the left hand, and some osteo-arthritis of the left shoulder. On the radial side of the left forearm, extending downwards, and including the radial half of the palm and the base of the thumb, was an area where neither heat nor cold could be felt. This corresponded to the minimum area of supply of the seventh cervical posterior root, that is to say, the area over which there is no overlap from adjoining nerves. On March 15th, 1902, the weakness and wasting of the left hand had increased, and there was some atrophy of the intrinsic muscles of the right hand also. The area of loss of sensation to heat and cold remained as before, but now over this area the sense of pain was also diminished. Tactile and muscular sensibility were normal. On June 19th, 1903, the area of loss of sensation to cold was found to have considerably increased. On the left side the eighth, seventh, sixth, fifth, and fourth cervical areas had become affected; while on the right side the seventh and eighth cervical, and the first thoracic areas, had become involved. On September 28th, 1903, on the left side all the cervical roots, with the exception of the first and second, were involved, while on the right side the loss of sensation had affected the fifth and sixth cervical roots. On February 29th, 1904, the condition had again slightly advanced on both sides of the body. On February 27th, 1905, the area over which the sensation of coldness was lost included on the left side all the cervical areas, and also a part of the area of the distribution of the fifth cranial nerve upon the left cheek. On the right side all the cervical root areas were involved, but the fifth cranial nerve had not been encroached upon.

In the second and much rarer method of spread, a lesion apparently in the pons, but also probably extending down the length of the cervical cord, cuts off the paths of afferent impulses from below. This method is illustrated by the case of M. B., *aet.* 29, who was first seen on February 3rd, 1902.

In this case heat and cold sensibility were lost over the whole of the right forequarter and the right side of the face and head. This shows that the lesion must have included the entire fifth root on the right side. The same loss of sensation was also present on parts of the left arm and the left side of the neck, and, together with impairment on the left thigh, on the inner side of the left leg and on both feet. A similar condition was found on February 24th, 1902. She was not seen again until April, 1905, when there was total analgesia and thermo-anæsthesia, while the lightest touch was well appreciated and perfectly localised. The only explanation that can be offered for such a remarkably rapid increase in the loss of sensation is that a lesion in the pons had rapidly spread and involved the sensory path.

The resemblance of this case to certain cases of hysteria is striking, but there were definite signs of organic disease in the patient. She had much wasting of the small muscles of both hands, and trophic changes in many of the joints. The legs were extremely spastic, and showed increased reflexes and extensor plantar response. She had often burnt and cut herself at her work as a cook, without remarking the injury, and there were extensive scars on both her arms.

A similar case of syringomyelia, with total analgesia and thermo-anæsthesia, is recorded by Charcot (9).

(D) *The segmental representation of the parts below at the level of the lesion.*

It is well known that a sacral or lumbar posterior root can be traced upwards in the posterior columns as a definite bundle of fibres into the cervical region of the cord. This bundle, too, always occupies a definite position in the posterior columns. It is, therefore, not impossible that a sharply defined lesion of this portion of the spinal cord might produce a segmental loss of sensibility corresponding to that caused by destruction of a certain number of sacral or lumbar roots. These tracts are, however, merely the central processes of peripheral nerves, and the loss of sensation would resemble that found when posterior roots have been divided. The question of the segmental arrangement of those afferent fibres within the spinal cord which pick up impulses at the first relay can be studied only in those cases where a high local lesion, for example, in the cervical region, has produced a loss of sensation at some distance below the point of injury. In a later portion of this Thesis, it will be shown that there are good reasons for concluding that the fibres conveying thermal and painful sensory impulses, almost immediately after their entrance into the spinal cord, arborize around cells in the posterior horn, the axons of which carry up these impulses to the brain. These axons rapidly pass over to the opposite side of the spinal cord.

A case of great interest in this connexion is that of C. F., *aet.* 26.

On August 14th, 1905, he dived from the top of a bathing machine at Westgate. His feet slipped, and he fell head foremost into the water. He became unconscious, and on recovery found that his right arm was weak, and that he could not feel on his left side. When examined on November 20th, 1905, the condition had greatly improved, but there was still some weakness in the right hand and arm.

Sensibility to heat and cold was lost over the entire left half of the body below the fifth thoracic segment, but the pain of a pin-prick was only slightly diminished over the greater part of this area. Over the area of the lumbar posterior roots, total analgesia was present.

The distribution of the analgesia is shown in the annexed diagram. In this condition a lesion in the upper thoracic segments of the cord had produced a weakness of the right hand and arm, while there was profound analgesia only over the area of distribution of the lumbar posterior roots of the left side.

In this case it would seem that, at the level of the local lesion on the right side of the cord (upper thoracic and lower cervical), the lumbar segments of the cord were represented. In other words, there is reason to infer that the fibres of the secondary sensory neurons are arranged segmentally.

We have therefore some evidence for supposing that the fibres in the tract conveying the secondary sensory neurons for pain, heat, and cold up the spinal cord, are arranged in the tract according to the parts of the body which they represent.

CHAPTER IV.

The Diagnosis of a Central Spinal Cord Lesion.

The fact that until the last twenty-five years cases of syringomyelia were regarded as peculiar hysterical manifestations, will illustrate the difficulty attending the diagnosis between these conditions. The clinical manifestations of syringomyelia may for a long time consist merely of certain sensory changes. This loss of sensation may also be found in hysterical subjects. The trophic changes which so often occur in syringomyelia, such as local œdema, ulceration, and mutilation, may also be imitated to a remarkable degree in hysteria. Paralysis of muscles may occur in both diseases. It is, therefore, in certain cases a matter of some difficulty to distinguish between the two conditions. The difference in the sensory symptoms must first be considered.

In hysteria the onset is sudden, and recovery of sensation may also occur suddenly.

In syringomyelia the onset of the sensory changes is gradual, as was well shown in the case of F. A. R., described in the last chapter. The sensory changes become gradually more marked, and spread over a larger and larger area of skin; but as was mentioned there may be remissions, and when the condition of the patient improves the area over which sensibility is lost may appear to be slightly smaller than on a previous occasion.

In hysteria, as a rule, all forms of sensation are lost together. The patient feels neither heat nor cold, neither pain nor touch, and has no "feeling" in the limb at all. But in France certain undoubted cases of hysteria have occurred in which thermo-anæsthesia and analgesia were alone present. Such a case is recorded by Charcot (10). Here the patient was very excitable and suffered from vertigo and noises in the head. A loss of power occurred in the right hand, the onset being sudden during sleep and the recovery rapid soon afterwards. There was a violet œdema of the back of the hand, and a total loss of pain and temperature sensibility of the fingers and hand and the lower fifth of the forearm, the upper border of the area extending circularly round the arm. A similar condition had existed in this patient once before, except that on that occasion the anæsthesia had included all forms of sensation. That condition had disappeared suddenly.

Here the "glove" distribution of the anæsthesia, the sudden onset and equally sudden disappearance of the paralysis and sensory changes, determined the diagnosis of hysteria. Consequently, although in the majority of cases the partial sensory changes (loss of thermal and painful sensibility, tactile sensibility being present) are sufficiently characteristic of a central spinal lesion, yet hysterical patients, especially those at whose bedside the differential diagnosis has been discussed, may also show this sensory dissociation.

In hysteria, moreover, the anæsthesia has extremely sharp borders, while in syringomyelia the borders, except in the region where distant posterior segments are in contact, are often rather ill-defined. Again in syringomyelia sensibility to heat may be lost over an area differing from that of sensibility to cold or to painful stimuli. The distribution of the anæsthesia is also very characteristic. In hysteria the anæsthesia is limited to the "glove" area, to the hand and forearm, or to a limb, and the upper limit is always a complete circle round the limb. Unilateral and bilateral analgesia and thermo-anæsthesia may occur in both conditions, but are much more common in hysteria.

In syringomyelia and other central cord lesions the area of anæsthesia has a segmental distribution, and corresponds to a greater or less extent with the cutaneous areas supplied by the posterior roots.

The motor symptoms in hysteria consist of sudden paralysis of a limb, or of part of a limb. It is often possible, by persuading the patient to exert muscular force against resistance, to show that there is no actual loss of power. The paralysis disappears as suddenly as it appears. Muscular contracture is also more common in hysteria than in syringomyelia.

In syringomyelia on the other hand there is a slowly progressive wasting of muscles supplied from the cervical region, usually the small muscles of the hand. If the cavity formation has been extensive enough to lead to pressure on the pyramidal tracts there will be spastic paralysis of one or both legs. In both conditions the knee jerks are likely to be increased, but in syringomyelia this increase will be accompanied by true ankle clonus and by the extensor plantar response.

In hysteria the superficial skin reflexes are likely to be increased, while in syringomyelia they will be diminished both over the anæsthetic area and in the leg below this area.

Such are the main distinguishing features between hysteria and syringomyelia. The difficulty in diagnosis between the two conditions is well illustrated by the case of the following patient, who was first seen on May 24th, 1905.

T. S., *æt.* 26, a carman, with no address and no friends, had been ill for six years. One day he cut his right hand on an iron spike. He recovered from this, but four or five months afterwards he suddenly lost power in the right hand and arm, and the right hand became swollen, blue, and numb. Two months later the left hand also began to swell, but he had never lost power in this hand. His lower jaw was fractured when he was 16 years old. He was a typical degenerate in appearance, and was exceedingly troublesome in the hospital wards. His speech was somewhat lisping, but this was possibly accounted for by the deformity of his jaw. His lower jaw projected forward markedly, and there was a scar under the chin, where an operation was performed at the time of the fracture, ten years ago. The right hand was swollen, limp, blue, and cold, and the capillary circulation returned very slowly. The greatest swelling was on the dorsum of the hand, but this scarcely pitted at all on pressure. On the centre of the dorsum was a black scab. The initial lesion at the site of the scab—so the patient stated—had burst, and yellow matter had come away. There were similar scars on the dorsum of the middle and ring fingers at the proximal interphalangeal joints. The circumference of the right middle finger was $1\frac{1}{4}$ in. greater than that of the left middle finger at the proximal interphalangeal joint. On the dorsum of the left hand there was also a swelling with a black scab in the centre, almost exactly similar to that on the right hand. The right hand could not grasp, and the flexors and extensors of the wrist seemed to be quite powerless. All the muscles of the left hand acted well. The legs were perfectly normal, as were the reflexes and the cranial nerves All forms of sensation were lost over the right hand and forearm, the upper limit being a complete circle just above the elbow joint. Neither pin-prick, thermal stimuli, nor touch could be appreciated; he stated that he could not tell which way his fingers were being moved. Over the left hand and arm sensation was normal.

In January, 1906, the patient again became an in-patient of the hospital. The swelling of both hands had greatly diminished, and the sores on the backs of the hands had healed. Sensation and motion had been suddenly recovered in the right hand some months before, but a few days previous to coming into the hospital the left arm had become completely paralysed, and there was a total loss of all forms of sensation over the whole of the left arm, ending above at a very definite border which extended circularly around the shoulder.

In this case the sudden onset of the paralysis and anæsthesia and their equally sudden disappearance were greatly in favour of hysteria. The trophic changes in the hands were however so marked that at first organic disease was suspected. Greatly in favour of the functional character of the case was the total loss of all forms of sensation and the limitation of the anæsthesia to a limb or a segment of a limb.

The diagnosis of a lesion of the spinal cord from a lesion of the peripheral nerves is not difficult when the lesion of the latter occurs outside the spinal cord, since in these cases the history of trauma or the indication of the point of entry of a knife assist in the diagnosis. It is very different, however, when the diagnosis has to be made between a lesion of the spinal cord and a lesion of the nerve roots as they enter the spinal cord. This has been practically impossible, at any rate from the sensory side, until quite recently, for the changes in sensation produced by lesions of the peripheral nerves were unknown. The recent work of Head, Sherren, and Rivers has, however, thrown a new light upon the afferent sensory nerves, and the following chapter will be devoted to a *résumé* of their recent work.

CHAPTER V.

The Grouping of Sensory Paths in the Peripheral Nerves.

Before considering the grouping of the paths of sensory impulses in the spinal cord, it is essential that the arrangement in the posterior roots, as these paths are entering the cord, should be clearly understood. This arrangement of afferent impulses in the peripheral nerves has been fully investigated by Head and Sherren, who have shown that in the peripheral nerves a certain definite grouping of sensory paths can be observed.

These authors consider that sensory impulses are conducted along the peripheral sensory nerves in three great groups, which they distinguish as those of protopathic, epicritic, and deep sensibility.

Included in the protopathic group of sensory impulses are the impulses conveying pain, the extremes of heat and cold, and painful interrupted electric currents. The essential characteristics of this group of sensory impulses are as follows:—

Every stimulus to which the part reacted produced a sensation that radiated widely, and was accompanied by a peculiar tingling quality. The power of localising the specific stimulus was extremely defective, and the sensation either seemed to be situated in some area of normal sensibility, remote from the point of the application of the stimulus, or to extend widely around the point stimulated.

The occurrence of atrophic changes in the skin was found to be intimately associated with disturbances of protopathic sensibility. It was found that ulceration and blister formation tended to

appear over those portions of the skin where protopathic sensibility was absent and to disappear when it returned.

Further it was found, after a peripheral sensory nerve had been completely divided and protopathic sensibility had become lost, that this form of sensibility tended to return more quickly than other forms to be presently mentioned. Under the most favourable conditions of primary nerve suture, protopathic sensibility began to be regained in about sixty days, while it was completely restored in about two hundred days.

This form was also found to differ in its distribution from the epicritic form of sensibility, to be presently mentioned. The areas of skin supplied by peripheral nerves with protopathic sensibility were found to overlap considerably on the extreme periphery of the limbs, while as one passed nearer to the trunk this overlap became less and less marked. Thus while complete section of the ulnar nerve produced a loss of protopathic sensibility of a very much smaller area of skin than that over which epicritic sensibility was destroyed, injury of the brachial plexus produced a loss of epicritic and protopathic sensation over the same area of skin. Division of the posterior roots of the spinal nerves produced loss of protopathic sensibility over an even greater skin area than that over which epicritic sensibility was destroyed.

The epicritic sensibility included those sensations produced by light cotton wool touch and the painless interrupted current, the recognition of the two points of the compasses, and the discrimination of the minor degrees of heat and cold. The sense of area and the perception of the form of objects were also found to belong to this group. This form of sensibility is characterized by the accuracy of its localisation. The point of application on the skin of the stimuli included in this group was very correctly localised by the patient, and there was no sign of the peculiar radiation characteristic of protopathic sensibility.

This form of sensibility was not restored until a date much later than that at which protopathic sensibility was regained. It was found that epicritic sensibility did not begin to return until two hundred days had elapsed from the date of the primary nerve suture, *i.e.*, as a rule not until protopathic sensibility had been entirely regained. This form of sensation also took longer to return completely than did protopathic sensibility. It did not completely re-appear until from 350 to 450 days after the primary nerve suture, and cases were observed in which epicritic sensibility had continued to be absent even as long as five years after the accident.

The distribution of this epicritic sensibility was also very different from that of protopathic sensibility. Thus the areas of epicritic sensibility scarcely overlapped at all on the hands and feet, while the overlap of the skin areas supplied by the different peripheral sensory nerves became greater and greater as one passed up the limbs. So much was this the case, that while as a result of division of a nerve trunk at the wrist epicritic sensibility was lost over a much larger area than protopathic sensibility, division of the posterior roots caused a far greater loss of the latter form of sensibility.

The last of the three great groups of peripheral impulses was that of deep sensibility. This is the residual sensibility which was discovered when the cutaneous sensory nerves had been completely divided and all cutaneous sensation was lost. It included sensibility to pressure, which could be localised to a really remarkable degree, even when all cutaneous sensation was abolished. The sickening sensation produced by a heavy crush was also included under deep sensibility, and will be spoken of as the "pain of deep pressure." The muscular sense and all that it implies were included, together with the sense of passive position at the various joints.

These sensations were found to be unaltered when the cutaneous sensory nerves had all been divided, and it is therefore clear that the impulses must run up mainly in the motor nerves to the

muscles, and from the fingers along the tendons passing to them. Thus division of the median and ulnar nerves at the wrist produced little loss of deep sensibility; but if the tendons passing under the annular ligament were also severed there was a profound loss, the fingers became insensitive to pressure, and there was no recognition on the part of the patient of their position in space.

The actual facts upon which these statements were based included the results of sensation testing upon a number of cases in which the median and ulnar nerves had been divided, either together or separately. It was found that, if no tendons had been divided, deep sensibility was intact; that is to say, pressure was well felt and localised, the pain of deep pressure was present, and the perception of the position in space of the fingers was retained. Over the fingers and a smaller or larger portion of the adjoining part of the hand, both protopathic and epicritic sensibility were lost; that is to say, over this area there was no perception of any stimulus, except when deep sensibility was aroused. Over the remaining portion of the territory assigned by anatomy to the nerve in question epicritic sensibility was lost, while protopathic sensibility was retained. That is to say, over this area a pin-prick caused pain, while water below 20°C, was called "ice-cold." It mattered little whether water at 18° or ice was used; both were regarded as ice-cold and were not distinguished the one from the other. Water above 45°C was recognised as hot or burning. On the other hand test-tubes filled with water between 20°C and 40°C produced no sensation of either heat or cold. Moreover, a test-tube filled with water at 38°C could not be distinguished from one filled with water at 25°C, although on the normal skin these test-tubes were readily recognised as "warm" and "cool" respectively. Over this area also, the patient was unable to recognise correctly the two points of a pair of compasses when simultaneously applied to the skin of the hand at distances apart which could be with certainty discerned on normal skin. The touch of cotton wool, lightly applied to such an area, was also unrecognised, whereas as soon as the boundary of the insensitive skin was passed, the patient declared that he felt that he was being lightly touched.

An excellent opportunity for studying protopathic sensibility was afforded by testing the sensibility after the first stage of recovery (namely, the return of protopathic sensibility) had taken place. It was then found that application of a pin-prick, or of ice, or of water above 50°C, caused a sensation even more vivid and unpleasant than that produced by similar stimuli upon the normal skin. The stimuli caused a sensation characterized by radiation over a considerable area of skin, and by a peculiar unpleasant tingling which was always complained of by the patient, and which frequently lasted for some time after the stimulus was withdrawn. So far Head and Sherren had been able to study skin areas upon which protopathic sensibility was present and epicritic sensibility absent. Could an area be found upon which epicritic sensibility was present while protopathic sensibility was abolished? By a fortunate chance it was found that, in two cases in which the radial and external cutaneous nerves had been divided, an area of protopathic loss overlapped the boundary of the loss of epicritic sensibility. Over this area a remarkable condition of sensibility was found; a light cotton wool touch was perfectly felt and localised, but the pain of a pin-prick was entirely absent, while to temperature stimuli a very remarkable response was obtained. Thus the patient declared that a test-tube filled with water at 35°C appeared to him to be distinctly warm, while a test-tube filled with water at 50°C produced no sensation of heat at all. If the two test tubes were applied successively it was found that the test-tube at 38°C appeared to be hotter than that at 50°C. When test-tubes filled with ice and with water at 25°C were used, it was found that the former produced no sensation of cold, while the latter was at once recognised as cool. Applied successively to this area the test-tube at 25°C was declared to be cooler than that containing ice. Moreover, when test-tubes containing water at 34°C and 24°C were applied successively to this area they were readily recognised as warm and cool respectively.

Thus it was shown that not only might protopathic sensibility and epicritic sensibility be lost over an area of skin, but also that protopathic sensibility might be retained while epicritic sensibility was absent, and that on other skin areas epicritic sensibility might be present while protopathic was destroyed.

Charles E. Ham (11), working in conjunction with Dr. Head, has published a thesis for the degree of M.D., London, in which the histology of the peripheral nerves is re-examined, in the light of Head's work on the peripheral nervous system. His conclusions are briefly as follows:—

(1) That no new formation of either axis cylinder or medulla takes place in an afferent nerve which has been completely severed from its central connexions and has not been allowed to unite with either its own central end or with neighbouring nerves. The work of earlier observers was vitiated by the fact that they omitted to take precautions to avoid the latter possibility.

(2) That in the normal radial nerve of a cat (a purely afferent nerve) two groups of nerve fibres can be recognised. The bulk of the fibres measure 12—15 μ in diameter; they contain a medulla staining dark-blue with Weigert's method, and show well-marked sausage-shaped nodes of Ranvier. In addition to these there are others, 6—9 μ in diameter, which differ from the above in containing less medulla.

(3) When a nerve is divided and at once sutured, it is found that after about fifty days small axis cylinders with medulla around them have begun to develop; and it would appear that these are laid down in the peripheral part of the divided nerve, which has been stimulated to this action by the approximation of the divided ends. After a period of 180 days, the regeneration of these small fibres (6—8 μ) is complete. It will thus be seen that the re-appearance of these fibres 6—8 μ in diameter is coincident with the return of protopathic sensibility.

(4) It was found that about 200 days after the suture there was a fresh growth of small ill-developed fibres, which even in nerves that had been sutured 412 days previously had only slightly increased in size and development.

It will be recognised that these new fibres make their re-appearance about the time when epicritic sensibility returns.

(5) By studying the nerves in the superficial plexus in the palm, it was found that after division of one of the nerve trunks entering into its formation, normal nerves were present among the degenerating fibres, and these nerves were found to consist of fibres measuring 6—8 μ in diameter, while no fibres of 12—15 μ in diameter were found.

This would lead to the conclusion that in the palm there is considerable overlapping of the system of fibres measuring 6—8 μ in diameter, while in the system of fibres of 12—15 μ in diameter no such overlap is observed. This clearly corresponds to the relative overlap in the palm of protopathic and epicritic sensibility.

Thus both clinical and histological evidence points to the conclusion that in a sensory cutaneous afferent nerve there are two systems of fibres, along which certain groups of sensory impulses are transmitted to the spinal cord.

As before mentioned, deep sensibility runs towards the spinal cord mainly along the afferent fibres which accompany motor nerves. The existence of afferent fibres in the motor nerves was demonstrated by Sherrington in 1894 (12). He found that if the nerve twig to the vastus externus was examined after all the ventral spinal roots and the spinal ganglia had been removed, the nerve was completely degenerated. If, however, the spinal ganglia were left intact when the ventral spinal roots were divided, a certain number of healthy well-myelinated nerve fibres remained three months after the division. As regards the distribution of these fibres, there must be very considerable

overlapping. Thus, in a case recorded by Head and Sherren (5, Case 53), in which the fifth, sixth, and seventh cervical posterior spinal roots had been divided, deep sensibility was lost over an area which was certainly not larger than the hand below the wrist. Protopathic and epicritic sensibility were, however, lost over the hand, forearm, and greater part of the upper arm.

In this connexion Anderson's (13) observations upon the date of myelination of the afferent fibres are of great interest. He has shown that both somatic and splanchnic myelinated nerves consist of two distinct sets of fibres, which develop their myelin sheath at different dates. Taking the myelinated splanchnic fibres, he finds that there is one set which terminates in the Pacinian corpuscles of the abdominal viscera and larynx. He finds that these large myelinated fibres develop their myelin sheath simultaneously with the large myelinated fibres which end in the Pacinian corpuscles of the skin and also in the spindles and other end organs which lie in the muscles, tendons, and joints. This set of fibres, which may be named the "great Pacinian system," probably consists of the fibres of deep sensibility. A set of smaller afferent nerves myelinate at a later date than the former set, but simultaneously in the viscera and in the skin, where they end in the epithelial structures.

As the body is almost entirely covered by hairs it is of great importance to know with what forms of sensation these structures are associated. Head and Sherren have found that they react to both epicritic and protopathic stimulation. Thus over an area of loss of epicritic sensibility only, the hairs reacted to cotton wool touch, and the stimulus evoked a curious radiating sensation with a characteristic tingling quality. When the skin was shaved it was quite insensitive to cotton wool. It was found too that when protopathic sensibility had returned, while epicritic was still defective, the plucking out of the hair caused pain.

As regards the condition of the hairs when protopathic sensibility was absent, while epicritic was present, they found that stimulation of the hairs was exactly localised but plucking of the hair caused no pain.

The power of detecting the movements of hairs, and of localising the point at which the stimulus is applied, may be present in cases where the skin is insensitive to all forms of temperature, and even when, after shaving, it becomes entirely anæsthetic to those forms of stimulation which do not involve deep sensibility. The hair may be pulled out perpendicularly to the skin, without evoking any response, even when deep sensibility is present.

Thus on the periphery sensibility is divided into three great groups :—

(a) *Protopathic* ..... Pain.
Heat over 40°C.
Cold below 20°C.

This form is also concerned with the trophic regulation of the skin. It is the form of sensation possessed by the viscera, and on the skin represents the afferent somatic fibres of the sympathetic.

(b) *Epicritic* ... ... Heat below 40°C.
Cold above 20°C.
Light touch.
"Compass sensation."
Sense of form and area.
Localisation of light touch.
Acuæsthesia.

(c) *Deep Sensibility* ... Pain of deep pressure.
Pressure.
Muscular sense.
Sense of passive position.
Localisation of pressure.

The hairs appear to be supplied with both protopathic and epicritic sensibility.

CHAPTER VI.

The Grouping of Sensory Paths within the Spinal Cord.

In cases of central lesions of the spinal cord, sensation is affected, and it has been found that certain forms of sensation tend to be lost together.

Thus when the sensation of pain is lost in consequence of the lesion of the spinal cord, it is generally associated with a loss of sensibility to heat and cold.

Sensations of Pain, of Heat, and of Cold.

The first group of sensations to be considered includes those of pain, heat, and cold.

In syringomyelia it is almost always these three forms of sensation which are lost, while tactile sensibility both to cotton-wool touch and to pressure is retained. The sense of passive position, the muscular sense, and the ability to distinguish the two points of a pair of compasses, are likewise unimpaired.

These facts are well illustrated by the case of Thomas B., *aet.* 48, who came to the London Hospital on November 8th, 1905, complaining of a swelling in his left elbow.

For the last two years he had noticed wasting of the hands. It first began in his right hand two years ago. He was able to do his work as a porter, so he did not continue to attend. Soon afterwards his left hand began to waste. For the last three years his hands had always felt cold, even when he was in front of a large fire. So much was this the case that as soon as he got home he would go to bed, as he found that when he had been in bed an hour his hands became warm. He had never burnt or hurt himself. He dated his illness from a fall on to his head, which occurred at the People's Palace three years ago.

He had been six years in the army, and five years in India, going through the Afghan War in 1879 and 1880. After leaving the army he had worked at the docks until three years ago, when he obtained a place as a porter in a sugar factory.

He had been married twenty-four years, had had thirteen children, and had never suffered from syphilis or gonorrhœa. He was a small cheerful-looking man, and very intelligent. His speech was normal, and he had never suffered from fits. His gait was normal, and co-ordination in the legs was good. The knee jerks were equal, and there was no ankle clonus. The plantar reflex was normal. Sensation was perfectly normal over both legs. The small muscles of the right hand were much wasted. The thenar and hypothenar eminences were very small, and the thumb was extended and lay in the same plane as the palm. The interossei were profoundly wasted. The proximal phalanges of the fingers were over-extended at the metacarpo-phalangeal joints and the two terminal phalanges were flexed.

Over the outer part of the right thenar eminence no contraction could be felt when he attempted to make the movements of abduction and opposition to the thumb, and true abduction and opposition were impossible. Interosseous movement could not be obtained. The abductor indicis and abductor minimi digiti were completely paralysed. All movements at and above the wrist were perfect. The long flexors and extensors of the arm acted well.

In the left hand, the greatest wasting was found in the outer thenar group, but the hand as a whole was not much wasted. The interossei could be thrown into action, and opposition and abduction of the thumb could be performed. The muscles of the left arm and forearm were normal. In the right hand the outer thenar group did not react even to strong faradism, but to the galvanic current they reacted definitely, the response to the cathodal closing current being slightly greater than to the anodal closing current. The first dorsal interosseus reacted in the same manner.

The other muscles of the body reacted normally.

Light touch was normal and perfectly localised. The palms of the hands were thick and horny, and over them he neither appreciated cotton wool touch, nor even touches with the finger.

Deep touch or pressure was perfectly recognised everywhere and extremely well localised.

The compass test over both hands at 2 cm. apart produced normal response.

The sense of passive position was normal.

The sense of area was perfect in both arms and forearms; he was able to recognise correctly areas of 2 cm., 1 cm., and 2 mm. as "large," "smaller," and "very small," when applied indiscriminately.

Acuæsthesia was perfectly preserved in both hands and forearms. The point of a pin was described as like a needle point and the head as blunter. Neither point nor head caused pain.

The sensation of "pins and needles" produced by an electric current was normal over the arm, but even a strong faradic current caused him no pain. A similar current over the legs caused him instantly to jump and cry out that he was hurt. There was complete analgesia over both arms, over the trunk as far down as the level of the umbilicus, and over the head and neck, with the exception of the face.

Crushing pressure applied over the ulna caused him no sensation of pain or unpleasantness.

Sensibility to heat and to cold was lost over an exactly similar area. The boundaries of this area were remarkably sharp, and as soon as the limit was passed the patient at once recognised the nature of the stimulus, and when pricked drew himself away reflexly.

Moreover, upon this area he was unable to recognise heat or cold, whatever the temperature of the test-tube. Water at 70° C, 50° C, 30° C, 18° C, and ice, were all said by the patient to be "touches."

Occasionally, however, when he knew he was being tested for the appreciation of temperature, he said that the test-tube was "cold." It will be remembered that over the hands and arms he had intense subjective sensations of cold. He could always be puzzled however over the above area, but as soon as its limits were reached he at once recognised all degrees of temperature in an extremely accurate manner.

The fundi were normal; the pupils were equal and reacted both to light and accommodation. The other cranial nerves were unaffected.

The sphincters were acting perfectly.

There was a slight degree of scoliosis. The left elbow was disorganised, and there was much hypertrophic outgrowth from the bones and synovial exudation into the joint. Creaking could be felt on moving it. These changes were clearly shown in the radiograph, which also showed the peculiar opacity of the synovial liquid, so commonly found in trophic joints, and said to be due to the excess of calcium salts which such effusions contain. The radiograph of the right elbow also shows some slight bony changes.

Summary. A case of syringomyelia of three years' duration. Wasting of the small muscles of both hands, especially the right. Loss of the sensations of pain, heat, and cold over both forequarters. Other forms of sensation normal. Trophic joint changes.

The results obtained in the other eight cases of syringomyelia which I had the opportunity of examining may be summarised as follows:—

NAME.	AGE.	PAIN.	LOSS TO COLD.	HEAT.	OTHER SENSATIONS.
W. H.	30.	Right forequarter.	Right forequarter and right side of neck.	Same area as cold.	Nil.
C. H.	30.	Right forequarter.	Right forequarter and right side of neck.	Similar but larger areas than cold.	Nil.
F. A. R.	28.	Both forequarters.	Similar but larger area.	Same area as cold.	Nil.
A. R.	34.	Both forequarters and lumbar areas on right leg.	Similar area.	Similar area.	Nil.
M. B.	28.	At first both arms and left leg, then universal.	Same area.	Same area.	Nil.
L. G.	30.	Both arms. Area not sharply defined.	Both forequarters and right side of neck.	Similar to heat. Slightly larger area on right side.	Loss of passive position and compass sensibility on right hand.
F. C.	53.	Both sides of neck and left side of face.	Same area.	Same area.	Nil.
L. B.	17.	Right foot.	Same area.	Same area.	Nil.

Thus in every one of my nine cases of syringomyelia, the sensations of pain, heat, and cold were lost together, over areas that were almost exactly the same. In only one case (that of

L. G.) was any other form of sensation affected, and here there was loss of the ability to distinguish the two points of a pair of compasses on the right hand.

It is interesting also to note that in six of the nine cases trophic changes occurred; in five cases these were the so-called "Charcot's joints," and in two cases there were trophic ulcers and blisters on the skin.

This threefold association of the loss of sensation of pain, of heat, and of cold in cases of syringomyelia has been long recognised, and is extremely striking. Cases have been described in which other forms of sensation have been affected, yet it has been invariably found that their loss extends over a much smaller area of skin than the loss of pain, heat, and cold sensibility.

Similar results are found in cases of hæmotomyelia, as in Case 2 of my series, where pain, heat, and cold were lost as the result of a sudden hæmorrhage into the grey matter of the spinal cord, the extent of which was determined after death. Thus, at their very entrance into the spinal cord, the paths conveying the impulses of pain, heat, and cold are closely associated, for in both of these affections sensory impulses are interrupted immediately after they have entered the spinal cord.

Not only is this the case, but the paths of these three forms of afferent impulses are contiguous in their further course up the spinal cord. This question can be studied only in those cases in which a lesion in the upper part of the cord has produced sensory changes in the lower limbs.

Thus, in the case of J. F. already described, in which a left-sided tumour of the spinal cord was after death found in the cervical region, the loss of sensation in the legs was as follows:—

On the right leg: complete loss of pain, heat, and cold. Other sensations normal.

On the left leg: loss of the sense of passive position.

In the six other cases of unilateral lesions of the spinal cord detailed in this Thesis, the sensory phenomena were equally definite. In all cases the sensory loss below the lesion consisted of anæsthesia to pain, to heat, and to cold on the opposite leg.

The tabulated statement is as follows:—

CASE.	NAME.	NATURE.	PARALYSIS.	SENSORY LOSS.
	F. C., m., 37.	Came on during sleep. Mitral regurgitation.	Weakness of left arm.	Loss of pain, heat, and cold in the right leg and right side of trunk.
	G. G., f., 30.	Came on while asleep, during first day of menstruation.	Right leg spastic.	Loss of pain, heat, and cold on the left side to level of umbilicus. Loss of passive position in right great toe.
	A. H., m., 25.	Fell down the hold of a ship.	Left arm weak and wasted. Left leg rigid and spastic.	Loss of pain, heat, and cold in the right leg, right side of trunk, and inner side of right arm.
	C. F., m., 26.	Fell on to his head while diving from a bathing machine.	Weakness in right arm.	Loss of pain, heat, and cold over the left leg, and left side of trunk up to the nipple.

CASE.	NAME.	NATURE.	PARALYSIS.	SENSORY LOSS.
	E. T., f., 24.	Tumour of cervical cord.	Weakness of left hand. Rigidity of left leg, and later paraplegia.	Loss of pain over right leg and over right side of trunk. Loss of heat and cold over right leg and both sides of trunk.
	C. B., m., 59.	Fracture and dislocation of 4th cervical vertebra.	Spasticity and paresis right arm and leg.	Loss of passive position right great toe. Loss of painful and thermal sensibility left arm and leg.

In all these six cases it will be seen that a unilateral lesion of the spinal cord produced a loss of sensibility to pain, to heat, and to cold on the opposite side below the lesion.

These observations upon unilateral spinal lesions, and their effect upon the sensibility to pain, heat, and cold, are in close agreement with the results of other observers. Thus Karl Pétren (14), who has made a careful analysis of 128 cases of unilateral lesions of the spinal cord, found that in 59 cases such a lesion produces a loss of painful and thermal sensibility on the opposite side of the body, there being no loss of sensibility to pain, heat, or cold on the same side of the body as the lesion. In the remaining 69 cases he found that this loss of sensibility to pain, heat, and cold on the opposite limb below the lesion was associated with a loss of the pressure sense.

Thus, not only are the paths for pain, heat, and cold, contiguous at their entrance into the spinal cord, but in their further course up the spinal cord they also lie very closely together.

In many cases also where the spinal cord has been pressed upon from outside, it has been found that a loss of pain has been accompanied by a loss of sensibility to heat and cold.

Thus Böttiger (15) found that in a case of tumour, pressing upon one side of the spinal cord in the region of the eighth cervical segment, thermo-anæsthesia and analgesia were present on the opposite lower limb, the pressure sense remaining normal.

In syphilitic lesions of the spinal cord, the same association of the sensibilities to pain, heat, and cold is observed. Such a condition I found in Case 20., H. A., *aet.* 29, who suffered from a syphilitic myelitis supervening eighteen months after infection. Here both legs were affected with spastic paralysis, and there was a good deal of trouble with micturition. Over the right leg and the right side of the trunk, sensibility to pain, heat, and cold, was lost completely, while it was slightly diminished over the left leg as well.

Enough has been said to show that when sensibility to pain is lost from a lesion of the spinal cord, it is almost invariably accompanied by a loss of sensibility to heat and to cold. So well recognised has this become, that a loss of pain, heat, and cold, has come to be known as the "typical dissociation of sensation" that is found in certain lesions of the spinal cord.

Moreover it is the sensation of pain as a whole that is lost, when its loss is caused by disease or injury of the spinal cord. As has been previously shown, when a peripheral sensory nerve is cut, the pain caused by a pin-prick is lost over a certain area. Over this area the "pain of deep pressure" (tested either by a crush over a bone with a hard instrument or by the algometer) is still present. On the other hand it has been found that, in cases where the skin was analgesic owing to spinal lesions, firm pressure over a bone produced no sensation of pain.

It will be clear that in these cases it is extremely difficult to obtain a corresponding point on the opposite side of the body for purposes of comparison with the sound side. This difficulty is caused by the impossibility of testing satisfactorily for the pain of deep pressure, unless the skin over the bone which is tested is analgesic.

However, it appeared that in cases where the pain of a pin-prick was absent, a crushing thrust over a bone produced no unpleasant effect, only a sensation of heavy pressure. In one of my cases, however, the ideal conditions were by a fortunate chance presented:—

This was in Case 4 (E. L. T.), in whom a rapidly growing tumour was present, chiefly on the left side of the cervical spinal cord. Here the sensibility to pain, heat, and cold, was lost over the right leg, and over both sides of the trunk, the left leg being quite normal Thus the skin over both anterior superior iliac spines was analgesic. Firm pressure with a wooden handle over the left anterior superior spine caused the patient to cry out and to move away. She said that she felt a very unpleasant sickening pain. A similar pressure over the right anterior superior iliac spine caused no reflex movement, and the pressure could be increased greatly without causing the patient any discomfort. She said that she felt no pain at all, but she knew that heavy pressure was being made over the bone. Thus in a case where it was possible to compare deep sensibility on opposite sides of the body, it seemed that the pain of deep pressure disappeared coincidently with the pain produced by pin-prick, and this occurred even though pressure over the right leg was perfectly appreciated and accurately localised.

Again, just as with the sensation of pain, sensation of heat as a whole is lost in cases of gross lesions of the spinal cord. In other words, the degree of temperature of the stimulus is of no import, since the skin remains insensitive to heat stimulation by all temperatures which produce any sensation of heat or warmth upon normal skin. This point was very carefully tested in all the cases investigated, in order to compare the loss of sensation occurring in disease of the spinal cord with that which obtains when a peripheral nerve is divided. As has been shown in the previous chapter, the impulses giving rise to sensations of heat and cold are carried in two definite groups, and most probably by different sets, of nerve fibres. In the protopathic group run the impulses giving rise to sensations of heat aroused by stimuli of a temperature over 50° C, and when protopathic sensibility is present alone, stimuli of varying temperatures above 50° C cannot be distinguished one from the other. On the other hand, there is another group transmitting sensory impulses, the epicritic group, with which are associated the impulses giving rise to sensation of heat below 50° C. If this form of sensibility be alone present, it is possible to differentiate between temperatures below 50° C which differ by a certain liminal number of degrees. No such distinction occurs when the sensation of heat is lost owing to a lesion of the spinal cord. A very similar condition obtains when the sensation of cold is lost owing to disease or injury of the spinal cord. When lost at all it is lost as a whole.

Thus over an area where sensibility to both heat and cold is lost, a stimulus at a temperature of 0° C, 20° C, 24° C, 40° C, 50° C, or 60° C, produces no sensation of either heat or cold.

Paradoxical heat and paradoxical cold.—Occasionally over an area, especially one situated at the periphery of a region of complete thermo-anæsthesia, a cold test-tube is called hot, or a hot test-tube may be called cold. These so-called "paradoxical" sensations are difficult to understand. In the first place it is necessary to be certain that the patient does not expect to be tested by temperature stimuli. If he is aware that he is to be so tested, it has been frequently found that he answers all temperature stimuli as "warm" or as "cool." This fallacy is avoided

by testing him with an empty test tube and observing if he still continues to ascribe to this stimulus the same sensation. If he does so, the testing must be given up for the moment, and again resumed some time afterwards when the patient is unaware of the form of stimulation that is being used.

Still, in certain skin areas, when this fallacy has been avoided, stimulation by all degrees of temperature elicit from the patient the response of either "hot" or "cold." In testing these cases a remarkable correspondence has been observed between this so-called paradoxical temperature sensation and the subjective sensations experienced by the patient in the part affected.

Thus in the case of F. C. (Case 5), who suffered from a local hæmatomyelia, over the skin at the upper border of the totally anæsthetic area an ice test-tube was called "warm" or "hot." This patient stated that the affected leg always felt burning hot and was always warmer than the opposite leg, in which sensation was normal.

Again, in the case of syringomyelia already detailed in full, all temperature stimuli were called "cold" near the limits of the anæsthetic area. This patient experienced such extreme subjective sensations of cold, that he was forced to go to bed as soon as he got home from his work.

Possibly this correspondence between the subjective sensations experienced by the patient and the quality ascribed by him to temperature stimuli may explain the occurrence of "paradoxical" sensibility for heat and cold.

Consequently we conclude that in the spinal cord, the fibres transmitting impulses of pain, heat, and cold are associated and that their paths lie very closely together, not only as they enter, but during their whole course within the cord.

Tactile Sensibility and Pressure Sense.

With the actual paths of these sensory impulses in the spinal cord we are not concerned in this section: these will be fully discussed in a later chapter of this Thesis. We are only concerned with the question as to whether light touch and pressure sensations are lost together when the lesion is situated in the spinal cord.

That, on the periphery, these two sensory impulses are carried by different sets of nerve fibres has been proved by Head and Sherren (5). They have shown that when a nerve is divided near its termination, for example, the median nerve at the wrist, the sense of light touch is lost over the corresponding area of the palm: the stimulus used being the lightest touch that can be applied with cotton wool. On the other hand the sensation of pressure is well recognised and well localised over the same area. Hence, from the periphery the impulses giving rise to sensations of light touch travel up in the cutaneous sensory nerves, while those giving rise to the sensation of pressure are conveyed up to the central nervous system along the nerve trunks which supply the muscles. In reviewing the literature of the subject, we are met with the difficulty that many authors fail to make any distinction between the sensation of light touch and the sensation of pressure. Not only is this the case, but when this distinction is made, improper tests for sensation have been used. Thus light tactile sensibility has been tested by "stroking the skin lightly with the finger," etc., a form of stimulation which always affects the nerves which subserve deep sensibility. The question is further complicated by the peculiar innervation of the hairs. It is therefore of the utmost importance that the skin of the part should be shaved before testing for light tactile sensibility.

However it is clear that, when deep tactile sensibility has been shown to be absent, statements as to the presence or absence of light tactile sensibility may be assumed to be correct in those cases in which it is stated that "light touch" has been tested. For, even if the stimulus used

for "light touch" has been sufficient to arouse deep sensibility as well, no pressure stimuli can reach the spinal cord. Unfortunately the material at our disposal in this connexion is not nearly so extensive as that for the examination of pain, heat, and cold sensation. One of the most striking features in gross lesions of the spinal cord is the great extent of the destruction that may be present, without producing any loss of tactile sensibility.

First, then, to examine the paths of these sensory impulses as they enter the spinal cord, we must examine lesions which have produced a loss of sensation on the same side as the lesion in the area of skin corresponding to the affected segments of the cord.

Case 1. J. F., who had a tumour in the left side of the cervical cord, is of interest in this connexion. Here, as has been already stated, the left arm had, when first examined, lost all forms of sensation except those of light touch and pressure. A few weeks later deep touch was also abolished, light touch being a good deal diminished. Thus the fibres transmitting impulses of light and deep tactile sensibility are associated together soon after their entrance into the spinal cord.

In the literature several similar cases are described. Max Laehr (16) records two cases of syringomyelia in which there was tactile anæsthesia.

Case 1, a man, 27 years of age, affected with tuberculosis, received in 1890 a severe blow in the back, followed by paraplegia of short duration. He was a blacksmith, and did much heavy work with his hands. About the end of 1892 he noticed wasting of the small muscles of the left hand. In the spring of 1895 he presented himself at the hospital with an abscess of the left forearm. There was then chronic tuberculosis of the right lung, atrophy and weakness of the muscles of the left hand and in lesser degree in those of the right hand. There was also weakness and wasting of the posterior shoulder muscles, chiefly of the left side and of the deep back muscles. There was spastic paralysis of both legs, and scoliosis. On the arms and on the upper part of the trunk there was a loss of sensibility to pain, heat, and cold.

Laehr states that at this period light tactile sensibility was diminished between the third and fifth thoracic segments inclusive on the right side, the loss extending over a small region upon the anterior and posterior parts of the right side of the thorax and over a small strip down the inner side of the right arm. The area had no sharp boundaries. There were no other changes in contact and pressure sensation (Druckempfindlichkeit); localisation was also good.

The disease progressed slowly, and in March, 1896 (one year later), the following condition was found:—

The zone of tactile hypæsthesia on the right side of the trunk was a little broader and extended a little further down the inner side of the right arm, and in this area pressure with a rod (Stieldruck) was often not recognised.

In this rare case of syringomyelia it is clear that pressure and light tactile anæsthesia tended to disappear over the same areas.

In Laehr's second case of syringomyelia in which there was tactile anæsthesia the record is as follows:—

A widow, aged 23, in 1889 received a blow on the head. In 1893 there were subjective sensations of cold about the left shoulder and wasting of the muscles around it. In June, 1895, she attended with an abscess on the left arm, paresis of left leg, and cystitis.

There was then wasting of the muscles about the shoulder with fibrillary twitchings, and kypho-scoliosis. There was a widespread loss of sensation to pain, heat, and cold, over the left arm, left side of neck, and upper part of the trunk. Pressure was everywhere recognised, and localised. In the region of the left deltoid, light touches (feine Pinsel-berührunungen) were often not perceived.

In March, 1896, the disease was found to have progressed slowly, the loss to pain, heat, and cold, being more extensive. There was also a zone on both shoulders of light tactile anæsthesia, and over this area there was also deficient recognition of pressure (Stieldruck). Later on, areas over which there was loss of light tactile and pressure sensibility were found on both shoulders and the corresponding parts of the neck.

In both these cases then, in which the excavation in the cord had been sufficiently extensive to involve the fibres conveying tactile sensibility, sensation to both light touch and pressure was lost over approximately the same areas.

These cases merely show that near their entrance into the spinal cord the paths for pressure and light touch lie closely together. The correspondence is not complete however, nor indeed would one expect it to be, since impulses leading to a sensation of pressure enter the cord in the group of fibres for deep sensibility, while the impulses for light tactile sensibility enter with the epicritic fibres.

Secondly, it is of importance to study the effect of cutting off the paths of light and deep tactile sensibility after they have entered the cord and are passing up towards the brain.

As Pétren (14) has shown in his masterly analysis of the literature of hemilesions of the spinal cord, pressure sensation is lost in the parts below the lesion in a far smaller number of cases than are sensations of pain, heat, and cold. Like the sensations of pain, heat, and cold, the pressure sense is lost on the opposite side of the body to the spinal lesion, and its loss is always associated with analgesia and thermo-anæsthesia. So far as can be gathered, light tactile sensibility is lost as well as the pressure sense, since no mention of the presence of the former is ever made.

The case of H. L. (No. 3) is of great importance in the consideration of this question. Here, a lesion beginning on the left side of the cervical cord had produced a profound loss of the sensation of pain, heat, and cold, below the level of the fifth cervical root.

Tactile sensibility was however lost only on the right leg and trunk, and on a small area on the right forearm and hand, as figured in the diagram. In this case the relation between the two forms of tactile sensibility was carefully investigated.

On the right arm an area of loss to pressure could be determined, occupying part of the preaxial half on both dorsal and ventral surfaces of the forearm, and including the back of the hand and the whole of the palm. Over the remainder of the arm pressure was at once recognised and well localised.

This area was then tested with cotton wool, and it was found that the loss to cotton wool touch corresponded in a most remarkable way with the loss to pressure. The back of the forearm was then shaved, and it was found that this made absolutely no difference to the extent of the loss to cotton wool.

From these observations, although they are admittedly insufficient, one may perhaps hazard the conclusion that in their course up the spinal cord the paths for pressure and light touch are closely associated, and that the two may be classed together as tactile sensibility.

It is clear too that the fibres conveying tactile impulses up the spinal cord are not associated with the fibres conveying the impulses of pain, heat, and cold. This is shown not only by the rarity with which tactile sensibility is affected in syringomyelia, but also by the fact that in nearly one half of the recorded cases of hemilesion of the cord, the sensation of pain, heat, and cold were lost below the lesion, while there was no affection of tactile sensibility.

The sense of locality and extensity.

To the third and last group of sensations which seem to be lost together in gross lesions of the spinal cord, I have ventured to give the name of "locality and extensity."

This group includes the recognition of position on the skin (topognosis), of the points of a pair of compasses (stereognosis), and of the position of the limbs in space. In this group are also included the sensations of form and area. The afferent impulses which arise from the end organs in the tendons and muscles also probably pass up with this group (sense of muscular movement).

This is the group of sensations that is so profoundly affected in tabes dorsalis, and gives rise to the ataxia and the loss of localisation in space characteristic of the disease. As Dr. Head stated, in his Marshall Hall lecture, it is possible to find cases of tabes where the sensation of light touch is perfectly preserved, but where the power to distinguish the two points of the compasses is entirely lost even when these are separated for a distance of 20 cm.

Just as in the case of the two previous groups, the association of the various sensations composing this group must be studied as their fibres are entering the spinal cord, and as they are passing up after entering its substance.

Among my series the case of L. G. (No. 17) is of great interest in the study of these sensory impulses as they enter the spinal cord. The patient was aged 30, and had suffered from cervical syringomyelia for four years. In January, 1904, she had lost the sensations of pain, heat, and cold, only in the hands and forearms, but in May, 1905, the area over which these sensations were lost had extended so as to include both arms, the shoulders, and the right side of the neck. On the right palm there was definite loss of sensation to the compass test, the records being :—

	Palm of hand. Right.	*Transverse.* Left.
1 cm. apart.	All called "one."	1 | III IIII III 2 | XII IIII III
3 cm. apart.	1 | IIII III III 2 | XXXX XXX XXXX	1 | IIII III III 2 | III IIII III
4 cm. apart.	1 | IIII III III 2 | XIID IDX DXI	Perfectly normal.

Absolutely lost up to 4 cm.

This loss of the power of distinguishing the points of the compasses was associated with a loss of the sense of passive position and of the sense of form and area.

The case of J. F., in whom a tumour in the left side of the cervical cord had partially destroyed sensation in the left arm, must be mentioned again here. On the left arm, in addition to a loss of the sensation of pain, heat, and cold, there was an inability to recognise the two points of the compasses, with loss of the sense of area, a deficient localising power, and a complete absence of the sense of passive position at the joints of the arm and fingers. At the same time the sensations of pressure and light touch were recognised.

In this case the sensations comprising the group I have included under the term "locality and extensity" seem to have been associated in their loss.

Several similar cases where the loss of the sense of passive position has been associated with a loss of localisation occur in the literature dealing with syringomyelia. The association is not however uniformly found, as one would have expected from the different ways in which this group of sensory impulses enter the cord.

It is of much greater interest to study the changes in this form of sensation when the parts below the lesion are affected.

In a unilateral lesion of the cord, sensibility to pain, heat, and cold is always lost on the opposite side, while the sense of locality and extensity is lost on the same side as the spinal lesion.

A summary of the cases of hemilesion described in this Thesis will show how constantly this is the case.

CASE.	PARALYSIS.	SENSORY LOSS.
(1) J. F. Tumour of Cord.	Paralysis of left arm and both legs.	Right. Analgesia and thermo-anæsthesia. Left. Loss of passive position in great toe.
(2) G. G. Hæmatomyelia	Spastic paralysis of right leg.	Right. Loss of passive position in leg. Left. Analgesia and thermo-anæsthesia.
(3) C. B. Fracture of 3rd cervical vertebræ.	Spastic paralysis of right leg and right arm.	Right. Loss of passive position in leg. Defective localisation right leg and arm.
(4) E. T. Tumour of Cord.	Paralysis of all four extremities, beginning in left arm.	Right. Analgesia and thermo-anæsthesia. Left. Loss of passive position in great toe.
(5) L. K. Syphilis of Cord. (Laehr 16).	Weakness of right leg.	Right. Ataxy. Loss of passive position. "Kussmaul bodies" slowly recognised. Left. Analgesia and thermo-anæsthesia.
(6) A. R. Syphilis of Cord. (Laehr 16)	Paresis of left arm and leg.	Right. Analgesia and thermo-anæsthesia. Left. Loss of passive position in left arm and leg.

Other cases could be added, but the above are sufficient to show that the effect of a unilateral lesion of the cord is to cut off the impulses giving rise to sensations of locality and extensity on the same side as the lesion.

Thus in the spinal cord we may recognise three groups of afferent impulses.

(1) The group of painful and thermal impulses.

(2) The group of tactile impulses.

(3) The group of impulses concerned with locality and extensity.

CHAPTER VII.

Contrasting the Grouping of Sensory Paths in the Peripheral Nerves with that of the Paths in the Spinal Cord.

In contrasting these two groupings of sensory fibres it will be convenient to consider the various forms of sensation *seriatim*.

From the periphery, sensory impulses giving rise to *pain* are conducted by two of the great afferent systems, namely the protopathic nerves along which sensations of pain are conducted from the skin, and the nerves of deep sensibility along which the painful impulses of deep pressure pass up to the spinal cord.

But when the sensation of pain is lost from gross lesions of the spinal cord, no such distinction is found; all forms of painful sensibility are abolished together.

Thus section of a cutaneous nerve produces a loss of cutaneous sensibility to pain without loss of the pain produced by pressure over a bony prominence.

On the other hand in cases of syringomyelia, crushing pressure produces no such feeling of discomfort in the part where cutaneous painful sensibility is absent. This is very well seen in the case of E. T. (No. 4) already referred to, in which it was possible to compare the two sides of the body. Here cutaneous painful sensibility was lost over a very large extent of skin on the right side and over a very small area on the left side. The pain of deep pressure was lost over the right anterior superior spine but was present over the left, the skin over the two spines being analgesic. Hence it is clear that whether the analgesia be due to interruption of the paths for painful sensory impulses near the entrance of the fibres into the cord (as in syringomyelia), or be situated over the lower part of the body in consequence of a lesion of the cervical spinal cord, sensibility to all forms of painful stimulation is destroyed simultaneously.

I would therefore suggest that there are certain cells in the posterior horns of the spinal grey matter which react to painful stimulation alone, and that around these both the protopathic fibres and those of deep sensibility send collateral branches. Further, from the observation that, when the axons of these cells are interrupted in their course up the spinal cord, pain is lost entirely, it seems probable that the axons of these posterior horn cells, and these axons alone, convey the specific stimulus of pain.

The sensory impulses derived from stimuli of *heat and of cold* are also carried up to the spinal cord by two distinct afferent systems. In the protopathic fibres are carried the impulses giving rise to sensations of extreme heat (that is to say, temperatures over 50° C) and of extreme cold (that is to say, temperatures below 18° C), while in the epicritic group of nerves transmit impulses giving rise to sensations of the minor degrees of heat and cold (that is, temperatures ranging between 45° C and 18° C) and the power of differentiating between them.

When sensations of heat and of cold are lost as a consequence of gross lesions of the spinal cord, it is found that they are lost in their entirety. Thus over any area where "heat" is lost, test-tubes at all temperatures, which on normal skin produce sensations of warmth or heat, fail to produce any sensation of heat whatever. Again, over an area where "cold" is lost, test-tubes at all temperatures, which on the normal skin produce sensations of coolness or cold, fail to produce any such sensation.

This is entirely different from the result produced by the division of cutaneous nerves. Areas of skin have been investigated where in consequence of injury to a peripheral nerve protopathic sensibility was present, while epicritic sensibility was absent; as a result, water above 55° C or under 18° C was appreciated as hot or cold, while the intermediate degrees were unrecognised. Again, areas of skin have been found where epicritic sensibility was present and protopathic sensibility absent, and over these areas water of 35° C seemed to be actually hotter than water at 55° C, while water at 20° C was said to be colder than a test-tube filled with ice.

Another observation of great importance in this connexion is that disease of the spinal cord may produce a loss of the sensation of heat in a part of the skin where the sensation of "cold" is subjectively present and *vice versa*. This is exceedingly well seen in the case of C. H. (No. 15). In this patient all sensations of heat were lost over the right side of the forehead, the cheek, and right lower jaw, while over this area of skin, all forms of stimuli, which on the normal skin produced a sensation of cold or coolness, evoked the normal response.

Consequently these observations tend to show that there exist, in the posterior horns of the spinal grey matter, certain cells which respond to the impulses produced by heat and certain others which respond to cold. These cells and their axons may be termed the secondary sensory neurons: they seem to convey specific stimuli of heat and of cold up the spinal cord, and they apparently cross to the opposite side soon after the axon leaves the cell body.

The question of *tactile sensibility* is more complicated. On the periphery light touch is carried up by the epicritic system, while the impulse of pressure is transmitted by the fibres for deep sensibility and mainly by those afferent fibres which accompany the motor nerves. As Dr. Anderson has shown, however, a part of the great Pacinian system seems to be present in the skin, especially over the palms and soles of the feet.

It seems clear that as a consequence of unilateral lesion of the cord, light touch and pressure are lost over exactly coincident areas on the side of the body opposite to the lesion. Probably then a group of cells exists in the spinal grey matter, whose axons travel upwards on the opposite side of the cord to the skin they represent. These are concerned in conveying tactile impulses of all kinds up the spinal cord. As the epicritic fibres and the fibres for deep sensibility run up in the posterior columns, they give off collaterals to this group of cells all the way up the spinal cord.

The *sense of locality and extensity*, when lost by reason of unilateral lesions of the spinal cord, is always absent on the side of the lesion, the side of the spastic paralysis.

On the periphery this group of impulses is conducted partly by the deep and partly by the epicritic systems of fibres. In the former run the impulses concerned with localisation of pressure, the sense of passive position, and the sense of muscular movement. In the latter pass up the impulses by which light touch is localised and the two points of a pair of compasses are distinguished; acuæsthesia and the sense of form and of area also pass up in this system. In the spinal cord it is most probable that all these impulses pass up in the posterior columns of the same side being, conducted by primary sensory neurons. It is not until the gracile and cuneate nuclei are reached that the secondary sensory neurons of this great group of impulses appear. Thus the posterior columns probably represent the central ramifications of peripheral nerves, and contain fibres identical with those which on the periphery are known as fibres for epicritic and deep sensibility. It is not until the gracile and cuneate nuclei are reached that any completely specific impulse can be isolated; the impulses of pain, temperature, and touch, which pass up in these two systems as they lie in the posterior column probably (if one may use the expression filter out by means of the arborization of collaterals around the more specific sensory neurons of the second order. Consequently a lesion of the posterior columns

of the cord can be expected to produce only undifferentiated loss of sensation, a loss which will be similar in kind to that produced by a lesion of a peripheral nerve.

The following table summarises the different grouping of sensory paths in the spinal cord and in the peripheral nerves.

	Peripheral Nerves.	Spinal Cord.
Protopathic	Pain of pin-prick	Painful and Thermal Impulses
	Heat over 40° C.	
	Cold below 20° C.	
Epicritic	Heat below 40° C.	Tactile Impulses
	Cold above 20° C.	
	Light touch	
	Power of distinguishing two points	
	Sense of form	
	Sense of area	
	Localisation of light touch	
Deep Sensibility	Pain of deep pressure	Impulses of Locality and Extensity
	Pressure	
	Muscular movement	
	Passive position	
	Localisation of deep pressure	

CHAPTER VIII.

The Path for the Impulses of Pain and of Temperature.

As has been previously shown, an overwhelming mass of evidence is forthcoming to prove that the paths for impulses of pain, of heat, and of cold, are associated together in the spinal cord, as a well defined group.

(1) *The path of entry.*

The path by which the impulses for pain and for temperature enter the spinal cord can be investigated only in cases when a local lesion has produced a loss of sensibility on the same side of the body in the area of skin supplied by the segments in which the lesion lies.

It is clear that for this purpose the most suitable cases are those of unilateral syringomyelia, since in an early example of this disease, the sensations of pain, heat, and cold, are alone destroyed, while all other forms of sensation remain normal.

In such cases it has been found that when sensibility to pain, heat, and cold, is lost over one arm, *post-mortem* examination shows that the posterior horn on the corresponding side has been completely destroyed, at a level which receives the different fibres from the area of skin effected. The following cases may be cited in proof of this:—

Rossolimo (17). Ein Fall von Gliomatosis eines Hinterhornes des Rückenmarks. The patient was a girl of 18 years. She had complete analgesia and thermo-anæsthesia over the left arm, with

slight impairment of pressure and tactile sensibility. Death resulted from sepsis following an abscess on the left forearm.

On *post-mortem* examination a glioma of the left posterior horn was found, extending from the first cervical to the tenth thoracic segment, the maximum lesion being in the fifth cervical segment. The posterior columns, the lateral columns, and the right posterior horn were unaltered.

Dercum and Spiller (18). This is a case of syringomyelia limited to one posterior horn in the cervical region. The right arm showed a loss of sensibility to pain and to temperature. The pressure sense was normal. Below this a complete motor and sensory paralysis of both legs was found.

On *post-mortem* examination a complete transverse lesion in the thoracic region was found, and in addition there was in the cervical region a glioma, which had destroyed the right posterior horn, but had not otherwise affected the spinal cord.

Fürstner-Zacher (19). Zur Pathologie und Diagnostik der spinalen Höhlenbildung.

This is a complicated case, in which many parts of the spinal cord were affected by gliomatosis with fissure formation.

Over the right arm there was analgesia and inability to distinguish heat and cold.

On *post-mortem* examination, in the cervical region on the right side, the posterior and anterior horns, together with the grey commissures, were the only parts affected.

Déjerine and Sottas (20). (Sur un cas de syringomyélie unilatérale et à début tardif suivi d'autopsie).

The patient was a man of 60 years of age. There was muscular atrophy of the right arm, and a diminution of sensibility to temperature over both legs: there existed in the right arm a total loss of sensibility to pain, heat, and cold. Tactile sensibility and the muscular sense were normal. The man died of a right-sided pneumonia.

At the *post-mortem* examination the following parts of the cervical cord were found to be destroyed: the entire right posterior horn and the posterior two-thirds of the right anterior horn, together with the anterior two-thirds of the right posterior column. In the thoracic and lumbar regions the disease had destroyed only the bases of the posterior horns.

These cases show, with the utmost clearness, that destruction of one posterior horn in the cervical region of the cord gives rise to a loss of painful and thermal sensibility on the arm of the same side; and consequently we must conclude that all the paths for pain and temperature pass directly into the grey matter of the posterior horn at their entrance into the spinal cord.

As was shown in the last chapter, the difference between the peripheral and the central grouping of pain, heat, and cold impulses, makes it most probable that the secondary sensory neuron starts in the posterior horn of the same side. Consequently the anatomical findings in the above series of cases serve to strengthen this conclusion, and to locate the cell bodies of the secondary neurons for impulses of pain, heat, and cold, in the grey matter of the posterior horn.

(2) *The path of the secondary sensory neurons.*

The cell bodies of the secondary sensory neurons lie in the posterior horn of the same side.

As was shown in Chapter VI., the path of the impulses for pain and for temperature rapidly crosses over to the opposite side of the cord. This crossing, in all probability, takes place in the posterior grey commissure. In syringomyelia it is the posterior commissure which is usually and early destroyed, while the anterior commissure frequently remains intact. For some reason the gliomatous process seems to pass backwards to the bases of the posterior horns rather than forwards. It is in this disease that loss of painful and thermal sensibility characteristically

occurs, and the fact that the posterior grey commissure is usually implicated no doubt accounts for this sensory loss.

The following case seems to show that the anterior commissure may be completely destroyed without any loss of thermal or painful sensibility:—

J. Sander. Ein Fall von Paralysis atrophica.

Paralysis and wasting of the hand muscles were present. Sensation was scarcely, if at all, affected. Later the patient suffered from pains in both arms and over the left breast.

At the *post-mortem* examination, a glioma was found involving the anterior grey commissure and both anterior horns in the thoracic region. The anterior horns were almost completely destroyed.

We may therefore conclude that the secondary neurons pass across to the opposite side of the cord in the posterior grey commissure.

In Chapter VII., evidence was given to show that in cases of syringomyelia the limits of the cutaneous areas of loss of painful and thermal sensibility do not always coincide. In the majority of cases of syringomyelia the morbid process spreads outwards from the central canal of the spinal cord, and it is usually found that sensibility to heat is lost over a larger area than sensibility to cold, and sensibility to cold over a larger area than sensibility to pain.

The results obtained in my series of cases of syringomyelia with regard to this point may be summarised as follows:—

CASE.	PAIN.	COLD.	HEAT.
C. H. Case 15. Syringomyelia.	Lost over the right arm as high as the root of the neck.	Lost over the right arm and the right side of the neck.	Lost over the right arm, right side of neck, and right side of the face.
F. R. Case 11. Syringomyelia.	Lost over the two arms and the neck and part of both sides of the face.	Lost over a slightly larger area.	Lost over a slightly larger area than cold.
W. H. Case 14. Syringomyelia.	Right arm.	Right arm, right upper trunk, and right neck.	Same as cold.
T. B. Case 10. Syringomyelia.	Lost over exactly the same extent.		
H. L. Case 3. Tumour of cord.	Right side below clavicle. Left leg and left arm.	Whole body below clavicles.	Same as cold.
L. C. Case 17. Syringomyelia.	Arms.	Arm, upper trunk, and right side of neck including right ear lobule.	Same as cold, but including the whole of right ear.
A. R. Case 18. Syringomyelia.	Above including the neck, below to umbilicus.	Above to clavicles, below including lumbar areas of right leg.	Same as cold.

CASE.	PAIN.	COLD.	HEAT.
E. T. Case 4. Tumour of cord.	Right side up to nipple line.	Right side up to nipple line, and also left side of trunk.	Same as cold.

This summary of cases shows that soon after their origin the axons of the cells of the posterior horn, along which pain and temperature impulses are carried, are arranged in the following order :—

Those along which heat impulses are transmitted are nearest the centre of the cord, those along which cold impulses are carried are slightly farther away, whilst those along which painful impulses are conducted lie at a slightly greater distance from the central canal.

(3) *The further course of the secondary sense neurons.*

In order to study the further course of these paths, it is necessary to adopt a different method. We must now use cases in which a local lesion in the upper part of the cord has produced a loss of painful and thermal sensibility in the lower limbs.

It is usually assumed that these paths run up in the grey matter. That this is extremely unlikely is clear from a general consideration of syringomyelia. In the great majority of cases this disease causes a very extensive destruction of the grey matter in the cervical region of the cord, yet it is only in a few cases that there is any loss of sensibility in the lower extremities. Thus of the eight cases of syringomyelia I have investigated, in only one was there any marked loss of sensibility to pain, heat, and cold, in the legs ; and an examination of the literature shows that this is usual in the disease. The following cases in which the necropsy is described seem to prove this point :—

Taylor (22). A case of syringomyelia with necropsy. Analgesia and thermo-anæsthesia were present in both arms and on the trunk, above the level of the fifth dorsal segment. The sensibility of the legs was normal.

At the *post-mortem* examination, in the upper cervical region the posterior horns were entirely destroyed, together with the adjacent parts of the posterior columns. The anterior horns were also slightly affected.

In Schlesinger's classical Monograph on Syringomyelia the following case is described :—

Schlesinger (23). Die Syringomyelie.

Clinically heat and cold were confused over both forearms and hands.

At the *post-mortem* examination, a condition of hydromyelia with some gliomatous formation around the dilatation was found. This was most marked in the cervical swelling. The ganglion cells of the anterior horns and the entire posterior horns had disappeared, the white substance being relatively little affected.

Rotter (24). Zur Casuistik der Hydro- und Syringomyelie.

Sensation in the legs was normal. Death resulted from pneumonia. The necropsy showed that in the fourth cervical segment the spinal cord was represented only by a narrow ring of white substance around a large central cavity. Of the grey substance, both posterior horns were totally destroyed, only a small part of the anterior horns remaining intact.

These cases show clearly that painful and thermal impulses from the legs are not transmitted through the cervical region in the grey matter of the posterior horns. It is in the highest degree unlikely that these paths pass up in the grey matter of the anterior horns. In acute

anterior poliomyelitis the anterior horns are destroyed by an acute inflammation, so intense that in the majority of cases only a shrunken scar remains. These cases are characterized by an entire absence of any change in sensation. The following case serves to confirm this conclusion :—

Schultze (25). Klin. und Anat. über die Syringomyelie.

Here, clinically, there was a loss of pain and temperature sensibility in both arms, while the sensation in the legs was normal.

At the *post-mortem* examination, in the upper thoracic and lower cervical region a gliomatous process had destroyed the anterior horns and the grey commissures completely, together with the greater part of the posterior horns.

Consequently we may justifiably conclude that the path of pain and temperature sensory impulses from the legs do not pass through the grey matter of either the anterior or posterior horn in the cervical region; this path must therefore run in the white matter.

We must now consider more fully whether this path runs up on the same or upon the opposite side of the cord. This point has been carefully considered by Karl Pétren (14). He has analysed the literature of unilateral cord lesions, and has come to the conclusion that the original teaching of Brown-Séquard was correct, namely that a unilateral lesion of the cord produces anæsthesia of the opposite side of the body below the lesion.

This is also confirmed by the clinical and pathological findings in Case 1 of my series, where I was able to obtain a *post-mortem* examination. The whole of the left side of the cord, with the exception of the anterior columns, was destroyed by a glioma in the cervical region. Sensibility on the left leg to pain, heat, and cold, was perfectly normal, while on the right side of the body below the lesion it was absolutely lost.

Pétren finds that if anæsthesia of both legs is produced by an apparently unilateral lesion the loss is always greater and of longer duration on the opposite than on the corresponding side. These conclusions are based on a study of over 150 cases.

Consequently we must assume that the paths transmitting pain and temperature impulses pass over to the opposite side of the cord soon after they enter it. As has been shown, it is practically certain that this decussation takes place in the posterior commissure and not in the anterior.

The path thus lies in the white matter of the opposite side of the cord, and most probably passes up in the lateral column.

Pétren points out that the path for pain and temperature impulses must lie farther from the middle line than the paths for the other forms of sensory impulses; for example, that of pressure. He found that in thirty-five cases in which the condition known as the "Brown-Séquard symptom complex" had been produced as the result of a wound of the spinal cord by a knife stab, in twelve, sensation to pain, heat, and cold alone was lost; while in twenty-three all forms of sensation were destroyed.

Of the twelve cases in which pain, heat, and cold sensation, were alone affected, not a single one showed bilateral spastic paralysis in the legs; while of the twenty-three cases in which sensation was lost completely in one leg, there was bilateral spasticity in eighteen.

Consequently one may argue that, in order to produce a total loss of cutaneous sensibility, the lesion must extend considerably nearer the pyramidal tract of the opposite side than it need do in order to produce loss of sensibility to pain, heat, and cold alone.

As the anterior and posterior columns are both much nearer to the middle line than the lateral column, it follows that the latter is probably the situation in which the path for pain and temperature sensations will be found.

This has been confirmed by several cases in which a *post-mortem* examination has been obtained. Thus Gowers' (26) well-known case may here be quoted. The spinal cord was damaged by a spicule of bone driven against it by a bullet, which, entering the mouth, lodged in the body of the third cervical vertebra. The only lesion found was a hæmorrhage into the lateral column and the adjoining grey matter. The posterior column was slightly swollen, apparently by œdema. The effect of this lesion was to produce an entire loss of sensibility to pain on the opposite side of the body, without any impairment of tactile sensibility.

Henneberg's case (27) is a similar one (Ueber ein Fall von Brown-Séquard'scher Lähmung in Folge von Rückenmarks-Gliom). Here there was paralysis of the left arm and leg, with right-sided analgesia and thermo-anæsthesia. A gliosarcoma had destroyed the left lateral column, but had left the anterior and posterior columns intact.

Evidence from several other cases could be cited, all pointing to the same conclusion, namely, that the paths for pain and temperature impulses run up in the opposite lateral column.

I shall further attempt to show that this path lies, not in that portion of the lateral column which adjoins the grey matter, but in the external or marginal part of the lateral column.

The evidence from the following cases may be adduced in support of this statement.

Schlesinger (28): Die Syringomyelie, p. 541. This is a case in which there was no loss of pain or temperature sensibility on the legs. The test was made a few days before death occurred from septicæmia. In the upper part of the spinal cord was a huge cavity. In the cervical swelling almost the whole of the grey matter was absent, and on one side, in addition to the anterior horn, the neighhouring white substance was entirely destroyed.

In Rotter's case (24), vide supra, although sensibility in the legs was normal, the cord at the fourth cervical segment was represented only by a narrow ring of white matter.

In Rosenblath's case (29). Zur Casuistik der Syringomyelie und Pachymen. Cervic. hypertroph. Painful stimuli were recognised as such on the legs. In the cervical region a gliosarcoma had destroyed the entire grey substance, and had left only a narrow marginal annulus of the cord.

Williamson's case (30) of acute myelitis is in this connexion of much importance. Both pyramidal tracts were destroyed, leading to a spastic paraplegia. The marginal parts of the right lateral column had been destroyed, but on the left side the marginal part of the lateral column was intact. In life this case presented a loss of pain, heat, and cold sensibility on the left leg: sensibility on the right leg was normal. Hence it would seem that the destruction of the marginal part of the right lateral column had caused analgesia and thermo-anæsthesia on the left leg. Sensibility on the right leg remained intact, although the whole of the left lateral column had been destroyed with the exception of its marginal portion.

Beck's case (31), Ueber Verletzungen der Wirbelsäule und des Rückenmarks, is interesting in this connexion. The patient had received a knife stab on the right side of the neck. There was paresis of the left arm and leg. On the right side the sensibility was normal. Sensibility on the left side was somewhat diminished, but the pressure and painful stimuli were well recognised. The lesion was found in the fourth cervical segment. The whole of the grey matter on the left side of the spinal cord, and the greater part of both the posterior and the anterior columns on that side, had been destroyed. The left lateral column however was only partially severed, the margin remaining intact. The absence of sensory changes in the right leg is accounted for by the escape of the marginal part of the left lateral column.

From the evidence supplied by the above cases, one may logically deduce the conclusion that the paths for transmitting impulses of pain and temperature pass up in the external or marginal part of the opposite lateral column.

The following two cases seem to show that the path we are considering lies in the anterior rather than in the posterior part of the lateral column.

In Hanot and Meunier's case (32) of syphilitic disease of the spinal cord, there was complete paraplegia with analgesia and thermo-anæsthesia on both legs. Pressure sense was only slightly deficient; the muscular sense was normal. At the level of the first thoracic segment was a gumma in the left half of the spinal cord, and in the second thoracic segment a similar gumma was found on the right side. In a transverse lesion between these two segments the following parts were seen to be destroyed:—

The grey matter of the anterior horns, the anterior two-thirds of the posterior horns, the white substance of the anterior columns, and the greater part of the lateral columns.

In this case a lesion of the anterior portions of the lateral columns (the posterior parts remaining intact) had given rise to bilateral thermo-anæsthesia and analgesia.

In Long's case (33) of syphilitic disease of the spinal cord there was paresis of the right leg, and on this leg loss of sensibility to painful thermal and tactile stimuli. The muscular sense was absent in this leg. On the left leg there was diminution of the muscular sense only. The patient died of pleurisy.

At the level of the sixth thoracic segment was found a syphilitic lesion which had destroyed both posterior columns, a part of the right anterior column, the posterior part of the right lateral column, and the marginal part of the left lateral column.

The degenerated parts of the left lateral column extended over half its breadth.

In this case the paresis of the right leg is accounted for by the destruction of the posterior half of the right lateral column. The loss of painful and thermal sensibility on the right leg was due to the destruction of the anterior and marginal part of the left lateral column. The escape of the anterior and marginal parts of the right lateral column accounts for the retention of these sensibilities on the left leg.

Spiller (34) records a case of caries of the vertebræ and disease of the spinal cord. In life there was a loss of painful and thermal sensibility in both legs, while tactile and muscular sensibility was normal.

At the *post-mortem* examination, it was found that tuberculous spinal meningitis had followed caries of the vertebræ. A small tuberculous deposit had invaded the spinal cord at the lower limit of the thoracic region on both the right and the left sides. The tuberculous deposits occupied the anterior and marginal parts of the lateral columns.

These cases seem to prove conclusively that the impulses giving rise to sensations of pain and temperature are conveyed up the cord in the anterior and marginal part of the lateral columns.

We have thus come to the conclusion that the secondary neurons, which convey the impulses of pain and temperature, run up the spinal cord in the opposite lateral column; but the actual level at which these paths from the legs have completely crossed to the other side has not yet been discussed.

In the case of G. G., Case 8 of my series, a lesion at the level of the twelfth thoracic segment gave rise to a crossed anæsthesia.

In Singer's case (35), a stab at the level of the ninth thoracic vertebra produced a loss of sensation of pain, heat, and cold, on the opposite side to the paralysis. This would correspond to the twelfth thoracic segment of the cord. Consequently, at this level, the crossing of the paths of these impulses from the lower limbs is complete.

With a lesion below this level however there is usually anæsthesia of the paralysed leg.

Thus in Sieletzskij's case (36) the patient had been stabbed in the back at the level of the

tenth thoracic vertebra. This corresponds to a point between the first and second lumbar segments. There was paresis of both legs, especially of the right, with loss of sensation in both legs. Later the left leg became normal in every respect; but the right leg remained paralysed, and on this leg there was analgesia and thermo-anæsthesia with diminished pressure sensibility. The upper limit of the anæsthesia coincided with Poupart's ligament and the gluteal region. Thus between the first and second lumbar segments the path has not yet crossed to the opposite side.

Similarly in Prestat's Case (37). (Plaie de la moelle épinière par instrument tranchant.) A stab between the tenth and eleventh vertebræ produced paralysis and loss of sensibility in the right leg, the left leg being normal.

We may conclude, therefore, that the paths for pain and temperature impulses have not crossed below the level of the first lumbar segment of the spinal cord, but that above this level their decussation is complete.

Summing up the evidence that has been obtained from a consideration of gross lesions of the spinal cord, with regard to their bearing upon the paths of painful and thermal impulses, we reach the following conclusions:—

The differences between the peripheral and central loss of pain, heat, and cold, lead to the inference that in the spinal cord sensory impulses for pain, heat, and cold, are carried up by an entirely fresh system, the secondary sensory neurons.

The cell bodies of these neurons lie in the posterior horns of the spinal grey matter, and their axons almost immediately cross to the opposite side in the posterior grey commissure. This crossing is not however complete for the legs until the first lumbar segment is reached.

The axons of these cells then pass out to the marginal part of the lateral column of the opposite side, and they lie rather in the anterior than in the posterior part of the lateral column.

Such then is the result of the clinical analysis. Now it happens that in the spinal cord there is a definite tract, which corresponds in every way with the requirements formulated in the clinical research.

There are only two ascending tracts in the lateral columns, the direct cerebellar tract and the tract of Gowers. Of these, the fibres of the former are derived from the cells of Clarke's column on the same side, and will therefore not be likely to carry impulses from the opposite side. It lies moreover in the posterior part of the margin of the lateral column.

The antero-lateral ascending tract of Gowers, on the other hand, has been proved to arise from the cells of the opposite posterior horn, the axons of which cross over in the posterior grey commissure. As is well known, Gowers' tract is not definitely formed until the level of the first lumbar segment. Moreover it lies in the anterior part of the margin of the lateral column.

In its further course up the spinal cord the tract of Gowers, as has been shown by many observers, becomes more and more dorsal as it proceeds upwards, and then passes through the medulla and pons, a portion of its fibres entering the cerebellum with the superior cerebellar peduncle. But several neurologists have lately shown that a large number of the fibres of this tract pass on to the corpora quadrigemina, the optic thalamus, and the other basal ganglia.

We may therefore conclude that all the evidence, clinical and anatomical, goes to show that the paths for pain and temperature impulses are conducted up the spinal cord in the antero-lateral ascending tract. This view has always been maintained by Gowers himself.

CHAPTER IX.

The Path of the Fibres Conveying Impulses of Tactile Sensibility.

In a previous chapter evidence has been brought forward to show that when the two forms of tactile sensibility, namely, "light touch" and "pressure," are destroyed by gross disease within the spinal cord, they are affected equally. Sensibility to cotton wool touch and to pressure may therefore be considered together.

At its entrance into the spinal cord, the path of tactile sensibility cannot run with the paths of "pain," "heat," and "cold." The dissociated anæsthesia, so constantly found in cases of syringomyelia, shows conclusively that the paths for these two groups of afferent impulses must separate almost as soon as the posterior root fibres enter the spinal cord. Consequently it is almost certain that the fibres for conveying impulses of light touch and pressure pass at once into the posterior column of the same side.

The following instances show that this supposition is probably correct:—

I refer to the cases in which a loss of sensibility to pain, heat, and cold was produced by a lesion of the posterior horn without any loss of pressure sensibility. The cases of Dercum and Spiller (18), of Fürstner and Zacher (19), and of Déjerine and Sottas (20), *vide* p. 35, all show that the posterior horn may be destroyed on the same side as the analgesia, without any loss of tactile sensibility.

In Critzmann's case (38) (Essais sur la syringomyélie), thermo-anæsthesia and analgesia were found on the arms, though sensibility to pressure and muscular movement was normal. In the fifth, sixth, and seventh cervical segments the posterior horns were almost completely destroyed, the spinal cord having the appearance of a narrow ring around a large central cavity.

From these cases it is clear that the paths for the impulses subserving sensibility to temperature and to pressure cannot run together at their entrance into the spinal cord, and it is almost certain that the path of tactile sensibility, after the posterior root has entered the cord, runs into the posterior columns so quickly that it usually escapes when the posterior horn is destroyed. If however, in consequence of increased destruction of the posterior horn, the posterior column is completely isolated from the remainder of the cord, tactile sensibility is affected.

Thus Korb (39) (Ueber einen Fall von Syringomyelie mit Sections-befund) found in the arms a minimal loss of pressure sensibility. In the cervical cord one posterior horn was destroyed, and the posterior columns were completely isolated from the rest of the cord by the cavity.

To study the further course of the path of tactile sensibility it will be necessary to investigate the nature of the loss of sensation in parts of the skin innervated from the normal spinal cord below the lesion. The fact that sensibility in the legs may be normal, although the grey matter is completely destroyed in the cervical region, is proved by the evidence of many *post-mortem* findings collected in the previous chapter. Consequently the path of tactile sensibility must be sought in the white substance.

In studying the literature concerned with tactile anæsthesia in cases of gross lesions of the spinal cord, two points stand out clearly.

First, that, when tactile anæsthesia of the skin below the lesion does occur, it occurs on the opposite side of the body to the lesion; and

Secondly, that a large extent of the cross section of the cord must be destroyed to produce

any tactile anæsthesia at all, and that, as a consequence, it is rarely found in lesions of the cord that are merely partial.

As the first of these conclusions is extremely important, the evidence for it is set out in a tabular form below :—

Only one case in the seventeen of my series showed any loss of tactile sensibility in segments supplied from below the lesion. This was the case of H. L., in whom a tumour of the spinal cord at first caused paralysis of the left hand. On the right side of the body below the lesion, tactile sensibility was completely abolished, and thermo-anæsthesia and analgesia existed on both sides of the body below the clavicles.

AUTHOR.	CASE.	MOTOR LOSS.	SENSORY LOSS.
R. F. Woods (40).	Knife stab in the median line between the fifth and sixth cervical vertebræ.	Paralysis of the left leg. Muscular sense diminished.	On the right leg, thermo-anæsthesia, analgesia, and loss of tactile sensibility.
F. Hartman (41).	Injury of cord following a fall.	Paralysis of right leg. Paresis of left leg.	Total loss of sensation in the left leg.
L. Urriola (42).	Knife stab between the seventh and eighth thoracic vertebræ.	At first paraplegia, then only paresis of left leg.	Total loss of sensasation on the right leg.
Larrabee (43).	Syphilis.	Paralysis of left leg. Paresis of right leg.	Total loss of pain, cold, heat, and pressure on right leg.
Capoulade (44).	Knife stab on right side of seventh cervical vertebræ.	Paralysis of left leg. Paresis of right leg.	Total anæsthesia of right leg to pressure and thermal stimuli.
Fischer (45).	Knife stab between sixth and seventh cervical vertebræ on the left side.	Paralysis of right leg. Paresis of left leg.	Total anæsthesia on the left leg.
Burresi (46).	Knife stab on right side between 7th cervical and first thoracic vertebræ.	At first paraplegia, then paresis of left leg.	Analgesia thermo-anæsthesia and loss of pressure on right leg.
Alessandrini (47).	Knife stab in region between fifth and sixth cervical vertebræ.	Paresis of left arm. Paralysis of right leg.	Warmth, pain, and pressure sense lost on left side.
Brown-Séquard (48).	Followed a fall on the back.	Paralysis of right leg and both arms.	Total anæsthesia of left leg.
Brissaud (49).	Fracture of spine.	Paralysis of right leg.	Total anæsthesia of left leg.

Other cases could be added to this list, but enough have been brought forward to show that, when the pressure sense is lost in an apparently unilateral lesion of the spinal cord, it is lost on the opposite side to the lesion, while sensibility on the side of the lesion is unimpaired.

This must dispose of the commonly accepted view that the pressure sense travels up in the posterior columns alone. For in cases of section of the posterior roots, the posterior column degenerates upwards on the same side, and no degeneration is found in the opposite posterior column. Thus a lesion of one posterior column must produce loss of sensation on the same side of the body. Hence it is impossible that the pressure impulses can travel up in the posterior column of the same side only.

The pressure sense is not lost even when both posterior columns are destroyed. This is well seen in Long's case, already described, *vide* p. 40, where a lesion of both posterior columns at the sixth thoracic segment caused no loss of pressure sense in the left leg.

The above contention is maintained by the case of Charcot and Gombault (50) (Note sur un cas des lésions disséminées des centres nerveux chez une femme syphilitique). This was a case of syphilitic disease of the spinal cord. On the left leg there was a loss of the muscular sense, and partial paralysis. The right leg was not paralysed, but there was analgesia of that limb. Pressure sensibility was normal everywhere. At the level of the third thoracic segment a patch of sclerosis was situated, which had completely destroyed both posterior columns. The whole of the left half of the spinal cord was destroyed, with the exception of the left anterior column.

This case shows that tactile sensibility is not destroyed by a complete bilateral lesion of the posterior columns. The paresis of the left leg was caused by the lesion of the left pyramidal tract, and the analgesia on the right leg by the destruction of the left antero-lateral tract.

That the pressure sense cannot run up in the crossed lateral column is shown by the cases of Gowers and Henneberg above described, *vide* p. 39, in which a lesion of one lateral column destroyed sensation of pain, heat, and cold on the opposite side below the lesion, but left the pressure sense unimpaired.

Can the fibres for tactile sensibility run up in the crossed anterior column? This may be completely destroyed, and yet pressure sensibility may remain normal.

I was able to make a *post-mortem* examination in the case of L. G., Case 2 of my series. He was attacked by a paraplegia of sudden onset ten days before admission. There was a loss of sensibility to pain, heat, and cold, on the trunk below the nipple line, but there was no marked loss of sensibility on the legs. There was no loss of pressure sensibility anywhere. The boy died of gangrene of the bladder and ureters.

In the cord a small tumour was found in the left anterior column at the level of the fifth thoracic segment, and there was hæmorrhage into the grey matter between the first and ninth thoracic segments. The rest of the cord was normal. The appearance of the cord at various levels is shown in the following diagrams.

The loss of the sensibility to pain, heat, and cold, was evidently caused by the hæmorrhage into the grey substance destroying the axons of the antero-lateral column in their passage through the grey matter. The anterior column was completely destroyed at the level of the fifth thoracic vertebra, and there was no loss of pressure sense.

The same conclusion may be drawn from Piatot and Cestan's case (51).

In this case the right leg was paralysed. There was loss of sensibility to painful and thermal stimuli upon the left leg, the pressure sense being normal.

Death occurred one month later. A syphilitic lesion of the cord was found at the *post-mortem* examination.

In the upper thoracic region of the cord the lesion had attained its maximum dimensions. At this level the left anterior column, the central portion of the grey substance, and almost the whole of the right lateral column had been destroyed. The destruction of the right lateral column produced paralysis of the right leg and loss of thermal and temperature sensibility on the left leg. The destruction of the left anterior column does not however appear to have caused any change in sensation.

We are thus led to the conclusion that although the anæsthesia is crossed, it cannot be produced by a lesion of the posterior, the lateral, or the anterior columns, of the opposite side of the cord alone. Hence we must assume that an alternative path for pressure impulses exists.

The suggestion which I shall endeavour to uphold is that there are two possible routes for the conduction of pressure impulses. From a consideration of the differences between the peripheral and the intraspinal groupings of tactile impulses, we have shown that it is most probable that there are secondary sensory neurons for tactile impulses in the spinal cord. I suggest that these start from cells in the posterior horn of the same side, and pass over to the opposite side of the cord to run up in the anterior column of the opposite side. Tactile impulses pass into the cord, as we have shown, along the deep and epicritic systems of fibres, and immediately ascend in the posterior column of the same side. From these fibres collaterals are given off, which arborize around the cell bodies of the sensory neurons for tactile impulses along the whole length of the cord. Thus an alternative path is provided for tactile sensibility. If a lesion of the posterior columns occurs between two regions of the cord A and B, the path of tactile sensibility from A passes by collaterals to cells in the posterior horn, and then upwards in the crossed anterior column. If, however, there is a lesion of the anterior columns between A. and B, tactile impulses from A can pass up directly in the posterior columns to the region B. Collaterals are then given off to the cells of the posterior horn concerned with tactile impulses at this level of the cord. Their axons pass over into the anterior ground-bundle of the opposite side.

The extremely able deductions, which Pétren (14) draws from his study of lesions of the spinal cord caused by knife wounds, must be mentioned here.

He analysed thirty-five cases; in twelve of these cases there was loss of sensibility to pain, heat, and cold, on the opposite side of the body to the lesion, while in the remaining twenty-three cases this loss was associated with loss of the pressure sense.

He further found that, in the twelve cases of dissociated anæsthesia, the spastic paralysis was limited to the side of the lesion, while in eighteen of the remaining cases there was paralysis or paresis of the opposite leg as well, showing that the lesion in these cases must have been sufficiently extensive to involve the opposite pyramidal tract. From this he argues that the pressure sense is not affected unless the lesion has transgressed the middle line. Pétren observed the remarkable fact that in these eighteen cases, in which total cutaneous insensibility on the opposite side of the body had been produced, the stab had passed through the skin on the side of the body opposite to the lesion of the spinal cord. Consequently, in these cases, the middle line could have been transgressed only in the hinder part of the cord.

Thus in Homén's Case (52), the patient received a stab on the left side of the middle line, at the level of the spinous process of the seventh thoracic vertebra.

There was paralysis of the right, and slight paresis of the left leg. On the left leg, sensibility to pain, heat, and cold, was lost, and pressure sensibility was impaired, the muscular sense being diminished. On the right leg the muscular sense was completely lost, the skin sensibility being normal.

The paralysis of the right leg shows that the right pyramidal tract must have been completely destroyed, while the paresis of the left leg shows that the left pyramidal tract must have been slightly injured. Since the knife passed in from the left side it must certainly have destroyed the left posterior column, the right tract of Gowers, and probably the right anterior ground-bundle. The right posterior column would probably have been destroyed also.

The loss of sensibility to pain, heat, and cold, on the left leg is explained by the lesion of the antero-lateral ascending tract on the right side; the loss of the pressure sensibility on the left leg by the simultaneous implication of the left posterior column and the right anterior ground-bundle; the loss of muscular sense on the two sides by the lesions of the posterior columns.

Although there were no *post-mortem* examinations, these cases seem to show that before the pressure sense can be lost below the lesion, the latter must include the posterior column of the same side and the anterior ground-bundle of the opposite side.

The following cases are of great interest in this connexion :—

Cases in which both posterior columns were affected, both anterior columns being intact. Pressure sensibility normal.

In Case No. 1 of my series, there was loss of sensibility to pain, heat, and cold, in the right leg, pressure sense being normal. Tactile sensibility in the left leg was normal.

On section of the cord, both posterior columns, the whole of the grey matter, and the whole of the left side of the cord, with the exception of the left anterior column, were found to be destroyed.

In this case the pressure impulses from the right leg passed up in the left anterior column, and from the left leg in the right anterior column.

Charcot and Gombault's case (50), *vide* p. 44, already described, is almost exactly similar. Here the only possible path for the pressure impulses from the right leg was in the left anterior column.

Case of unilateral lesion of the cord. No loss of pressure sense.

In Müller's case (53), (Ein Fall von solitär. Tuberculose des Rückenmarks) the whole of the right side of the cord was destroyed. On the left leg there was analgesia and thermo-anæsthesia, but the pressure sense was normal. Consequently the pressure impulses from the left leg must have escaped this lesion by passing up the spinal cord in the posterior column of the left side, and then passing out by means of collaterals to the anterior ground-bundle of the right side.

Case of destruction of both anterior columns, both posterior columns being intact. No loss of the pressure sense.

Such a case has already been referred to in Chapter V. In Hanot and Meunier's Case (32), *vide* p. 40, both anterior columns were entirely destroyed, while the posterior columns remained intact. Pressure sensibility was only very slightly diminished.

Thus we have shown that the pressure sense may be normal :—

(*a*) When both posterior columns are affected;

(*b*) When both anterior columns are affected; and

(*c*) When the posterior columns on the one side, and the anterior column on the other side are destroyed.

In the following case, tactile anæsthesia was produced by a lesion of the posterior column of the same side, and the anterior column of the opposite side. Destruction of the right anterior ground-bundle and the left posterior column produced a loss of pressure sense on the left leg.

Volkmann (54), Beitrag zur Lehre von Gliom des Rückenmarks. Paralysis and loss of muscular sense was found in the right leg. On the left leg there was thermo-anæsthesia, analgesia, and loss of pressure sense. Later, paresis of the left leg supervened. A glioma in the thoracic region

had destroyed the entire right half of the spinal cord, and had encroached upon the left side, especially in the region of the left posterior column.

In this case both possible paths for the transmission of pressure sensations from the left leg had been destroyed, with consequent loss of pressure sensibility in that leg.

The following case of Albanese (55) is somewhat similar:—

The patient received a knife stab in the region of the left masseter muscle. At first there was paralysis of all four extremities, and total loss of sensibility on the right side. Twenty-seven days afterwards a left-sided hemiplegia remained, and the pressure sense was still absent on the right side. The patient died from tetanus. Three centimetres below the top of the calamus scriptorius the following parts were cut across: the entire left half of the spinal cord, with the exception of the central part of the anterior column, the right posterior column, and the posterior half of the right lateral column.

Here the lesion of the left lateral column (left Gowers' tract) explains the loss of sensibility to pain, heat, and cold, on the right side. The loss of pressure sense on the right side is explained by the simultaneous lesion of the right posterior column and the left anterior ground-bundle.

The persistence of all forms of sensibility on the left leg is explained by the freedom from injury of Gowers' tract, and of the anterior ground-bundle on the right side.

Many other cases could be brought forward from the literature of the subject, but sufficient evidence has been given to make it probable that the pressure impulses can pass up the spinal cord in two different ways. They may continue along the central process of the cells of the posterior root ganglion until they reach the upper part of the cord, then pass off by collaterals to the secondary neuron for tactile sensibility, and so cross to the opposite side; or they may at once pass out by collaterals to the secondary neuron, and run up the spinal cord in the anterior ground-bundle of the opposite side.

I have not been able to find a properly recorded case in which pressure sensibility had been lost on one side, and in which the autopsy did not show that the posterior column on the same side and the anterior ground-bundle on the opposite side had both been destroyed.

We must now face the important question whether the alternative path herein assigned to tactile impulses is consistent with the clinical facts furnished by cases of syringomyelia.

It will be found that the hypothesis of the alternative path for tactile impulses is in accordance with the clinical findings in this disease. Here, most extensive lesions of the grey matter of the spinal cord occur, and consequently the secondary neurons for tactile impulses must be interrupted whenever the destruction of the grey matter is at all extensive. The alternative path however allows the pressure impulses to travel up the posterior column of the same side until the cavity in the grey matter has been safely passed, when they pass out by collaterals to the secondary tactile neurons, and so to the opposite side.

The objection might be raised to this hypothesis that pressure is well localised in syringomyelia. I would suggest however that the localisation of a touch is very different from the perception of the touch itself, and that the fibres subserving the former follow an entirely different path from the fibres for the latter. Thus, in a certain lesion of the cord, as has already been shown, "touch" may be perfectly perceived while its localisation may be extremely imperfect, the touch as felt being erroneously referred to an entirely different part of the body. This constitutes the well-known phenomenon of "allocheiria."

Thus a tactile stimulus in the legs is interpreted in the sensorium as a tactile sensation with equal clearness, whether the secondary tactile neuron conveying the impulse has its origin in the lumbar or in the cervical region.

The fibres subserving localisation of tactile stimuli almost certainly pass up to the gracile and cuneate nuclei of the same side, and thence by the fillet to the cerebrum. The consideration of the problem of localisation will form the subject of the next chapter.

CHAPTER X.

The Path of the Sensory Impulses of Locality and Extensity.

In the chapter on the grouping of sensory impulses within the spinal cord it was shown that the sense of passive position at the joints, the sense of active movement, localisation in space, and recognition of area formed a definite group in relation to the functions of the spinal cord.

At their *entrance* into the spinal cord this group of impulses passes at once into the posterior columns of the same side.

The evidence for this has already been given. It consists in the fact that the entire grey matter in the posterior horn of the same side may be destroyed without producing any loss of the sense of locality and extensity.

When however the posterior column also is destroyed, this group of sensations is lost. This was seen in the case of J. F., Case 1 of my series.

Here a glioma in the left half of the cervical region of the cord had produced a loss of sensibility to pain, heat, and cold, of the sense of area, of the power of distinguishing the two points of the compasses, and a diminution of the power of localisation in the left arm.

Somewhat similar loss of sensation may occur in syringomyelia when the posterior column has become completely isolated from the rest of the spinal cord by the enlargement of the central cavity.

Of greater interest are the cases in which a local lesion of the cord has produced a loss of this group of sensations in the parts below the lesion.

In the table given in Chapter VI., p. 31, it will be seen that in unilateral lesions of the spinal cord this group of sensations was lost on the paralysed side, and therefore on the opposite side to that on which painful, thermal, and tactile sensibility were destroyed.

The case reported by Long (loc. cit) (33), *vide* p. 40, in which it was found on *post-mortem* examination that both posterior columns were destroyed, showed a loss of the muscular sense in both legs. On the other hand, in the case of Hanot and Meunier (32), *vide* p. 40, in which almost the whole cord with the exception of the posterior columns was destroyed, the muscular sense was perfectly normal.

These cases show with almost mathematical certainty that the impulses for locality and extensity pass up in the posterior column. The evidence of unilateral lesions also proves that these sensations pass up in the posterior column of the same side. This of course is in accord with the fact that section of the posterior roots produces degeneration in the posterior column of the same side only. The evidence for including among those which are lost on the same side as the lesion the impulses concerned with the power of discriminating two points is not yet conclusive. For in certain cases it has been found that a unilateral lesion of the cord produces a loss of this power upon the opposite side below the lesion. Thus in the case of F. C. (No. 5), a sudden hæmorrhage into the left side of the cord produced a loss of power in the left arm.

Over the right leg and the right side of the trunk below the level of the nipple there was a loss of sensibility to pain, heat, and cold, while tactile sensibility also was considerably diminished on this side. On the anæsthetic side it was found that the compasses could not be distinguished at 20 cm. apart, while on the side of the lesion there was no loss of the power of distinguishing two points. It must however be recognised that if tactile sensibility is affected, the power of distinguishing two points is seriously impaired. It is probable that in this case the inability to distinguish the two points on the right side was due to the diminished tactile sensibility on that side. Thus it seems most probable that the power of distinguishing the two points of the compasses is lost on the opposite side to the lesion only when tactile sensibility is impaired on that side. When tactile sensibility is perfect in both legs, this power is lost in association with the loss of muscular and articular sensibility on the same side as the lesion.

The pathology of tabes dorsalis is also greatly in favour of this conclusion. Here the great outstanding result of all observations is that the sensory loss is mainly that of the sense of locality and extensity, while changes in cutaneous sensibility are not usually found until late in the course of the disease. This is strikingly illustrated in the case referred to by Head in his Marshall Hall lecture. A case of "arm tabes" showed complete inability to distinguish the points of a pair of compasses separated for 15 cm. on the skin of the forearm, where they are normally well recognised at 3 cm. apart. Over the same area touch was recognised with extreme ease, both light cotton wool touch and pressure being at once discriminated by the patient. All the evidence goes to show that the fibres conveying impulses of locality and extensity pass up in the posterior column of the same side.

The further course of these sensory impulses has been demonstrated by many neurologists. There seems to be a cerebral and a cerebellar path. The former begins in the nuclei of the posterior columns, decussates, and ascends to the basal ganglia and the cerebral cortex as the fillet. The latter begins in the cells of Clarke's column, around which collaterals from the posterior column fibres arborize. The axons of secondary neurons arising from these cells of Clarke (posterior vesicular column) pass outward to form the direct cerebellar tract, which runs up as a well-defined bundle of fibres to pass into the cerebellum among the fibres of the restiform body. By these paths the afferent fibres concerned in localisation are distributed to the cerebrum and to the cerebellum.

The conclusions here set forward are strikingly illustrated by the case of C. B., *aet.* 59, who was first seen by Dr. Head on May 21st, 1905.

On April 11th, 1904, he was thrown out of a trap and pitched on his head. He was unconscious for a second or two only. He could not get up because his right arm and leg were completely paralysed. There was no loss of speech, and consciousness was absent only for a few moments. Power in the right leg gradually returned, and he was able to get about after four months. The right arm recovered its power rather sooner, so that he was able to write two months after the accident. Ever since the accident he has been unable to feel pain, heat, or cold, on the left side of his body. There is a certain amount of burning sensation in the right hand and foot, and also in the left hip.

On May 30th, 1905, his condition was as follows:—

Motion. He walked with a hemiplegic gait, swinging the right hip and lifting the right foot, so that his toes might clear the ground. As he lay on the couch there was very little loss of power in the muscles of the right foot and leg against resistance. He could dorsiflex the right foot. The muscles of the right hand were not wasted, but the right grasp was decidedly weaker than the left. Movements at the elbow and wrist were fair, but were weaker than those on the left side. The supra- and infraspinati were somewhat wasted, and there was a little wasting of the right trapezius and the right deltoid, but the right shoulder was partly fixed by adhesions.

Reflexes.	Right knee jerk much exaggerated.	Left diminished.
	Right, ankle clonus.	Left, no ankle clonus.
	Right toe goes up.	Left toe goes down.
	Right wrist jerk stronger than left.	

The sphincters acted normally.

The cranial nerves and fundi were normal.

Sensation. He complained that he had a burning sensation all down the left leg.

On this leg there was absolute loss of sensation to pain, heat, and cold in all forms below the line marked. The border on the arm could be marked out very accurately in the proximal parts, but on the distal parts seemed to fade into normal areas.

All degrees of temperature sensibility were lost; the loss of sensibility to water at 20° C corresponded to the area of loss of sensibility to stimulation by ice, by water at 38° C, and by water at 50° C.

Deep touch was everywhere felt. Tuning fork was good everywhere and localisation was quick and accurate. Sense of passive position was equal on the two sides. Cotton wool touch was perfectly recognised on both sides.

On examination of the spine, the second vertebral spine was found to be sunk in consequence of the collapse of the third cervical vertebra. The first cervical vertebra had shifted forwards. A radiograph showed a fracture of the third cervical vertebra, which was dislocated forwards.

Compasses. The two points were perfectly recognised on the right and left palm at 1 cm. apart.

Electrical. Everywhere on the left half of the body he felt the current, but a strong current produced a sensation similar in character to a weak one. He said that the current gave him exactly the same feeling as when he got hold of a hot plate. There was no pain even with strong faradism. All his muscles reacted perfectly to faradism, and he could perceive well the contraction of the muscles on both sides of the body.

The condition was clearly a lesion of the right lateral column, causing spastic paralysis of the right arm and leg, and loss of thermal and painful sensibility on the left arm and left leg.

An operation was performed on his cervical spine by Sir Victor Horsley on June 25th, 1905. The spines of the second and third cervical vertebræ were removed. He was well and free from pain for two weeks, but after this period he noticed that the right side was stiff and cramped, and that the burning on the left side increased. A burning heat or gnawing pain seized him soon after meals. The right hand appreciated heat and cold, but it had a feeling of puffiness, "as if you had been washing in warm water for a long time." All the way up the right arm and leg—"it is exactly as if it was a cork leg—the muscles feel tight," and he thought he had lost power in the right side.

On October 19th, 1905, he walked with a definite hemiplegic gait. He could stand on the right foot, but did so with difficulty, and tended to fall when his eyes were closed. There was weakness of the right hand grasp. Movements of the right foot were performed less easily than the left. There was definite slight inco-ordination of the right hand. He had difficulty in touching his nose with his right forefinger. When he tried to bring the two fingers together with his eyes closed, the right hand wandered. There was no wasting and no tremor.

SENSATION.

Left side. Light touch perfect and localisation good. There was loss of heat and cold in all forms. Deep touch was perfect, and the compasses excellent. Tuning fork was good, and the sense of passive position perfect.

Right side. Light touch, heat, and cold, were all perfect. The sense of passive position was absent in the fingers and the thumb, also in the toes and ankle. There was deep tenderness to pressure on the right side.

Right knee jerk, much exaggerated.	Left, normal.
Right, ankle clonus.	Left, absent.
Right toes go up.	Left toes go down.

As the result of the operation the sense of passive position on the right side was lost, inco-ordination of the right hand appeared, and he stated that he felt as if the right leg was made of cork.

This points to a lesion of the right posterior column of the spinal cord. The operation relieved the pressure, but the subsequent scar tissue seems to have pressed upon the right posterior column and produced a loss of the sense of position in the right leg.

Spearman (56) gives an account of a case of unilateral injury to the cord. The case is treated from a psychological point of view, and is chiefly interesting for the very close analysis of the processes concerned in localisation.

Briefly the case is as follows:—

S, a miner, *aet.* 45, presented himself at the Leipzig Nerve Hospital on September 12th, 1903. Twenty-six years before he had been stabbed in the back with a knife, and for some time he had been unable to use his left arm. This, however, improved, and he had not been further troubled until two-and-a-half years before admission, when he began to be conscious of weakness in the left foot, and this weakness spread up the left leg.

The symptoms found were of the Brown-Séquard type, and examination by the Röntgen rays revealed a foreign body within the spinal canal at the level of the sixth cervical vertebra. At operation this proved to be a rusty knife point, which pressed upon, but did not sever, the dura mater on the left side of the spinal cord. Numerous tests were applied by Spearman, and the general result of his investigations may be summarised as follows:—

Motion. There was reduction in strength and restriction of movement in both legs, but this was much more marked on the left side. There was marked ataxia on the left side, very little on the right.

Sensation. Sensibility to pain, touch, and temperature was lost over the lower part of the body below a horizontal line 10 cm. above the navel, and over the right leg; over the left leg sensibility to touch was slightly diminished.

The *sense of movement* was lost in the left leg. The knee failed to distinguish a movement through twenty degrees at the rate of one degree a second. The left ankle failed to appreciate a rotation through forty degrees. On the right leg the patient was able to distinguish a rotation through 0·8° at the knee and 1·2° degrees at the ankle; these amounts are quite normal.

The *sense of passive position* was absolutely lost in the left knee and ankle joints, the patient being quite unable to appreciate the position of the leg or foot.

On the right side he was normal in this respect.

With the *compasses* the right leg was slightly better than the left, but nearly normal values were obtained. After exercise the threshold distance was much increased.

Localisation by "spot finding" was tested in three different ways.

(1) The simple method. The patient's eyes were closed, and the leg was covered by a large piece of cardboard at a distance of 1 cm. from the skin. In the centre of the cardboard was a hole; through the hole a bent wire 2 mm. thick was introduced and the patient's skin was touched. He was given a stylographic pen, and his eyes still being closed, he was told to mark on the cardboard the place where he had been touched.

(2) Volkmann's "looking" method. "The patient's eyes are closed; some selected spot is touched for one second; a pause is given long enough for the mark made to die away; then he is told to open his eyes and point out the place where he had been stimulated, without, however, touching the skin with his finger."

(3) Weber's "groping" method. "The patient's eyes are closed and he is touched and then allowed to grope for the spot. The results were found to be the same whether the patient used his finger or a wire or stick."

By the first of these methods, the power of localisation on the left leg was found to be very greatly impaired, while on the right leg it was slightly impaired.

By the second and third methods localisation, though somewhat poor, was almost equal on both legs.

The following example of the actual values obtained is taken from Spearman's article :— CALVES.

TEST.	LEFT.	RIGHT.
Motor power.	Diminished.	Good.
Ataxia.	Present.	Absent.
Passive position.	Not recognised	Recognised.
Pain, heat, and cold.	Good.	Absent.
Touch (von Frey's hair test) (normally 5.8 gm.)	7.2 gm.	14.4 gm.
Compasses (4 cm.)	3.5 cm.	3.5 cm.
Localisation. Simple (3.5 cm.)	10 cm.	4.6 cm.
Looking (2 cm.)	3.1 cm.	3.6 cm.
Groping (1.5 cm.)	3.6 cm.	4.2 cm.
Movement of knee (0.8°)	20°	0.8°

The results are striking when looked at as a whole. On the *non-paralysed* side, pain, heat, cold, and touch were lost. On the *paralysed* side, the sense of movement, the sense of passive position, localisation, and the power of distinguishing the points of a pair of compasses were much more affected than on the non-paralysed side.

The importance of the case for the determination of the paths of sensory impulses, of course, cannot be great, as the spinal cord was not actually severed, and its interest lies chiefly in the differences obtained when the different methods of localisation were used.

Spearman's analysis leads him to the following conclusions :—

(A) The so-called "simple" method of testing localisation furnishes the least complicated method of ascertaining the perception of "thereness." The processes consist in the co-ordination of the tactile sensation with subconscious articular sensations from the chain of joints extending from the finger to the point touched.

(B) In the "groping" method of Weber, where the patient is allowed to grope for the spot stimulated with his eyes shut, a complication is introduced. The articular sensations are conveyed as before; this alone is responsible for the finger's original movement towards the spot stimulated. Contact with the skin, however, immediately reveals a discrepancy and the pointer is moved nearer to the spot stimulated. In this second or corrective phase, there arises a tactile mental image of the leg consequent on contact between the skin and the groping stick. This is compared with the tactile image obtained from the original stimulation of the skin, and thus the pointer is guided nearer to the place of the original stimulus.

(C.) In the "looking" method of Volkmann the first spatial idea is that derived from the articular sensations. As soon as the patient opens his eyes and sees the limb he compares the picture with the mental image aroused by the touch, and in this way can localise the touch without the aid of the articular sensations.

The results of testing localisation by the three methods seems to Spearman to be at variance with Förster's view that localising power is not dependent upon contact sensibility at all, but wholly upon movement sensations (Bewegungs-empfindungen).

The first method, where articular sensations alone were concerned, showed an exact correspondence between the loss of articular sensation and the power of localisation. On the other hand, the two other

methods failed to give this result. On both legs the results were less accurate than the normal, but differed little from one another.

I would suggest that this may be due to the greater diminution of tactile sensibility on the non-paralysed side, that is to say, the side where articular sensation was intact. This loss of tactile sensibility would no doubt interfere with the production of a tactile mental image of the affected limb, and for this reason the error in localisation on the right side is greater than it would have been if tactile sensibility had been normal.

I consider that the discrepancy in the results obtained by the first and the other two methods may thus be explained. Consequently the reduction in the power of localisation on the left side may be explained by the associated loss of articular sensibility, while that on the right side, when tested by the two methods dependent on the formation of a tactile mental image, depends on the diminution in the tactile sensibility on this side. If the three tests were performed upon a patient, in whom tactile sensibility was normal in both legs, while articular sensibility was absent in one leg only, the result should show a loss of localising power on this leg only. The impairment of tactile sensibility on the opposite leg introduces a complication, which invalidates the methods that depend upon the comparison of tactile mental images.

Here we must refer to the experiments of Mott (8) upon hemisection of the cord in monkeys. These excellent experiments were most carefully performed, and the methods of testing the animals are accurately described. Although Dr. Mott considered that his experiments were in opposition to the views of Brown-Séquard, they need only to be carefully considered to show that they are in complete agreement with the work of that great observer.

The result of Dr. Mott's beautiful experiments, when the monkeys were examined after death, showed that in nearly every case he had succeeded in making a complete hemisection of the cord, without encroaching on the opposite side to any appreciable extent.

In this series of experiments, we have, then, to deal with a complete hemisection of the spinal cord. The results are summed up by Dr. Mott in these words :—

When tested by heat, pricking, and stimulation by faradism, it was very difficult to decide, except in one case (Case 5), whether the animal felt better on the side of the section or on the opposite side. There was usually a delay before the animal responded on the paralysed side. On the non-paralysed side, the day after the operation, the animal, when tested, was often noticed to scratch the part irritated and try with its hand to remove the cause of irritation—a certain sign of conscious sensation and localisation. The *clip test* applied to most of the animals was the most valuable sensory test. It invariably showed the same result, namely, that the clip was not removed from the paralysed foot, but always removed from the non-paralysed, although various means were adopted by which the animal could not possibly have seen the clips applied, such as application when the animal was unconscious with ether; application when the animal's head was covered by the assistant's coat, or when its attention was engaged with raisins or apples; the result was always the same. The animal for some weeks after the operation always removed the clip from the non-paralysed side and never from the paralysed, unless he happened to see it. The peculiar phenomenon of allocheiria was noticed in nearly all cases, when this test was applied.

Dr. Mott concludes from his experiments :—

(1) That painful sensations can pass up either side of the cord.

(2) That sense of position in space passes up the same side.

The second of Dr. Mott's conclusions is in entire agreement with Brown-Séquard, and falls in with the instances I have already detailed. It is the former of his conclusions which is so directly at variance with the results of clinical observations in man.

Before proceeding to show how Mott was led to this erroneous conclusion, I would refer to an italicised remark made on page 7 of his paper: "All physiologists, who have had experience of testing the sensibility of the skin in monkeys, are agreed that the animals often behave indifferently to *pricking* and other sensory tests." Consequently it would be very difficult to say, on pricking the sole of a monkey's foot, whether the animal felt a painful or merely a tactile sensation.

According to the views put forward in this Thesis, assuming that conduction of sensory impulses in the monkey is similar to that in man, complete hemisection of the cord should give rise to the following sensory changes in parts below the lesion:—

(1) Loss of painful and thermal sensibility on the opposite side to the lesion. This would be caused by the lesion of the antero-lateral tract of Gowers.

(2) No loss on either leg of tactile sensibility. According to our hypothesis, tactile impulses from the skin on the side of the lesion would pass up in the opposite anterior ground-bundle. Tactile impulses from the opposite side to the lesion would pass up in the posterior column of this side.

(3) Entire loss of the sense of locality and extensity on the side of the lesion. This would be caused by the lesion of the posterior column of this side.

Dr. Mott pricked the animal with a needle on both feet, after having hemisected its cord. On the side of the lesion the animal would experience a painful and a tactile sensation, but would be unable to localise it. On the opposite side to the lesion the animal would experience a tactile but not a painful sensation, and would be quite able to localise the stimulus.

The animal was unfortunately unable to tell Dr. Mott whether it felt pain, or merely a touch; and bearing in mind Dr. Mott's remark about the indifferent response often made by monkeys to painful and other stimuli, it will be realised how difficult it was for the observer to find any difference between the behaviour of the monkey when pricked first upon one foot and then upon the other. The animal removed the clip from the non-paralysed leg and not from the paralysed side merely because it was able to localise the stimulus on the non-paralysed and unable to do so on the paralysed side.

We may therefore conclude that Dr. Mott's experiments are not in any way opposed to the theory of the complete decussation of painful impulses.

These considerations show how carefully animal experiments must be scrutinised before the results are applied to man. The response to so complicated a stimulus as a pin-prick can really be ascertained in man only by word of mouth. Thus the sensations produced by a pin-prick include:—

(1) Painful sensations.

(2) Acuæsthesia, that is to say, recognition of the sharpness of the point, apart from pain.

(3) Tactile sensations.

(4) Localisation of the place stimulated.

So complex a group can clearly be examined satisfactorily only in subjects who can distinguish and communicate its various components by means of speech.

Case 5 of Dr. Mott's series, upon which he lays great stress, occurred in a large tame Rhesus, where he found "almost complete abolition of sensation to heat and pricking on the paralysed side." This, as Dr. Mott himself remarks, may be explained by the almost total destruction of one half of the cord for some little distance, and the extensive degeneration of the anterior column of the opposite side. There was a well-marked degeneration of the antero-lateral tract on both sides, extending up into the medulla, but more marked on the side of the lesion. Consequently on the paralysed side, according to the conception of sensory conduction laid down in this Thesis, the following conditions would be present:—

(1.) Complete loss of all tactile sensibility due to the simultaneous implication of the posterior column on the side of the lesion and the anterior column of the opposite side.

(2) Diminution of sensibility to pain, heat, and cold, since the antero-lateral tract on the opposite side to the lesion was partly degenerated.

(3) Loss of all power of localisation due to section of the posterior column on the side of the lesion.

On the non-paralysed side there would be loss of pain, heat, and cold sensibility, but tactile sensibility and localisation would be intact.

It is not surprising, therefore, that in this case sensation was found to be more affected on the side of the lesion than on the opposite side. The loss of the sense of locality on the side of the lesion found by Mott in these monkeys is in complete agreement with the clinical results obtained in man; the phenomenon of allocheiria is due to the loss of the sense of position in the paralysed limb, and the consequent localisation of the sensation felt in a part where this sense was intact, namely, the opposite limb.

The results, therefore, of both animal experiments (rightly interpreted), and of clinical observations in man, show that the path for the sense of locality and extensity passes up in the posterior column of the same side.

It must, however, again be emphasised that as regards sensation the posterior column is a mixed column, and the fibres it contains are really the central processes of *peripheral* nerves.

The cells of the secondary sensory neurons are to be looked for in the gracile and cuneate nuclei. Just as do the secondary sensory neurons in the cord, their axons at once decussate and pass up towards the sensorium as the fillet of the opposite side.

SUMMARY.

In this Thesis an attempt has been made to determine the character of the loss of sensation which is produced by gross lesions of the spinal cord.

The results arrived at can be collected together, and summarised shortly, as follows:—

(1) A lesion of the grey matter of the spinal cord produces loss of sensation at the level of the lesion. No loss of sensation is produced below the lesion. Painful and thermal sensibility are alone destroyed, tactile and muscular sensibility remaining normal.

(2) A lesion of one posterior column produces a loss of the sense of locality and extensity on the same side.

(3) A lesion of the lateral column of the cord produces a loss of painful and thermal sensibility on the opposite side below the lesion.

(4) A lesion of the anterior column produces no change in sensation below the lesion.

(5) A complete unilateral lesion therefore produces loss of the sense of locality and extensity on the same side and a loss of painful and thermal sensibility on the opposite side below the lesion. Tactile sensibility is unaltered.

(6) A lesion of the posterior column on one side, and of the anterior column on the other side, produces a loss of tactile sensibility below the lesion on the same side of the body as the lesion of the posterior column. For example, a lesion of the right posterior column and the left anterior column will produce a loss of tactile sensibility on the right leg. Unless both these columns are destroyed there will be no loss of tactile sensibility on the right leg.

(7) The work of Head, Rivers, and Sherren, has shown that on the periphery there are three systems of fibres conveying sensory impulses to the spinal cord. These are known as the systems of fibres which conduct the impulses of protopathic, epicritic, and deep sensibility.

Along the protopathic fibres run the impulses conveying sensations of cutaneous pain, and of the extremes of heat and cold.

Along the epicritic fibres run the impulses concerned with the recognition of light touch, of minor degrees of heat and cold, of the two points of a pair of compasses, and of area and form.

Along the fibres of deep sensibility run the impulses concerned with pressure, with localisation, with the movements and position of the joints and muscles, and with the pain of deep pressure.

The intraspinal terminations of these fibres have been studied in this Thesis.

The protopathic fibres run at once into the grey matter of the posterior horn and soon end by breaking up around its cells.

The fibres for epicritic and deep sensibility pass at once into the posterior column, and run up in this column for the whole length of the spinal cord. They end in the nuclei of the posterior columns, namely, the cuneate and gracile nuclei.

(8) An analysis of the various lesions of the spinal cord shows that the ascending paths in the cord are probably three in number. One carries up painful and thermal impulses of all forms, a second carries up tactile impulses, while the third carries the impulses concerned with locality and extensity.

(9) The path for painful and thermal impulses enters the grey matter of the posterior horn, passes to the opposite side in the posterior grey commissure, and finally ascends in the anterior and marginal part of the lateral column of the opposite side. The paths from the lower extremities do not cross until the level of the first lumbar segment.

Anatomically this path is represented by a part of the antero-lateral ascending tract of Gowers.

(10) The path of tactile impulses is rather more complex: for these impulses an alternative path is open. They may run up either in the posterior column of the same side or in the anterior ground-bundle of the opposite side.

In the posterior column of the same side tactile impulses pass up along the fibres for deep and epicritic sensibility, in reality the central processes of peripheral nerves. All the way up the cord collaterals are given off from these long fibres. These arborize round the cell bodies of new neurons, and the axons of these cells pass to the opposite side of the cord and run up in the anterior ground-bundle.

Like the impulses for pain, heat, and cold, this crossing is not complete until the first lumbar segment.

(11) The path of the impulses concerned with locality and extensity lies in the posterior column of the same side.

The investigations undertaken for this Thesis place the conduction of sensory impulses upon a uniform basis.

Sensory impulses are carried to the brain by means of relays of neurons. The primary sensory neurons have their cell bodies in the posterior spinal ganglia. The results of embryological studies show that the peripheral sensory nerves represent the dentrites of these cells, while the intraspinal processes are the axons. These axons enter the spinal cord and arborize round the cell bodies of secondary sensory neurons.

The cell bodies of the secondary sensory neurons form a homogeneous system of cells.

They consist of the cells of the gracile and cuneate nuclei and of the cells of the posterior horn. Morphologically these cells form a compact group of identical origin.

The secondary sensory neurons cross to the opposite side of the cord soon after their origin, and then pass up to the basal ganglia of the cerebrum and cerebellum.

The secondary sensory neurons for pain and temperature impulses arise in cells of the posterior horn. Collaterals from the protopathic, epicritic, and deep fibres, arborize round these cells. Their axons cross the spinal cord in the posterior grey commissure, and run up in the antero-lateral ascending tract of the opposite side.

The secondary sensory neurons for tactile impulses arise from cells in the posterior horn. Along the whole length of the spinal cord the fibres for deep and epicritic sensibility, as they run up in the posterior columns, send collaterals to arborize round these cells. The axons of these secondary sensory neurons cross the cord, and pass up in the anterior ground-bundle on the opposite side. This arrangement provides an alternate path for tactile impulses; they may run up in the posterior column of the same side along the axon of a primary sensory neuron, or may pass up in the anterior ground-bundle of the opposite side in the axon of a secondary sensory neuron.

The secondary sensory neurons for impulses concerned with locality and extensity have their cell bodies in the gracile and cuneate nuclei. The terminations of the epicritic and deep sensibility fibres arborize round these cells. The axons of these cells cross over to the opposite side in the superior or sensory decussation, and are then known as the fillet. A second set of secondary sensory neurons arise from the cells of the posterior vesicular column, the axons of which pass up to the cerebellum as the direct cerebellar tract.

As Dr. Mott remarks in his paper on the results of hemisection of the cord in monkeys, the ultimate solution of the question of sensory conduction rests upon an accurate record of cases of disease and injury in man. I venture to hope that the views put forward in this Thesis have placed the conduction of sensory impulses within the spinal cord upon a uniform and intelligible basis.

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SUMMARY OF CASES.

TUMOURS OF THE SPINAL CORD	4
SYRINGOMYELIA	8
MORVAN'S DISEASE	2
UNILATERAL CORD LESIONS	5
SYPHILITIC DISEASE OF SPINAL CORD	1
Total number of cases	20

SHORT ABSTRACTS OF THE CASES.

Case 1. Glio-Sarcoma of Cervical Cord.

J. F. (male), *aet.* 44, under the care of Dr. Dawson.

The total duration of the illness was eleven months. The first symptom was weakness of the left hand. Incontinence of urine and fæces was present. Dysphagia. Bronchitis. Death.

Motor Symptoms.

Flaccid paralysis of left arm. Spastic paralysis of both legs, especially the left.

Sensory Symptoms.

Almost complete anæsthesia to all forms of stimulation in left arm and left side of neck.

Thermo-anæsthesia and analgesia on the right side of the body below the lesion.

Sense of passive position deficient in left leg. Examination of cord showed a glio-sarcoma extending from medulla to mid-thoracic region. Maximum point at level of third cervical segment. The whole of left side of cord, with the exception of the left anterior column, was destroyed. The right posterior column was partly destroyed.

Case 2. Glioma of Dorsal Cord. Hæmatomyelia.

L. G. (male), *aet.* 18, under the care of Dr. Warner.

The total duration of the illness was seventeen days. Paraplegia and retention of urine suddenly developed.

The legs were blistered, and the urine drawn off without aseptic precautions.

Death ensued from gangrenous cystitis and peritonitis.

Motor Symptoms.

Total flaccid paralysis of both legs. Reflexes absent.

Sensory Symptoms.

No change on the legs. Over the trunk below a horizontal line at the nipple level there was thermo-anæsthesia and analgesia. No loss of the pressure sense.

Examination of the cord showed a small tumour at the level of the fifth thoracic segment. It had destroyed the left anterior horn and adjacent parts of the left anterior column. There was a small hæmorrhage in the central grey matter extending from the first to the ninth dorsal segment.

Case 3. Glioma of the Cervical Cord.

H. L. (male), *aet.* 30, under the care of Dr. Schorstein.

The total duration of the illness was six months. Weakness of the left arm and leg were the first symptoms noticed. Incontinence of urine and fæces supervened. Death occurred from septic bronchitis. He had no pain.

Motor Symptoms.

Spastic paralysis of both legs. Flaccid paralysis of both arms. Respiration accomplished solely by diaphragm.

Sensory Symptoms.

No loss of sensation for one month. Then loss of painful and thermal sensibility of right arm, then thermo-anæsthesia and analgesia below the clavicles.

Loss of pressure sense on the right side of the body and a part of the right arm.

Loss of sense of passive position in fingers, wrists, elbows, hips. Good in other joints.

Two points could not be discriminated over either arm.

Since writing this Thesis, the patient has died. Examination of the cord showed a large intramedullary tumour at the level of the fifth cervical segment. The tumour extended along the whole length of the spinal cord.

Case 4. Tumour of Cervical Cord.

E. L. T. (female), *aet.* 24, under the care of Dr. Wall.

The duration of illness to present time was fourteen months. She first noticed wasting and loss of power in the left hand. Then the left leg, and afterwards the right, began to be weak. Incontinence of urine and fæces.

Motor Symptoms.

Spastic paralysis of both legs.

Paralysis and wasting of both arms; the left arm is more wasted than the right.

Sensory Symptoms.

Sensation was not affected for nine months. Then a loss of painful and thermal sensibility was found over the right side of the trunk and right leg.

Then thermo-anæsthesia and analgesia of both sides of trunk and right leg.

Loss of deep painful sensibility over the right anterior superior spine, but not over the left.

Recognition of passive position deficient in the left toes and left ankle.

Case 5. Unilateral Lesion of the Cord. Symptoms of the Brown-Séquard Type.

F. C. (male), *aet.* 37, under the care of Dr. Percy Kidd.

The onset occurred during sleep, on December 13th, 1903.

Weakness of the left hand and arm, with loss of sensation on the right side of the body and the right leg.

When seen on August 17th, 1903, he was suffering from acute rheumatism. He had a previous attack seventeen years ago.

The heart showed well-compensated mitral regurgitation.

Motor Symptoms.

Weakness of left arm.

Sensory Symptoms.

Subjective sensations of warmth in right leg.

Over the right leg and right side of the body, below a line midway between the nipple and umbilicus, there was analgesia and thermo-anæsthesia. Tactile sensibility was somewhat diminished.

The power of discriminating two points was deficient on the right side of the abdomen.

There was no loss of the sense of passive position.

Case 6. Fracture-Dislocation of the Third Cervical Vertebra. Symptoms of Unilateral Lesion of the Spinal Cord.

C. B. (male), *aet.* 59, under the care of Dr. Head.

In April, 1904, he was thrown out of a trap, and became paralysed in the right hand and arm. There was loss of sensation in the left leg.

Motor Symptoms.

Spastic paralysis of right hand and right arm.

Sensory Symptoms.

Thermo-anæsthesia and analgesia of left arm and leg.

The posterior parts of the third and fourth cervical vertebra were removed. Temporary improvement was followed by increase in the severity of the symptoms.

The physical signs remained as before, but in addition there was loss of the sense of passive position on the right side.

Case 7. Fall Down the hold of a Ship. Symptoms of Unilateral Lesion of the Spinal Cord.

A. H. (male), *aet.* 35, under the care of Mr. Rigby.

The accident occurred in February, 1902.

Motor Symptoms.

Wasting and weakness of the left arm. Spastic condition of the left leg.

Sensory Symptoms.

Thermo-anæsthesia and analgesia of the right side of the trunk and of the right leg. No loss of other forms of sensation.

The patient gradually recovered sensibility on the right leg and motion on the left side.

Case 8. Unilateral Lesion of the Cord.

G. G. (female), *aet.* 30, under the care of Dr. Head.

In March, 1898, the onset occurred during sleep and coincided with the first day of a menstrual period.

Motor Symptoms.

Spastic condition of the right leg.

Sensory Symptoms.

Loss of painful and thermal sensibility on the left leg. The upper level of anæsthesia corresponded exactly with the upper limit of the first lumbar posterior root area. The sense of passive position was lost on the right great toe and right ankle.

The condition remains stationary six years after the onset.

Case 9. Unilateral Lesion of the Cord.

C. W. F. (male), *aet.* 26, under the care of Dr. Head.

He was diving from the top of a bathing machine, when he slipped and fell head first. Paralysis of the right arm and loss of sensation on the left leg followed. The accident occurred August 14th, 1905. He was seen in November, 1905, and was rapidly improving.

Motor Symptoms.

Slight paresis of right arm.

Sensory Symptoms.

Partial analgesia on the left side below the nipple level. Total analgesia over an area corresponding to the lumbar posterior root areas.

Thermo-anæsthesia over the left leg and left side of trunk below nipple level.

Case 10. Syringomyelia.

T. B. (male), *aet.* 48, under the care of Dr. Head.

He came to the Hospital complaining of pain in and swelling of the left elbow for three days.

The muscles of his hands had been wasting for two years.

His hand and arms always feel very cold.

Motor Symptoms.

Wasting of the small muscles of the thumbs and little fingers, and of the interosseus muscles of both hands.

Electrical reactions diminished, but otherwise normal.

Sensory Symptoms.

Loss of painful and thermal sensibility over both arms, over the trunk, as far down as the nipple line, and over the neck and scalp.

No loss of sensation in the legs.

Case 11. Syringomyelia.

F. A. R. (female), *aet.* 24, under the care of Dr. Head.

In October, 1899, she complained of pain in the left shoulder and wasting of the left hand. There was then a small strip over the outer side of the left arm where thermal sensibility was absent.

In March, 1902, the wasting of the left hand had increased and the right hand had also begun to waste. The area above mentioned was now insensitive, not only to thermal but to painful stimuli.

In January, 1903, there was analgesia and thermo-anæsthesia over the greater part of the left arm and also over a small area on the right arm.

In February, 1904, both arms were affected and also the left side of the neck.

In March, 1905, there was analgesia and thermo-anæsthesia over the whole of both arms, except a narrow strip on the inner sides, and over both sides of the neck, the scalp, and the left side of the face.

Sensibility in the legs was unaffected.

Case 12. Syringomyelia with Bulbar Symptoms.

L. C. (female), *aet.* 53, under the care of Dr. F. J. Smith.

In June, 1905, she complained of loss of power in the left leg. There has been some difficulty in swallowing for the last two months.

Motor Symptoms.

Left hemiparesis.

Atrophy of the left half of the tongue.

Sensory Symptoms.

Loss of sensibility to painful and thermal stimuli over both shoulders and both sides of the neck, and also over the whole of the left side of the face and scalp.

No sensory changes in the legs, trunk, or arms.

In this case there was intermittent glycosuria.

Case 13. Syringomyelia (Sacral Region).

L. B. (female), *aet.* 17, under the care of Dr. Head.

Swelling of the left ankle and perforating ulcer of the left sole.

Motor Symptoms.

No wasting.

Sensory Symptoms.

Analgesia and thermo-anæsthesia of right foot and adjacent parts of legs.

Very marked trophic changes in the bones of the right foot.

Two perforating ulcers upon the sole of the right foot.

Case 14. Unilateral Cervical Syringomyelia.

W. H. (male), *aet.* 30, under the care of Dr. Head.

In August, 1903, he began to notice that his right arm felt colder than the left.

Motor Symptoms.

No wasting, no paralysis.

Sensory Symptoms.

Thermoanæsthesia and analgesia over the right arm, the right side of the neck, and the upper part of the right side of the trunk.

Case 15. Unilateral Cervical Syringomyelia.

C. H. (male), *aet.* 34, under the care of Dr. Head.

In December, 1903, he began to notice pain in the right shoulder. The joint subsequently became swollen.

Motor Symptoms.

No paralysis, no wasting.

Sensory Symptoms.

Thermo-anæsthesia and analgesia right arm, the right side of neck and scalp, and the upper part of the right side of the trunk.

Very marked trophic changes in the right shoulder joint.

Case 16. Syringomyelia (Pontine).

M. B. (female), *aet.* 27, under the care of Dr. Head.

Illness began in January, 1900. She noticed clumsiness in holding things, and swelling of the joints of her hands.

At her work, as a cook, she often burnt herself without feeling the burns.

In February, 1902, she was found to have well-compensated mitral stenosis. There was analgesia over both hands and both forearms.

Thermal sensibility was lost over both forearms, over the right side of the neck, face, and scalp, and there was partial loss on the left thigh and left leg.

In April, 1905, there was total thermo-anæsthesia and analgesia.

The small joints of the hands showed marked trophic changes.

The legs showed spastic changes.

Case 17. Cervical Syringomyelia.

L. G. (female), *aet.* 30, under the care of Dr. Warner.

The illness began in 1901: she noticed weakness and wasting of both hands.

In January, 1904, there was wasting of the small muscles of both hands, and thermo-anæsthesia and analgesia over both hands and forearms.

In May, 1905, the wasting of the hand muscles had increased. The anæsthetic area now included both fore-quarters and the left side of neck and scalp.

There was a loss of the power of discriminating the compass points on the left palm.

There was no loss of sensation in the legs.

Case 18. Morvan's Disease.

A. R. (male), *aet.* 34, under the care of Dr. Head.

In May, 1904, he began to notice that painless white blisters kept forming on his hands.

There was thermo-anæsthesia and analgesia over both arms, over the trunk, and over the lumbar areas on the right leg.

Trophic changes in the skin and joints of the hands were very marked.

Case 19. Morvan's Disease.

O. L. B. (female), *aet.* 6½, under the care of Dr. Kidd.

She had suffered from painless whitlows from two years of age. The ends of the fingers had been bitten off. There were trophic changes in the elbows, ankles, and small joints of the hands.

Analgesia and thermo-anæsthesia occurred in both legs and arms, but the age of the child made the testing of sensation very difficult.

Case 20. Syphilitic Myelitis.

H. A. (male), *aet.* 29, under the care of Dr. Dawson.

He contracted syphilis in June, 1902. In November, 1903, he had a severe attack of acute central myelitis. In June, 1905, there was spastic paralysis of both legs, more marked on the right side.

There was thermo-anæsthesia and analgesia on the left leg and left side of the trunk as far up as the nipple level.

Jarrold and Sons, Ltd., Printers, The Empire Press, Norwich.