TYPE AND DISTRIBUTION OF SENSORY DISTURBANCES DUE TO CEREBRAL LESION¹

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The researches of Head and others have determined typical combinations of various forms of sensory loss when due to lesion in the spinal cord. This is because of the fact that the various kinds of sensory impulses pass upward in the spinal cord in definite tracts which are well separated from each other, so that the forms of sensation which would be lost if only a part of the cord is destroyed, will usually be in fairly definite combinations. Dissociation of sensation results not only from spinal lesion; it also occurs from lesion of the peripheral nerves or of the spinal roots, but the combinations of sensory qualities lost through a peripheral nerve injury or an injury to the posterior spinal nerve root differ decidedly from those due to partial destruction of the spinal cord. Many investigations show that in cerebral affections, also, we can have a dissociation of sensation among the symptoms. The clinical studies of Gordon (1) show that following cerebral lesions, pain was the sensation most affected. Karl Schaffer (2) found the sense of motion and position frequently disturbed and speaks of defect of localization of sensation as especially characteristic of sensory defects due to subcortical lesion. Morton Prince (11) reported a case of compound fracture of the skull in the parietal region in which there was no anesthesia or analgesia but in which there was a marked defect in localization of sensation in the hand and, to a less extent, in the foot. The error of localization in the case was usually distal, which is the opposite of that found by Russel and Horsley in their cases. An interesting observation in Dr. Prince's case was the difference between the result when using the "naming" test, asking the patient to name the spot touched,

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and when using the Volkmann method, asking the patient to put his finger on the spot which had been touched. By the "naming" test the errors were marked, neither the finger nor the segment of the finger touched could be named correctly, but by the Volkmann method the localization of sensation on the palm of the hand was found nearly normal while on the dorsum the errors were not marked and were postaxial and preaxial oftener than errors as to the segment of the finger. There was also loss of the sense of pressure and of the sense of position and complete astereognosis in the affected hand. Sandberg (12) reported twenty-two cases of sensory disturbance due to cerebral lesion and found tactile sensation affected in all of them. Pain sense was retained and he calls attention to the feeling like an electric shock which these patients experienced when pricked with a needle on the affected side. The temperature sense was retained, but it took a longer time to obtain the reaction on the affected side than it did on the sound side. Errors in the localization of sensation were more marked in the "breadth" than in the "long dimension." Sandberg comes to the conclusion that the dissociation of sensation from cerebral lesion is similar in type to that seen when the posterior columns of the spinal cord are affected, "Hinterstrangtypus." Starr (3) speaks of meeting with cases of various forms of sensory dissociation due to cerebral lesion. Lewandowsky (13) reports a case of Jacksonian epilepsy followed by hemiplegia in which there was subjective sensation of coldness on the paralyzed side and he attributes this to a diminution of the temperature for heat; apparently showing that the sense for heat may be affected when that for cold is not affected, from an organic brain lesion. Klippel, Sergueeff and Weil (15) reported a case in which there was slight diminution of sensation to touch but none to pain sensation. A piece of ice was felt as pain but with no sensation of cold. Warm objects were not recognized as such but simply as contact. The application of something very hot was recognized as painful but there was no sensation of heat. There was marked defect in localizing sensation and in the sense of position. The authors regarded the symptoms as due to lesion in the optic thalamus.

It would appear however from an analysis of these studies that there was no definite combination of forms of sensation

which would be lost from a cerebral lesion but that all forms are likely to be affected on the same side of the body, the side opposite the lesion, and the form of sensation most affected will depend on the location of the lesion in the brain, whether it is cortical, subcortical, in the internal capsule or elsewhere. It may be of distinct value as a localizing sign.

The distribution of the sensory loss which was outlined by the above-named authors was a partial hemianesthesia with the more marked impairment of sensation toward distal parts of the extremities, thus the hand was more affected than the forearm, the forearm than the arm and so on, without however, any sharp lines of demarcation.

In 1904, C. K. Mills (quoted by Spiller) suggested "that the cortical sensory area for the upper limb is probably separate from that for the lower limb" and, according to the same authority, this view has been entertained by Campbell and others. Spiller (14) reported a case of a man who having received a blow on the parietal region had sensory disturbances in the hand and arm but none in the face or leg. There was hypesthesia, hypalgesia and thermanesthesia but the first was the most marked. There was decided defect in "spacing sensation" and the sense of position of the fingers. Spiller says, "The case seems to indicate that the sensory center for the upper limb must be distinct from those for the face and lower limb." Mills and Weisenburg² (4) when they found the radial side of the hand more affected than the ulnar in a case of hemiplegia with hemianesthesia attributed it to the fact that the radial side had more complicated function and so a higher cerebral organization which would be more easily and permanently affected.

According to Straussler (5), Muskens (6) was the first to suggest that the sensory projection of the body surface on the cortex of the brain followed the same principle of segmentation as is seen in the spinal cord. Benedict (9) reported cases of partial hemianesthesia from cortical lesion in which the sensory loss had a metameric distribution. Russel and Horsley (10) reported five cases which apparently bear out the view that there is a re-representation in the cerebral cortex of the type of sensory representation as it exists in the spinal cord and they regard it as "demonstrated that in cases of lesion of the kinesthetic cor-

²See Transactions of American Neurological Society.

tex the spinal type of sensory representation, i. e., one parallel to the principal axis of the limb, asserts itself in all compound movements and in conscious perception of touch and points of contact in space."

Straussler (l. c.) recently recorded a case which apparently upholds the view that the cerebral cortex contains sensory areas which represent spinal segments and hence re-represent the distribution of the sensory roots of those segments. The patient was an infantry man aged 22 years who had an endocardial lesion. He had convulsive attacks which began in the right arm and were followed by right-sided hemiplegia with the characteristics of an organic paralysis. Sensation to touch, pain and temperature were diminished over the right side of the body but were lost in areas which, according to Starr's (l. c.) diagrams, corresponded approximately to the sensory distribution of the fourth cervical and second, third and fourth dorsal segments of the spinal cord, except that posteriorly the area did not extend to near the median line.

Goldstein (16) has recently taken up the subject of "Cerebral Sensory Changes of Spinal Type" and reported a case in which, following a sudden attack of unconsciousness, there was slight right hemiparesis with some aphasic symptoms and disturbed sensation on the right side of the body and right arm and leg. The most interesting points about this sensory disturbance were, that it was much more marked in certain areas which nearly corresponded to the sensory distribution of certain spinal segments, particularly was this true of the areas of skin supplied by the fourth cervical to the fourth dorsal spinal segments; on the dorsal part of the arm, the ulnar side was more affected than the radial; this last observation is the opposite of the condition described by Mills and Weisenburg and seems to show that their explanation for the differences between the two sides in their case, i. e., the more complicated function of the radial side, is inadequate.

A case was admitted to my service at the University of Michigan Hospital on March 24, 1908, in which there was evidently from the history a post-operative cortical lesion in the right parietal lobe of the cerebrum. There were epileptic attacks with a sensory aura, a left hemiparesis and sensory disturbances on the left side of the body. There was dissociation of the different

forms of sensation and the analgesia had a distribution which in part was very suggestive of that which follows a lesion of spinal nerve roots, i. e., spinal segmental.

Martin M., aged 40 years, an American railroad conductor.

Family History.—Father died of old age. Mother died of heart trouble. Six brothers and four sisters all well.

Previous Medical and Social History.—Said he never was sick a day in his life. Denied venereal diseases. Never drank to excess. Married; had two children.

History of present illness, confirmed by his wife: He was in a railroad accident August 15, 1906, and was run over by a train, his right arm cut off and the right side of his head injured. He was paralyzed on the left side following the accident and was treated at the company hospital. His memory from the time of the accident up until the next January was very vague. He said that he was unconscious but meant that he did not remember anything that occurred during that time. In January he was operated on at Wheeling and said that at that time pieces of steel were removed from his head. He improved after the operation so as to be able to be about; for three months previous to his admission to the University Hospital he had been getting worse. There was never any difficulty in speech and he read and understood all that he read. Before his operation he was unable to move the left arm or leg, but after the operation the left arm improved to the present condition and he was able to walk about unsupported. The left arm remained in the improved condition but since he has been getting worse the left leg has become more parietic and when admitted to the hospital he was unable to walk without support.

He said that he had had spells (the last one a month before the examination) in which he felt as if his left leg was going up in the air, though it did not move. Then he pulled his head to the right and lost consciousness. His wife says that he jerked in the attacks and had bitten his tongue while in them but never had any incontinence of urine. When he regained consciousness he was weak and dazed. At other times he has a feeling of weakness with the above described aura but no convulsive attack. When he moved the stump of the right arm he had associated movement in the left arm. He has also noticed that when he gaped his fingers would straighten out and would slowly return to their former position after the gape ceased.

On March 25, 1908, an examination showed that the patient was a well-nourished man with the right arm off just below the shoulder. His gait was of the hemiplegic type and there was no ataxia and no Romberg symptom. There was a small depression in the parietal bone, four inches, vertically, above the external auditory meatus; about an inch and a half long and about an inch wide. The depression was tender to pressure but there was

no tenderness of other parts of the scalp. He said that the scalp on the right side was itchy. The pupils were equal, and reacted to light and to accommodation. The tongue protruded in the median line. In showing the teeth the right corner of the mouth was drawn back farther than the left but there was no asymmetry of the face at rest. There was no hemianopsia. There was anesthesia to touch in the region of the left cheek

FIG. 1. Sensibility to Touch. Shaded area, Hypesthesia. Double shaded area, Anesthesia. Lines of demarcation not sharply defined.

and lower jaw which did not involve the left side of the nose and there was hypesthesia to pinpoint in the same region. He could raise the left arm slightly at the shoulder; could flex it weakly and could extend it with fair strength. The left hand was contractured, the thumb being flexed into the palm of the hand and the fingers flexed over the thumb. There was slight contracture

at the elbow in flexion. He could not move the fingers in any direction. The contractures could be passively overcome and there was no atrophy in the muscles. The biceps and triceps jerks were exaggerated. There was weakness in the movements of the left lower extremity especially the ankle and toes and there was considerable spasticity but no contractures and

FIG. 2. Localization of Sensation. Numbers, Points touched. Numbers prime, Points located by patient. Numbers in circles, Points correctly localized.

no atrophy. The right leg had normal power. Plantar irritation caused flexion of all the toes on the left side and also on the right side. Plantar flexion was also caused by the methods of Gordon and Oppenheim, and of Shaffer. The knee jerks were somewhat exaggerated on both sides but especially the left. Ankle clonus was present on the left side but not on the right.

The cremasteric reflex was present on both sides and the abdominal reflex was present on both sides.

April 6, 1908, he was seen by Dr. de Nancrede, surgeon to the hospital, in consultation. Operation was suggested but refused and he left the hospital. The sensory changes which were found unchanged on repeated examinations are accurately shown in the

FIG. 3. Sensibility to Pinpoint. Shaded area, Analgesia. Stippled area, Hypalgesia, with peculiar sensation.

charts. On reference to the diagrams of the sensory changes it will be seen that the defect in the perception of tactile sensation involved almost the entire left side of the body, approaching the median line but not exactly reaching it. The hypesthesia in this case followed the rule in becoming more marked toward the distal portion of the extremities until, in the upper extremity, it reached a complete anesthesia just above the wrist. He was

able to appreciate touch in the left hand if any pressure was used. The patient spoke of a difference in the feeling caused by touches on the two sides of the body, the touches on the left side being felt more as tickling sensation. Tactile discrimination was much impaired on the left side; the compass points were not recognized as double unless separated six inches or more. There was a marked impairment of localizing sensibility as tested by naming the point touched. The amount of error was not constant but was more in the extremities than on the body and points near the median line were localized correctly. The direction of the error was toward the median line of the body. He showed no hesitancy in naming the points touched.

The distribution of the analgesia was peculiar in that the unaffected area on the upper chest and extending, tongue-like, down the inside of the arm was somewhat similar to the sensory areas of the first to the fifth dorsal segments, according to the sensory distribution of spinal segments as given by Starr (l. c.) and this area was the only part of the left side that was not affected. The total analgesia of the left leg gave place to a hypalgesia in the left thigh and left side of the lower chest and abdomen. In this area of hypalgesia the sensation was peculiar, inasmuch as while insisting that he did not feel the pinprick as such is still produced an intensely disagreeable sensation and the ordinary reflexes of pain. Deep pressure on the muscle masses of the left arm and leg was more painful, he said, than similar pressure applied to the right leg.

He called both hot and cold test tubes warm on the left half of the face and left half of the body. In the part of the body noted as anesthetic to touch he felt nothing. When both the hot and cold test tubes were placed on the chest at the same time he felt only the warm; did not feel two test tubes. The distribution of lost thermic sense did not correspond to the lost pain sense but occupied the entire left half of the body, coming very close to the median line.

Vibratory sensation was felt but not plainly over the external condyle of the left humerus and head of the left radius. It was not felt at all on the external condyle of the left femur, or the left patella, or the left external or internal malleoli. It was felt normally on the anterior superior spine of the left ilium, the spine of the left scapula, and the left side of the head and face. The sense of motion and the sense of position were both lost in the left wrist and finger joints, also in the left ankle and toes. Both were preserved in the left knee and shoulder joints. In the left elbow the sense of position was lost, motion was recognized but not its direction. There was no knowledge whatever of objects placed in the left hand.

It is evident from the history that in this case there was a lesion in the cerebral cortex. That there might also be a lesion deeper

in the brain in the basal ganglia cannot be denied, but the improvement in the symptoms which followed an operation, done some time after the injury, suggests that the lesion was confined to the cortex and subcortex; also the absence of hemianopsia in a case with such marked sensory symptoms would be against the location of the lesion in the internal capsule. The most marked disturbances were noted in the appreciation of light touch, the distinction between heat and cold, the discrimination of compass points and the localization of light touch, and these disturbances were greater in the extremities than on the body though there was no sharp line of demarcation. Vibratory sensibility and the appreciation of the position and movements of the fingers and toes were lessened on the same side. Sensibility to pain was not disturbed in the face nor in the first and second cervical segments but in an area which corresponds closely to the sensory distribution of the third cervical to the eighth cervical, inclusive, sensibility to pain was entirely lost. The perception of pain to pinprick was intact in the distribution of the first to the fifth dorsal segments inclusive. Below this there was an area extending well down on the lower limb below the knee in which pinprick was not perceived as such but as a most disagreeable tingling sensation. This area gradually shaded off into an area which included the lower half of the leg and the foot and which was totally analgesic.

REFERENCES

1. Gordon. *JOURNAL OF NERVOUS AND MENTAL DISEASE*, 1903.
2. Karl Shaffer. *Neurol. Centralbl.*, 24, 1905.
3. Starr. "Organic and Functional Nervous Diseases," Philadelphia, 1907.
4. Mills and Weisenburg. *JOURNAL OF NERVOUS AND MENTAL DISEASE*, October, 1906.
5. Straussler. *Monatsschrift für Psych. und Neurologie*, Mai, 1908.
6. Muskens. *Arch. f. Psych.*, Bd. 36.
7. Benedict. *Neurol. Centralbl.*, 24, 1905.
8. Max Löwy. *Prager med. Wochschr.*, Bd. 30, Nr. 2-3, 1905.
9. Benedict. *Wiener klin. Wochenschr.*, Nr. 7, 1907.
10. Russel and Horsley. *Brain*, April, 1906.
11. Morton Prince. *Transactions of the American Neurological Association*, 1907.
12. George Sandberg. *Deutsche Zeitschrift für Nervenheilkunde*, Bd. 30, H. 3-4.
13. Lewandowsky. *Deutsche med. Wochenschrift*, 1907, No. 21.
14. William G. Spiller. *JOURNAL OF NERVOUS AND MENTAL DISEASE*, February, 1906, also in *Contributions from the Department of Neurology and Laboratory of Neuropathology of the University of Pennsylvania*. Vol. II, 1906.
15. Klippel, Sergueeff and Weil. *Revue Neurologique*, July 15, 1908, page 694.
16. Kurt Goldstein. *Neurol. Centralbl.*, Feb. 1, 1909.