case the three branches have an arrangement the reverse of the usual one.

Variations in the number and position of the branches of the arch are frequent. There may be only one trunk, or there may be from one to six inclusive.

Descending Thoracic Aorta.—At the termination of the arch, at the lower border of the fourth dorsal vertebra, the descending aorta begins and continues down along the spine to the fourth lumbar vertebra, where it divides into the two common iliac arteries. Its direction is not vertical, for as it rests against the spine it necessarily follows the spinal curves, being concave forward in the dorsal region and convex forward in the lumbar. As its commencement is to the left of the spine and its termination nearly in the median line, its general direction throughout its whole length is inward, this being more marked in its upper part. The lower limit of the thoracic aorta is the *hiatus aorticus* at the level of the diaphragm. This part of the aorta is from seven to eight inches long and is contained in the back part of the posterior mediastinum, where it rests against the spine. Its branches are small, and consequently its size is little diminished (from 23 to 21 mm.).

Branches.—The branches of the descending thoracic aorta, though numerous, are small. They are the pericardial, bronchial, œsophageal, posterior mediastinal, and intercostal.

Varieties.—Now and then an obliteration of the aorta at the point of junction of the arch and thoracic aorta is observed, just below the connection between the ductus arteriosus and the arch, known as coarctation of the thoracic aorta. This condition results in the establishment of the interesting collateral circulation which takes place. Not infrequently variations in the number and position of the branches of this section of the aorta are observed.

Abdominal Aorta.—This name is given to the vessel between the diaphragm and its bifurcation into the two common iliac arteries. In relation to the spinal column it begins about the lower margin of the last dorsal vertebra and ends at a point about the middle of the fourth lumbar vertebra, most generally slightly to the left, sometimes almost exactly in the median line, at other times slightly to the right. This point almost corresponds to the level of a line drawn between the two iliac crests or to a point just below and to the left of the umbilicus. In length it is about five inches. As its branches are both numerous and large, its size rapidly diminishes. As mentioned before, its curve as it rests against the vertebræ has its convexity forward, being most prominent at the third lumbar vertebra, slightly above and to the left of the umbilicus.

Relations.—Anterior to the abdominal aorta are the lesser omentum and stomach, the solar plexus, splenic vein, pancreas, left renal vein, transverse duodenum, mesentery, aortic plexus, peritoneum, lymphatic vessels and glands, and dense areolar tissue; posterior to it are the bodies of the vertebræ and the left lumbar veins, the thoracic duct, and the receptaculum chyli. On the right are the inferior vena cava, right crus of the diaphragm, vena azygos major, thoracic duct, and right semilunar ganglion. On the left are the sympathetic nerve and the left semilunar ganglion.

Branches.—These may be classified under two heads: (1) Visceral—cœliac axis (gastric, hepatic, splenic), superior mesenteric, inferior mesenteric, suprarenal, renal, and spermatic or ovarian. (2) Parietal—phrenic, lumbar, and sacra media. The branches of the aorta mostly pass off at right angles.

Varieties.—Instances are known in which the aorta is divided by a septum for either a part or the whole of its course, so that two closely united tubes are the result. Sometimes this condition has a pathological foundation, at other times it is due to an embryological defect in the fusion of the double fetal aorta. The vessel has been known, as in certain quadrupeds, to divide up into an ascending and a descending branch, the former subdividing into three trunks to supply the head and upper extremities. The aorta may vary in position and extent. Its lower limit may vary to the depth of a lumbar vertebra, so that its bifurcation may take place at the third, or even higher, at the fifth. Its deviation from its normal position with reference to the vertebral column is generally due to pathological changes rather than to congenital causes. Quain speaks of two cases of a large pulmonary branch springing from the aorta very near the cœliac axis, which, after having passed upward through the œsophageal foramen in the diaphragm, separated into two branches and entered the lungs near their bases. Balfour, in writing of the simulation of aneurism by malposition of the aorta due to rickets, says: "In rickety chests the aorta may be so deflected, without any marked dilatation, as to make its pulsation visible either to the right or left of the sternum, and so to simulate an aneurism. It is of even greater consequence to have proof that in certain comparatively rare cases a similar abnormal pulsation may be due to a trifling divergence from the normal course of the vessel itself, apart from any marked change in the bony skeleton. But we must never forget that aortic aneurism may coexist with malformation of the thorax with or without scoliosis, and whatever may be the condition of the skeleton, any abnormal pulsation must be carefully considered from every point of view before we are able to give any definite opinion as to what it really is." Virchow has pointed out the relation of the reduction in size of the aorta to chlorosis, and he named the condition *aorta chlorotica*. Congenital stenosis of the aorta is seldom seen. Rosenbach has noted this condition found together with hypertrophy of the heart. It may cause sudden death, and when it is present, otherwise unimportant affections may assume a grave aspect, from sudden untoward cardiac symptoms. In congenital stenosis of the aortic system, a striking characteristic is the continuous subnormal temperature present in infectious diseases which normally show a high temperature. In women this condition is generally associated with infantile uterus and other signs of arrested development.

Structure.—The aorta is very strong and elastic and is enclosed, like most other arteries, in a sheath, which has more connective than yellow elastic tissue, so that, when cut, the vessel shrinks within the sheath. It is composed of three coats—(1) tunica intima; (2) tunica media; (3) tunica adventitia. The internal coat, smooth and offering but little, if any, resistance to the blood, consists of three layers: (*a*) Epithelial layer or arterial endothelium. This is made up chiefly of irregular, flat, polygonal cells with round or oval nuclei with nucleoli. (*b*) Subepithelial layer, which is well marked and consists of numerous anastomosing cells resting in a delicately fibrillated groundwork of connective tissue. There are, besides, elastic fibres which are in connection with the next layer. (*c*) Elastic layer, which forms the principal part of this inner coat. Sometimes this network assumes characteristics which have caused it to be designated as the "perforated" or "fenestrated" membrane of Henle. At times it is represented by a longitudinal network of fibres. The middle coat is muscular, consisting of bundles of plain muscle fibres, which are disposed circularly around the vessel, although not forming a complete ring. These fibres contract and relax, thus changing the calibre of the vessel. Elastic fibres are also found well developed in this tunic, and there is also considerable connective tissue. This coat is thicker than the corresponding coat in other arteries. It has also relatively more elastic tissue and less muscular tissue than is found in other arteries. The external coat consists of white connective tissue and elastic fibres. The connective-tissue bundles run chiefly diagonally around the vessel and connect it with its sheath. This is the strongest and densest coat.

Vessels and Nerves.—Both small arteries and veins ramify in the external coat of the aorta and are called *vasa vasorum*. They serve as nutrient vessels. Ranvier states that in health in the human subject they never penetrate to the middle coat. The inner coat is thought to be nourished by the blood circulating through it. Although the aorta is supplied by nerves, it is insensible when in a healthy condition. These nerves are chiefly non-medullated. The finer branches are distributed chiefly to the

muscular tissue of the middle coat. The aorta is supplied by both vaso-constrictor and vaso-dilator fibres. Nerve plexuses are formed around the aorta.

Physiology.—Gibson says that it has been discovered that the effect of an elastic tube on an intermittent flow of fluid is to convert it practically into a continuous stream. The elastic tube, also, by means of its distensibility, allows only part of the fluid to be driven on, and so lessens the propelling force. Hamel, in certain experiments on a frog's muscle, proved that a continuous stream of fluid causes œdema, while an intermittent stream does not have this effect. The pressure of the blood is controlled by the general laws of hydrostatics, excepting the influences resulting from cardiac energy, arterial tone, and peripheral resistance. The average blood pressure in the aorta is commonly taken as about 150 mm.

Velocity of the Blood.—In considering the blood in the arteries, two facts must be noted. During systole, the pressure is raised and is sent onward as wave motion. Besides this, there is the propulsion of the blood sent by the heart out into the organism. Arterial pulsation beginning in the aorta is "a wave of increased pressure, travelling from the centre to the periphery of the arterial system." On account of this constant variation of pressure the blood flows continually from the higher to the lower level of pressure. The three factors on which arterial pulsation depends are quantity of blood, cardiac energy, and the resistance offered by the blood-vessels. The force of the heart beat has two results—potential energy stored up in the elastic arterial walls, and kinetic energy, which forces the blood onward. During diastole there is a tendency to negative pressure in the large arteries leaving the heart, causing a "backward wave of pressure and to some extent a backward flow of blood."

Pathology.—Of all the arteries, the aorta, and particularly its arch, is most subject to pathological changes. First, as to aneurism. McLachlan enumerates many reasons why this affection should be more common in the thoracic aorta than in any other part of the vessel. "(1) It is much curved; (2) it gives off large branches; (3) the first part of the arch has no sheath, and the rest of the thoracic aorta is but feebly supported; (4) the jet of blood driven against its upper wall at each systole is apt to bulge the coats at that point; (5) sudden variations in pressure, according to the state of the heart; (6) the aspiration of the thorax affects it, for as the chest enlarges the vessel tends to expand." When aneurism is found in the sinuses of the ascending aorta, it is most generally seen in the right anterior, since this sinus, from its position, is most exposed to the impulse of the blood currents. When above the sinuses, the aneurism is generally in the anterior wall of the vessel, owing probably to the blood being pumped chiefly against this part. The aneurismal sac generally inclines toward the right and most frequently bursts into the pericardium, causing sudden death. Any of the structures in contact with, and pressed upon by, the aneurism may be affected, and the resulting discomforts are of various kinds. The heart may be displaced or its movements may be impeded. There may be palpitation or abnormal pulsation. Œdema occurs from pressure on the veins. Pressure on the œsophagus may cause dysphagia. Pressure on the trachea may produce sensations of choking, shortness of breath, or noisy respiration or brassy cough. Pressure on the different nerves may cause pain, anæsthesia, paralysis, alteration of voice, aphonia, hiccough, retching, nausea, inflammation of the lungs. The pupils may be either dilated or contracted. There may be pallor and coldness of the same side of the face and head from vaso-motor stimulation, and later redness and increased heat of the same parts from vaso-motor paralysis. Bone may be eroded, causing agonizing pain. Fatal hemorrhage usually results from the bursting of the aneurism. Death may occur from inanition in consequence of pressure on the thoracic duct. Sometimes the innominate, subclavian, or left carotid may be so blocked by clots as to cause a partial or complete disappearance of the pulse on that side. The thoracic aneurism generally inclines backward along the left side of the spine, and curvature of the spine may result from its effects on the bone. Osler speaks of a curious phenomenon which he has noticed in two cases of intrathoracic aneurism—the clubbing of the fingers and the incurving of the nails of one hand. In neither case was there any special distention or signs of venous engorgement. Aneurisms of the abdominal aorta are about equally divided between the anterior and posterior walls of the vessel, the most common situation being near the cœliac axis. The symptoms vary accordingly. In lower animals this vessel has been successfully tied, but, although the experiment has been tried on man, recovery has never followed. Sansom, in writing of the diseases of the aorta, speaks of the possibility of abscesses. The vessel walls may be invaded by pyogenic micro-organisms which give rise to abscesses. Aortitis may be acute or chronic. Acute aortitis, according to Gibson, may be of two kinds—(*a*) an accompaniment of acute disease; (*b*) primary, *i.e.*, having no apparent connection with acute disease. The former may originate in rheumatism, syphilis, scarlet fever, smallpox, measles, influenza, and tuberculous conditions. It has been associated with pericarditis, pneumonia, and pleurisy. It has followed renal disease, overfatigue, and traumatism, and it has complicated pregnancy and parturition. In short, it is caused by infections and intoxications. Chronic aortitis (atheroma or endarteritis deformans of Virchow) is generally a part of the process of arteriosclerosis, and its etiology is essentially the same, alcohol, syphilis, and overwork being responsible in most cases. Atheroma may be nodular (localized) or diffuse. Tuberculosis of the aorta has been reported. Hyaline degeneration may attack the aorta and, according to Osler, is commonly an initial stage of arteriosclerosis. Lardaceous (amyloid, waxy, or albuminoid) degeneration prepares the way for atrophy and fatty degeneration of these tissues. Fatty degeneration is common. Calcification of the aorta generally occurs in cases in which the nutrition of the vessel wall has been impaired and commonly follows fatty change and atheroma. Ossification of the arterial wall may take place. Wounds of the aorta at the base of the heart or in the descending portion are generally quickly fatal and not within the scope of surgical treatment.

Emma E. Walker.

APHASIA.—Aphasia is a term used to indicate any disturbance or perversion of intellectual expression. It includes all defects or disorders of intellectual expression, whether such disorders be the result of disarrangement or destruction of the receptive or of the emissive components of the speech mechanism, or of anything which may be employed as the substitute or equivalent of speech. Thus a person who, despite the integrity of the peripheral speech mechanism, is unable to utter his own name or to give expression to thoughts arising in the usual way, has aphasia. If he is incapable of communicating thoughts by the employment of some equivalent of spoken words, such as writing of any sort or by expressive mimicry or pantomime, he likewise has aphasia. Moreover, a person has aphasia who, with the extracerebral apparatus intact, is unable to understand the language in the use of which he has been trained; who does not even recognize, although he hears, the sounds of the most familiar nature and words to which he has for a lifetime been accustomed, such as his own name; and who—although he may be able to read—is unable to write voluntarily or from dictation, or to express his thoughts by words, by symbols, or by pantomime.

Yet even these shortcomings do not comprise all that is meant by aphasia. If a person with normal ocular apparatus looks upon a printed or written page, and the symbols there with which he has previously been entirely familiar, convey no meaning to him in the shape of approximate thought or idea, such person has aphasia, even though he may understand all that is communicated orally to him, and though he may himself be able to express his thoughts (incompletely and defectively, however) by spoken and written words.

Thus it will be seen that aphasia may be the result of conditions by which the patient is unable to part with the expressive equivalent of an idea which has been properly formed. The failure is not confined to words, but includes all modes of expression. Or it may be caused by any conditions that interfere with the reception of impulses or stimuli that enter into the genesis of ideas used in the construction of internal or external language. As movement in some form is requisite for the manifestation of all expressions, defect of this is the condition to which the term motor aphasia or aphasia of emission is applied.

In the second form of aphasia the sufferer is unable to adapt receptive communications and make them fit the idea represented by the verbal symbol, auditory or visual;—that is, he has lost the faculty of adapting the complement of the word to his own idea; it matters not whether these words be spoken or written, or communicated by some equivalent, such as music and pantomime. In a general way, this is aphasia of reception, or sensory aphasia.

Motor aphasia, or aphasia of emission, which was described by Broca as aphemia, and by many writers after him as ataxic aphasia, may be divided into as many forms as there are habitual avenues of externalizing thoughts. Ideas are usually exteriorized by spoken words, by written words, by symbols, and by pantomime. Thus we have aphasia of articulation, logaphasia; aphasia of writing, agraphia or logagraphia; asymbolia, and asemia. Aphasia of reception, or sensory aphasia, is also made up of a number of constituents, the two great divisions being auditory aphasia, or word deafness, and visual aphasia, or word blindness. Each form of aphasia admits in turn of further subdivision.

Aphasia may be classified as follows:

1. *True Aphasia.*—Aphasia of apperception. Due to lesion of any constituent of the speech region, the zone of language. It might be subdivided into (*a*) visual aphasia, due to lesion of the visual areas and centres; (*b*) auditory aphasia, due to lesion of the auditory areas and centre; (*c*) articulatory kinæsthetic aphasia, due to lesion of the centre in which are stored memories of the movements necessary to externalize the word by speech.

2. *Sensory Aphasia.*—Due to lesion of the central and peripheral sensory pathways leading to the zone of language.

3. *Motor Aphasia.*—Due to lesion of the motor pathways, over which the motor impulses travel in passing to the peripheral speech musculature.

4. *Compound Aphasia.*—Any combination of two or more of these.

5. *Associative or Transcortical Aphasia.*

The lesion may be:

(1) In the habitual pathway traversed by impulses going from the auditory to the visual area (the patient can hear a name, but cannot write it from hearing; cannot write it from dictation; he has paragraphia).

(2) It may be in the habitual pathway of impulses going from the visual area to the auditory area (the patient can see an object, but he cannot call up its name, because this requires the mediation of the auditory area).

(3) A lesion that interrupts the habitual pathway that impulses take when going from the auditory area to the seat of phonetic memories in Broca's convolution (the patient can hear, can interpret from hearing, but cannot talk correctly; paraphasia).

(4) The lesion may interrupt the pathway taken by impulses going from the visual area to the auditory area (the patient is dyslexic, paragraphic, and slightly paraphasic).

For purposes of convenience I shall adopt the following classification as a working one, and I desire to say at the outset that the word motor aphasia is never used synonymously in my own mind with the word ataxic aphasia; moreover, that the word motor is reserved for application to that form of aphasia which is anatomically characterized by lesion of Broca's convolution, solely because such usage has been consecrated by time. The images stored up in this centre are genetically sensory, the result of motion.

Motor aphasia.		1. Lesion of the kinæsthetic-articulatory centre, Broca's area (and probably of the associative fibres connecting them with the cortical motor areas in the Rolandic region), causing articulatory amnesia.
		2. Lesion of motor fibres which convey speech impulses: subcortical motor aphasia. Pure motor aphasia of Dejerine.
		3. Lesion of any part of the peripheral neuromuscular apparatus serving articulate expression: dysphasia, lallation, alalilia.
Sensory Aphasia.	Auditory.	1. Lesion of the receptive cells constituting the auditory centre causing abolition of function, word deafness and its entailment.
		2. Lesion of the subcortical sensory tract: subcortical auditory aphasia; pure auditory aphasia of Dejerine.
	Visual.	1. Lesion of the receptive cells of the higher visual centre causing word amnesia, graphic and visual word blindness, and its entailment.
		2. Lesion of the subcortical tract and the primary visual centre which entails loss of the recognition of things, words, and objects: that is, loss of their significance, object amnesia or apraxia, or true neural blindness.
Total or Compound Aphasia.		Lesion of the entire zone of language: disturbed function of visional, auditory, kinæsthetic-articulatory centres.

History.—The real history of aphasia dates from 1861. In that year Broca presented an epoch-making communication to the Paris Anatomical Society, which seemed to prove that the morbid anatomy of aphasia was a lesion of the posterior part of the third convolution.

Broca's communication opened a new era in the understanding of speech. It was not, however, more epochal than that which marks the direction of Wernicke's master mind toward a solution of the problems of aphasia in the early years of the seventh decade of this century.

Speech disturbances had been recognized clinically and studied even so far back as the time of ancient Grecian writers, who used the term anandia to signify loss of speech; but the first records of serious attempts to study the faculty of speech from the standpoint of its pathology were made by Bouillaud in France in 1825, by Jackson in this country in 1829, by Dax in 1836, and by Lordat in 1843. Many years before, the brilliant, misguided Gall had located the faculty of speech in the supra-orbital convolutions. Early in the century Lordat seemed to have an astonishingly accurate conception of aphasic speech disturbances as they are understood to-day. The Germans made several early contributions to the study of aphasia, among them being those of Bergemann in 1847, Hasbach in 1852, and Nasse in 1853. In our own country the observations of Jackson must be mentioned, and also those of Rush, Hun, Bigelow, and others. The most important early American contribution made to the subject of aphasia was that of Seguin in 1867.

In England the labors of Russell, Gairdner, Bastian, Broadbent, Ogle, and Bateman contributed largely to our present knowledge; in fact, the names of these physicians are landmarks in the history of aphasia.

In Germany, the work of Wernicke, Grashey, Lichtheim, and Freud did much to extend the conception of the term aphasia and to give rational explanations of some of its subdivisions. Wernicke particularly, in 1874, furnished a basis for the conception of sensory aphasia. He showed clearly that lesion of the first temporal gyrus produced a symptom-complex, constituting what he called sensory aphasia.

Aside from Broca and Wernicke, the three names that stand out above those of all the others who early increased our knowledge of aphasia are Trousseau in France, Hughlings Jackson in England, and Kussmaul in Germany, the latter having written in 1877 what is to-day the soundest and fullest treatise on the subject of aphasia in any language.

In 1881, Exner maintained that there is a separate area of the brain in which are stored the motor memories of writing, destruction of which area causes agraphia. This centre he placed at the base of the left second frontal convolution. The majority of modern investigators and writers on the subject of aphasia are opposed to the existence of such a centre.

Of Italian investigators, mention must be made of the works of Seppilli and of Banti.

In France, the numerous contributions of Trousseau were ably followed by those of Charcot, who suggested an explanation of aphasia based upon the theory of autonomous speech centres.

While Charcot's views were being disseminated the reign of diagrams began, an epoch which seems to-day to have been attended principally by the construction of schemata by which could be explained theoretically the different forms of aphasia that might occur from interruption in the conductive, receptive, and emissive parts of the various speech centres. The names that are particularly associated with this period are Wernicke, Lichtheim, Kahler, and Pick.

In 1881 appeared an excellent summary on "Word Blindness and Word Deafness," from the pen of Mlle. Skwortzoff. Three years later, sensory aphasia was discussed thoroughly by Seppilli. In 1886 Ballet contributed a very serviceable article on "Internal Language and the Various Forms of Aphasia," and in the following year Ross, of Manchester, published a most valuable series of papers on "Aphasia."

Meanwhile there had been accumulating evidence in the shape of apparently anomalous cases to show that the conception of aphasia as taught by Charcot and his school was largely erroneous, and the labors of Dejerine and of his pupils, of Serieux, and of others led to the overthrow of the reign of autonomous speech centres. The contributions of Dejerine from 1879 to 1899 bearing on this subject deserve special mention.

The investigations of Vialet, embodied in "The Cerebral Centres of Vision and the Intercerebral Visual Nervous Apparatus," in 1893, were also most important.

In 1893 and 1894 Wyllie's comprehensive lectures on the disturbances of speech appeared. In the following year Freund, of Breslau, published a short monograph, the thesis of which was that the conception of so-called subcortical sensory aphasia is entirely too narrow.

In the same year Redlich gave a detailed and instructive report of a case of pure word blindness. In 1896 Mirallié reviewed Dejerine's work, made an analysis of the cases of sensory aphasia which had been recorded with autopsy findings, and published several new observations of sensory aphasia.

In the following year Lantzenberg published a brochure on "Motor Aphasia" reflecting the teachings of Brissaud.

In 1897 three important English contributions appeared —those of Bastian, a pioneer in the subject whose early utterances on aphasia have been shown by time to be astonishingly correct; Bramwell's masterful lectures, including many most important cases; and Elder's studious monograph. In the following year the writer published a volume on the subject, entitled "The Genesis and Dissolution of the Faculty of Speech; A Study of Aphasia" (The Macmillan Company), upon which this article is based.

Thus even the briefest review of the history of aphasia shows that this symptom has attracted wide attention during the past third of the nineteenth century. Among the names without mention of which the history of aphasia should not be written, are those of Pick, Hammond, Ferrier, Eskridge, Wilbrand, Berlin, Grashey, Soury, Sachs, Starr, Henschen, Henshelwood, Thomas and Roux, Stricker, and many others.

The Zone of Language.—The zone of language is an area of the brain in which are carried on the processes essential to speech and its components. It is that part of the brain whose functioning is the necessary material substratum of conception, of comprehension, and of expression. This speech area or zone of language is not, in all probability, strictly delimited. It varies in individual cases, and at different periods of life in the same individual, *i.e.*, it is subject to phylogenetic variation and to ontogenetic variation as well; the latter depending somewhat on the speech acquisition of the individual, and on the range and number of avenues by which he receives or has schooled himself to receive information of objects. This area is a receptive and an emissive centre for all forms of stimuli or excitations that reach it, and that its individual developmental metamorphosis has accustomed it to accept, to give tenancy to, and to elaborate into new forms of stimulation. It is receptive chiefly to auditory and visual stimuli, which it emits to other centres, and also to kinæsthetic, olfactory, and gustatory stimuli. It is emissive to the frontal lobes and to the cortex of the Rolandic region, from which start the motor projection tracts and by which all thought externalization is mediated.

The speech area or zone of language is an area made up of neurons, some of which send their axones into the Rolandic region and into the frontal regions of the brain, while others confine their distribution to the speech area itself; and, as they do not pass outside of this area, they may be looked upon as intercentral neurons.

The zone of language has no projection fibres going directly through the motor projection tract; it sends no impulses directly to the projection tract which carries down neural impulses to be externalized as speech. On the contrary, the zone of language sends impulses composed, in the illiterate, of auditory and articulatory memories of the word, and, in the educated, of auditory, visual, and articulatory memories, to the Rolandic cortex and to particular areas of this region, depending on the manner in which the idea is to be externalized; that is, whether by spoken or written word or symbol, or by some form of mimetic or purposeful action. If the idea is to be expressed by articulate speech, the impulses are sent to that area of the Rolandic region in which there is separate allocation for the movements of respiration, vocalization, lingual and labial action. This area is in the front of the ascending central convolution, adjacent to the area in which are stored sensory memories of articulate movements (Broca's area). From here the real motor impulses start. They go down through the motor projection tract, the axones of which *en masse* form the pyramidal tract, and the central motor projections of the cranial nerves, to the various muscles whose contraction mediates articulate speech. There is reason to believe that these outgoing impulses are coordinated, given rhythm, force, and association, not in the cortex of the brain, but in stations situated in the brain ganglia, the cerebellum, and the pons-oblongata, the centres composing these stations acting automatically.

When the idea is externalized in writing, in complex movements such as mimetic movements, or by a simple nod of negation or affirmation, a simple movement of beckoning, the genesis of the symbol or the pantomime is exactly analogous to that of articulate speech. They are the result of internal language and require the absolute integrity of the zone of language. All thoughts or ideas are revealed through words, acts, and deeds, all of which are the immediate result of muscular action. This action is conditioned by influences operative on the Rolandic cortical area, for it is there, and there alone, that movements having differentiated functions have representation.

The centres of speech in the area of language are three in number: the centre for auditory memories, the centre for visual memories, and the centre for articulatory kinæsthetic memories. The latter, unfortunately, is often called the centre for motor memories. These three centres have a very definite localization, and their position is of great ontogenetic importance. The centre in which are stored the memories of articulation is situated in the third frontal convolution, immediately adjacent to that portion of the Rolandic cortex the cells of which give origin to the projection fibres going to the tongue, the lips, and the larynx; that is, to the parts which supply the peripheral mechanism of articulate speech. The centre in which are stored the visual images is situated in a definite part of the inferior parietal lobule, in that portion of the lobule known as the angular gyrus, and if we have in mind the central projections of the optic tract after it leaves the external geniculate body, the interior quadrigeminal body, and the pulvinar of the thalamus, until