this is the distance between the nucleus of the third nerve and the collection of motor cells in the lower part of the lumbar enlargement. For this reason lesions of this segment are more apt to cause paralysis of individual muscles or muscle groups, as distinguished from the more wide-spread paralysis due to lesions of the upper segment.

Reference to Starr's table will show that the muscles are represented in the spinal cord without relation to the nerves which supply them—that is to say, muscles that are supplied by a certain nerve may not be represented close together in the anterior horns; for instance, in the fourth cervical segment, movements of the diaphragm, deltoid, biceps, supinator longus, rhomboid, supraspinatus, and infraspinatus are represented. It follows from this that the distribution of a paralysis due to disease of the lower motor segment may enable us to distinguish the position of the lesion within the segment itself. We are often helped in this by the sensory symptoms, which may accompany the paralysis. Thus, if we have a paralysis with the characteristics of a lesion of the lower motor segment, and if the paralyzed muscles are all supplied by one nerve, and we discover anæsthesia in the skin of the arm supplied by that nerve, it is evident that the lesions must be in the nerve itself. On the other hand, if the muscles paralyzed are not supplied by a single nerve, but are represented close together in the spinal cord, and the anæsthesia corresponds to that section of the cord (see table), it is equally clear that the lesion must be of the cord itself or of its nerve-roots.

Irritative Lesions of the Lower Motor Segment.—We know of no lesion of this segment which has as its result abnormal muscular contraction unless the slow atrophy of the ganglion cells occurring in progressive muscular atrophy be considered as the cause of the fibrillary contraction so common in this affection.

(Certain tonic muscular contractions occurring in poisoning by strychnine and in tetanus are thought to be due to the perverted action of the lower motor centres, and Hughlings Jackson believes that certain convulsive paroxysms—"lowest level fits"—are due to discharging lesions of these centres, and claims laryngismus stridulus in this category.)

(*b*) **Lesions of the Upper, Cerebro-spinal Motor Segment.**—*Destructive lesions* cause, as in the lower motor segment, paralysis, and here again the secondary degeneration which follows the lesion gives to the paralysis its distinctive characteristics. In this case the paralysis is accompanied by a spastic condition, shown in an exaggeration of muscle reflex and an increase in the tension of the muscle. It is not accurately known how the degeneration of the pyramidal fibres causes this excess of the muscle reflex. The usual explanation is that under normal circumstances the upper motor centres are constantly exerting a restraining influence upon the activity of the lower centres, and that when the influence ceases to act, on account of disease of the pyramidal fibres, the latter take on increased activity, which is made manifest by an exaggeration of the muscle reflex.

It was stated above that each segment of the motor path is to be considered as a nutritional unit and that the secondary degeneration in the upper segment stops at the beginning of the lower. So the muscles paralyzed by lesions in the upper segment do not undergo degenerative atrophy, nor do they present the reaction of degeneration.

The upper motor segment is much more compact than the lower, and for this reason a paralysis resulting from a lesion in it is apt to involve many muscles. This is especially true in regard to the pyramidal fibres, which run in a compact bundle, a lesion of which usually involves all of the fibres and causes a paralysis of all of the muscles of one side of the body—i. e., *hemiplegia.*

The motor centres of the cortex are more or less separated from each other, and a sharply localized lesion in this region causes a more limited paralysis, and cerebral monoplegias are the result; but even in this case the paralysis is diffuse, affecting the whole limb or a segment of the limb, and not individual muscles or groups of muscles.

To sum up, the paralyses due to lesions of the cerebro-spinal motor segment are diffuse, wide-spread, often hemiplegic; the paralyzed muscles are spastic (the tendon reflexes exaggerated), they do not undergo degenerative atrophy, and they do not present the degenerative reaction to electrical stimulation.

Irritative Lesions of the Upper Motor Segment.—Our knowledge of such lesions is confined for the most part to those acting on the cortical motor centres, and we know a number of processes which have as their result abnormal muscular contractions. These have as their type the localized convulsive seizures classed under Jacksonian or cortical epilepsy, which are characterized by the convulsion beginning in a single muscle or group of muscles and involving other muscles in a definite order, depending upon the position of their representation in the cortex; for instance, such a convulsion beginning in the muscles of the face next involves those of the arm and hand, and then the leg. The convulsion is usually accompanied by sensory phenomena and followed by a weakness of the muscles involved.

A majority of lesions of the motor cortex are both destructive and irritative—i. e., they may destroy the nerve-cells of a certain centre, and either by their growth or presence may throw into abnormal activity those of the surrounding centres.

So far the motor system has been considered by itself, and we have endeavored to show how attention to the paralysis alone may help us to determine the seat of a lesion. It runs, however, in close connection with other systems of the nervous centre, which are often involved with it in morbid processes, giving rise to symptoms which aid us very much in making a local diagnosis.

Sensory Centres and Paths.—The association of the motor path with that for the conduction of sensory impressions is very intimate, but unfortunately our knowledge of the exact position of the sensory tracts is by

no means so precise. Some important facts are, however, known. Sensory fibres from different areas of the skin run in close connection with fibres of the lower motor segment in the mixed nerves. They separate from them and enter the spinal cord by the posterior roots. The regions which the different posterior roots supply is given in Starr's table. After entering the spinal cord the sensory fibres cross the middle line at once and pass up to the brain in the opposite half of the cord. Here they are again in close contact with the motor path, but with that of the other side of the body—i. e., the right half of the spinal cord contains the sensory fibres of the left side of the body and motor fibres of the right. The fibres which conduct the impressions for the muscular sense seem to be an exception and do not decussate in the cord. The exact position of the sensory paths in the cord is still somewhat uncertain, nor are we sure of their course in the medulla, pons, and peduncle. All the sensory fibres of the opposite side of the body are collected in the posterior third of the posterior limb of the internal capsule, just behind the motor fibres of the upper segment.

Much doubt and discussion still exist as to the areas for the representation of sensory impressions. Horsley has suggested that the muscular and tactile senses are localized in the motor cortex, and that two of the three chief layers of cells in this region subserve their functions. Dana's study shows that many lesions of the motor area, particularly in the hinder part, are associated with anæsthesia. On the other hand, Ferrier regards the hippocampal convolution, and Schäfer the gyrus formicatus, as the centres for sensory impressions.

The centres for sight, hearing, smell, and taste have been referred to under the nerves ministering to these senses, and we shall consider the speech centres in the next section.

In the centrum ovale the fibres of the motor path are more or less closely associated with other systems of fibres; those connecting the cortex with nervous structures lying below it, projection fibres; the fibres which join the two hemispheres, commissural fibres; and those which join different parts of the same hemisphere, association fibres. Our knowledge of the function of these fibres leaves much to be desired.*

The following is a brief summary of the effects of lesions from the cortex to the spinal cord:

1. **The Cerebral Cortex.**—(*a*) Destructive lesions cause *spastic paralysis* in the muscles of the opposite side of the body. The extent of the paralysis depends upon that of the lesion. It is apt to be limited to the muscles of an extremity, giving rise to the cerebral monoplegias (Fig. 3, *X*). A lesion may involve two centres lying close together, thus producing paralysis of the face and arm, or of the arm and leg, but not of

* The student will find in Starr's work, Familiar Forms of Nervous Disease, an admirable presentation of this subject.

the face and leg without involvement of the arm. Very rarely the whole motor cortex is involved, causing paralysis of one side—cortical hemiplegia.

Combined with the muscular weakness there is usually some disturbance of sensation, particularly tactile impressions and those of the muscular sense.

(*b*) Irritative lesions cause localized spasms as described above. These convulsions are usually preceded and accompanied by sensory impressions. Tingling or pain, or a sense of motion in the part, is often the *signal symptom* (Seguin), and is of great importance in determining the seat of the lesion.

Lesions are often both destructive and irritative, and we have combinations of the symptoms produced by each. For instance, certain muscles may be paralyzed, and those represented near them in the cortex may be the seat of localized convulsions, or the paralyzed limb itself may be at times subject to convulsive spasms, or muscles which have been convulsed may become paralyzed. In this manner it is often possible to trace the progress of a lesion involving the motor cortex.

We have seen in a previous section that lesions involving the centres for the special senses may give rise to focal symptoms, and shall simply refer to them here. The symptoms caused by lesions of the speech centre will be described under aphasia, and it is only necessary to note the near situation of the motor speech area (Broca's centre) in the left third frontal convolution to the centres of the face and arm on that side, and to state that motor aphasia is often associated with monoplegia of the right side of the face and the right arm. Accompanying the paralysis following a Jacksonian fit of the right face or arm there is often a transient motor aphasia.

(2) **Centrum Ovale.**—Lesions in this part of the motor path cause paralysis, which has the distribution of a cortical palsy when the lesion is near the cortex, and of that due to a lesion of the internal capsule when it is near that region. They may be associated with symptoms due to the interruption of the other system of fibres running in the centrum ovale, and there may be sensory disturbances—hemianæsthesia and hemianopia—and if the lesion is in the left hemisphere one of the different forms of aphasia may accompany the paralysis.

(3) **Internal Capsule.**—Here all the fibres of the upper motor segment are gathered together in a compact bundle, and a lesion in this region is apt to cause complete hemiplegia of the opposite side, and if the lesion involves the hinder third of the posterior limb there is also hemianæsthesia, including even the special senses (Fig. 3, *Y*).

(4) **Crus Cerebri.**—Here, again, all the motor fibres and all the sensory fibres of the opposite side are collected in a small space, and a lesion may produce hemiplegia combined with sensory disturbances. On account of its anatomical relation the third cranial nerve is often involved

in lesions of the crus, causing paralysis of the muscles of the eye on the same side as the lesion combined with a hemiplegia of the opposite side—i. e., a crossed paralysis.

(5) **Pons.**—In the pons, medulla, and cord the upper and lower motor segments are both represented, the first by the pyramidal fibres, the latter by the motor nuclei and the nerve-fibres arising from them. Lesions here often affect both motor segments, and produce combinations of paralyses having the characteristics of each. Thus a lesion in the lower part of the pons may involve the pyramidal tract and cause a spastic paralysis of the opposite arm and leg, and also involve the nucleus or the fibres of the facial nerve, and so produce a paralysis of the same side of the face, accompanied by loss of the muscle reflex, atrophy, and the reaction of degeneration—crossed paralysis (Fig. 3, *Z*). The abducens and hypoglossus nerves may also be paralyzed in the same manner. In lesions of the pons the patient often has a tendency to fall toward the side on which the lesion is, due probably to implication of the middle peduncle of the cerebellum.

The symptoms produced by involvement of the different cranial nerves have been considered in detail in a previous section.

(6) **Spinal Cord.**—Unilateral lesions cause, first, a lower-segment paralysis, due to the disease of the centres at the site involved; second, a spastic paralysis of all the muscles on that side of the body below the lesion, due to interruption of the pyramidal fibres; and, third, disturbance of sensation in the opposite side of the body. (See under Brown-Séquard's paralysis.)

Transverse lesions of the cord cause paralysis with atrophy, etc., at the level of the lesion, spastic paralysis below it, combined with sensory disturbance and trouble with the bladder and rectum.

Affections of the peripheral nerves have already been considered.

II. Aphasia.

The speech mechanism consists of receptive, perceptive, and emissive centres in the cortex cerebri, disturbances of which cause *aphasia*, and centres in the medulla which preside over the muscles of articulation, disturbance of which produces *anarthria*, the condition of gradual loss of power of speech, such as occurs in bulbar paralysis.

The studies of Bastian, Küssmaul, Wernicke, Lichtheim, and others have widened enormously our knowledge of speech disorders. Language is gradually acquired by imitation. Thus, in teaching a child to say *bell*, the sound of the uttered word enters the afferent path (auditory nerve) and reaches the auditory perceptive centre, from which an impulse is sent to the emissive or motor centre presiding over the nuclei in the medulla, through which the muscles of articulation are set in action. The arc in Lichtheim's schema (Fig. 5) is a A, Mm. The child gradually acquires in this way *word memories*, which are stored at the centre A, and *motor memories*—the memories of the co-ordinated muscular movements necessary to utter words—which are stored at the centre M. In a similar manner, when shown the bell, the child acquires *visual memories*, which are conveyed through the optic nerve to the visual perceptive centres, o O. So also the memories of the sound of the bell when struck. The memory picture of the shape of the bell, the memory of the appearance of the word bell as written, and the motor memories of the muscular movements required to write the word are distinct from each other; yet they are intimately connected, and form together what is termed the *word-image*. In addition to all this the child gradually acquires in his education ideas as to the use of the bell—intellectual concepts—the centre for which is represented at I in the diagram In volitional or intellectual speech, as in uttering the word *bell*, the path would be I, M m, and in writing the word, I, M, W, h. These various "memories" are as a rule stored or centred in the left hemisphere.

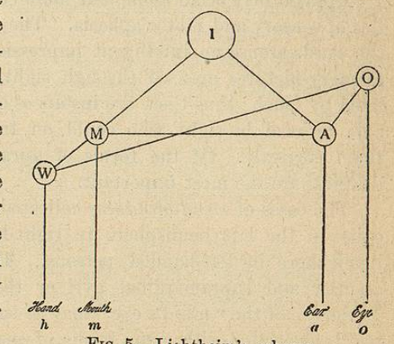

Fig. 5.—Lichtheim's schema.

The relations of written and spoken language are then with (*a*) sensory perceptive centres (hearing and sight and, in the blind, touch); (*b*) emissive or motor centres for speech and writing; and (*c*) psychical centres, through which we obtain an intellectual conception of what is said or written, and by which we express voluntarily our ideas in language.

There are two chief forms of aphasia—*sensory* and *motor*.

(1) **Sensory Aphasia; Apraxia; Word-blindness; Word-deafness.**—By apraxia is understood a condition in which there is loss or impairment of the power to recognize the nature and characteristics of objects. Persons so affected act "as if they no longer possessed such object memories, for they fail to recognize things formerly familiar. A fork, a cane, a pin, may be taken up and looked at by such a person, and yet held or used in a manner which clearly shows that it awakens no idea of its use. And this symptom, for which at first the term blindness of mind was used, is found to extend to other senses than that of sight. Thus the tick of a watch, the sound of a bell, a melody of music, may fail to arouse the idea which it formerly awakened, and the patient has then deafness of mind, or an odor or taste no longer calls up the notion of the thing smelled or tasted; and thus it is found that each or all of the sensory organs, when

called into play, may fail to arouse an intelligent perception of the object exciting them. For the general symptoms of inability to recognize the use or import of an object the term *apraxia* is now employed." (Starr.)

Apraxia may occur alone, but more commonly is associated with varieties of sensory and motor aphasia. The patient may be able to read, but the words arouse no intelligent impression in his mind. While blind to memory-pictures aroused through sight, the perceptions may be stimulated by touch; thus there are instances on record of apraxic patients unable to read by sight, who could on tracing the letters by touch name them correctly. Of the forms of apraxia, mind-blindness and mind-deafness are the most important.

The cases of *mind-blindness* collected by Starr indicate that the lesion exists in the left hemisphere in right-handed persons, and in the right hemisphere in left-handed persons. The disease usually involves the angular and supramarginal gyri or the tracts proceeding from them. Blindness of the "mind's eye" may at times be functional and transitory, and is associated with many forms of mental disturbance. In a remarkable case reported by Macewen, the patient, after an injury to the head, had suffered with headache and melancholia, but there was no paralysis. He was psychically blind and though he could see everything perfectly well and could read letters, objects conveyed no intelligent impression. A man before his eyes was recognized as some object, but not as a man until the sounds of the voice led to the recognition through the auditory centres. The skull was trephined over the angular gyrus and the inner table was found to be depressed and a portion had been driven into the brain in this region. The patient recovered. Mind-blindness is the equivalent of visual amnesia.

Word-blindness may occur alone or with motor aphasia. In uncomplicated cases the patient is no longer able to recall the appearances of words, and does not recognize them on a printed or written page. The patient may be able to pronounce the letters and can often write correctly, but he cannot read understandingly what he has written. It is rare, however, for the patient to be able to write with any degree of facility. There are instances in which the patient, unable to read, has yet been able to do mathematical problems and to recognize play cards. The lesion in cases of word-blindness is, in a majority of cases, in the angular and supramarginal gyri on the left side. It is commonly associated with hemianopia, and not infrequently with mind-blindness.

Mind-deafness is a condition in which sounds, though heard and perceived as such, awaken no intelligent conceptions. A person who knows nothing of French has mind-deafness so far as the French language is concerned, and though he recognizes the words as words when spoken, and can repeat them, they awaken no auditory memories. The musical

faculties may be lost in aphasics, who may become note-deaf and unable to appreciate melodies or to read music. This may occur without the existence of motor aphasia, and on the other hand there are cases on record in which with motor aphasia for ordinary speech the patient could sing and follow tunes correctly. Mind-deafness is also known as auditory amnesia. Word-deafness is a condition in which the patient no longer understands spoken language. The memory of the sound of the word is lost, and can neither be recalled nor recognized when heard. It is usually associated with other varieties of aphasia, though there are cases in which the patient has been able to read and write and speak. The lesion in word-deafness has been accurately defined in a number of cases to be in the posterior portion of the first and second temporal convolutions on the left side (Fig. 2).

Other manifestations of mind-blindness are met with; thus a young man with secondary syphilis had several convulsive seizures, after one of which he remained unconscious for some time. On awakening, the memory-pictures of faces and places were a blank, and he neither knew his parents nor brothers, nor the streets of the town in which he lived. He had no aphasia proper, and no paralysis.

(2) **Motor or ataxic aphasia** is a condition in which the memory of the efforts necessary to pronounce words is lost, owing to disturbance in the emissive centres. This is the variety long ago recognized by Broca, the lesion of which was localized by him in the third left frontal convolution. In pure cases the patient is able to read (not aloud) and understands perfectly what is said. He may not be able to utter a single word; more commonly he can say one or two words, such as "no," "yes," and he not infrequently is able to repeat words. When shown an object, though not able to name it, he may evidently recognize what it is. If told the name, he may be able to repeat it. A man knowing the French and German languages may lose the power of expressing his thoughts in them, while retaining his mother-tongue; or, if completely aphasic, may recover one before the other. As the third left frontal convolution is in close contact with the centres for the face and arm, these are not uncommonly involved, with the production of a partial or, in some instances, a complete right-sided hemiplegia. *Alexia*, or inability to read, occurs with motor aphasia and also with word-blindness.

As a rule, in motor aphasia there is also inability to write—*agraphia*. When there is right brachial monoplegia it is difficult to test the capability, but there are instances of motor aphasia without paralysis, in which the power of voluntary writing is lost. The condition varies very much; thus a patient may not be able to write voluntarily or from dictation, and yet may copy perfectly. It is still a question whether there is a special writing centre. It has been placed by some writers at the base of the second frontal convolution, but in a